A TREATISE ON THE DISEASES OF THE NERVOUS SYSTEM.

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NERVOUS SYSTEM.

BY

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BOOK II.

SPECIAL PATHOLOGY OF THE NERVOUS SYSTEM.

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PART III.—DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

CHAPTER I.

ANATOMICAL AND PHYSIOLOGICAL INTRODUCTION.

(I.)—STRUCTURE OF THE SPINAL MEMBRANES.

THE spinal cord is surrounded by a compound connective-tissue sheath, consisting of (1) the dura mater, (2) the arachnoid, and (3) the pia mater.

§ 349. (1) *The dura mater* is composed of lamellæ, each of which consists of a layer of parallel bundles of fine connective-tissue fibres. Flattened, more or less branched connective-tissue cells lie between the lamellæ; they lie in spaces which communicate with one another, the latter constituting the lymph-canalicular system. The inner surface of the dura mater is lined by a thin hyaline elastic membrane, which is covered by a continuous layer of nucleated endothelial plates. The outer surface is also covered with a continuous layer of endothelium. The dura mater is richly supplied with blood-vessels and nerves.

(2) *The arachnoid sheath* is a delicate membrane, composed of parallel bundles of connective-tissue fibres, longitudinally disposed, with connective-tissue corpuscles lying between them. The outer surface is covered by one or two layers of endothelial plates, constituting an endothelial membrane. On the inner surface is a fenestrated membrane, composed of anastomosing, transversely disposed trabeculæ of connective-tissue fibres, the

inner surface of which is covered by a single layer of endothelial plates.

(3) *The pia mater* consists of an external and internal portion. The former is composed of longitudinal bundles of connective-tissue fibres, and its external surface is covered by an endothelial layer (Klein). The internal portion, or intima piæ,

FIG. 100.

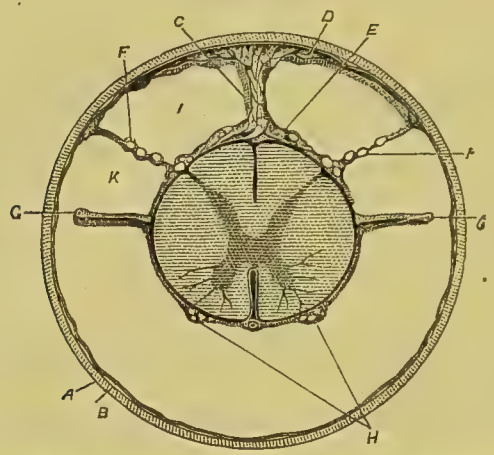

FIG. 100 (After Key and Retzius). *Transverse Section of the Spinal Cord in the upper dorsal region, with its membranes.*—Close on the inner surface of the dura (A) lies the arachnoid (B), which is thrown into longitudinal folds at intervals. In the posterior subarachnoidal space (the part behind the ligamenta denticulata, G), the septum posticum (C) may be observed in the middle, with its numerous partitions, along with the subarachnoidal spaces which they enclose. The septum becomes partly attached to the arachnoid externally, and partly spreads laterally over the inner surface of that membrane (D). The septum spreads internally over the pial sheath as the epipial subarachnoidal tissue (E), forming numerous small spaces. Two vessels may be observed in this epipial space. F, the posterior nerve roots, surrounded by the subarachnoidal membranes. The space (I) between the latter membranes and the septum posticum is of variable depth. K is the space between the posterior nerve roots, with their membranes, and the ligamentum denticulatum; this space being free from membrane throughout the entire length of the cord, the subarachnoidal fluid finds a freer passage through it than through any other part of the posterior subarachnoidal space. Anterior to the ligamenta denticulata (G), the anterior subarachnoidal space may be observed free from membrane. H, the anterior nerve roots.

FIG. 101.

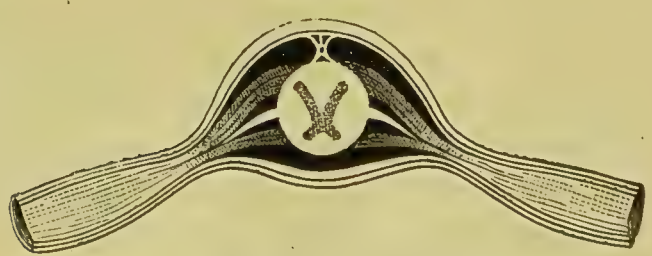

FIG. 101 (From Key and Retzius). *Diagram of a Transverse Section of the Spinal Cord and its membranes, showing the natural size and relative positions of the cord, membranes, and spaces.*

is a meshwork of bundles of connective-tissue fibres, its inner surface being lined by a layer of endothelial cells. The pia mater contains numerous blood-vessels, which lie between the external and internal layers, whence they penetrate into the substance of the cord being surrounded by a prolongation of the pial sheath.

The subarachnoidal tissue consists of a plexus of trabeculæ of fibrous connective tissue ensheathed in endothelium and containing a few elastic fibres. It forms a spongy tissue between the arachnoidal and pial sheaths, and subdivides the subarachnoidal space into numerous minute lacunæ. It is a prolongation of the inner portion of the arachnoid, and its trabeculæ contain larger and smaller blood-vessels.

Ligamentum denticulatum stretches like a diaphragm between the arachnoid and pial sheaths on each side of the cord, from the foramen ovale magnum down to the filium terminale, between the anterior and posterior nerve roots. The subarachnoidal space is consequently divided into an anterior and posterior chamber. The ligamentum denticulatum consists of trabeculæ of connective-tissue bundles, the trabeculæ being covered with endothelium. The tissue passes into the external layer of the pia mater (Klein).

Isolated connective-tissue trabeculæ also extend between the dura mater and arachnoid; they are ensheathed in endothelium, while blood-vessels and nerves pass from the one membrane to the other. These trabeculæ are most numerous in the posterior parts of the cord.

Between the dura mater and arachnoid is the subdural, and between the arachnoid and pia mater is the subarachnoidal lymph space. Neither of these spaces form one open and free cavity, inasmuch as numerous connective-tissue trabeculæ pass between the dura mater and arachnoid, and between the latter and pia mater. The two spaces do not, however, communicate with one another.

The nerve roots receive a prolongation from both the arachnoidal and dural sheaths, and consequently the lymph spaces of the peripheral nerves and their ganglia have been injected from the subarachnoidal and subdural spaces respectively (Key and Retzius).

(II.)—STRUCTURE OF THE SPINAL CORD AND MEDULLA OBLONGATA.

The spinal cord consists of a framework, with grey and white matter embedded in it.

§ 350. *The framework* consists of the following parts:—

(1) *Connective-tissue Processes.*—Processes of fibrous connective tissue pass from the intima piæ into the anterior fissure, and at different points of the circumference of the cord, where they form septa, which divide the white columns of the cord into segments. These prolongations of the intima piæ carry blood-vessels into the cord.

(2) *Neuroglia.*—The chief part of the framework consists of a semi-fluid substance named the *neuroglia-matrix.* This substance presents a granular aspect under certain reagents, but is homogeneous in the fresh condition. Numerous minute fibrils, which anastomose with one another in a network, are

FIG. 102.

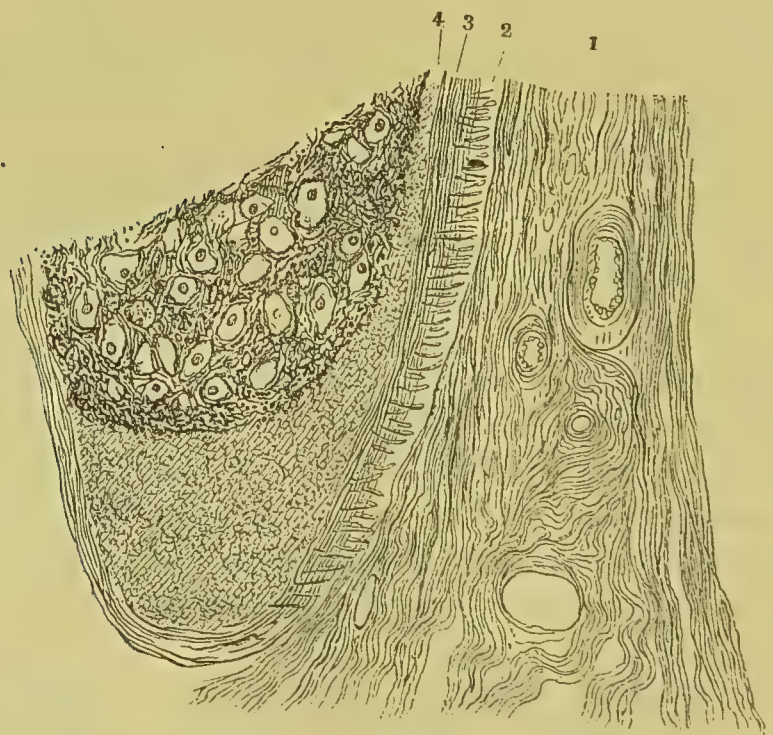

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FIG. 102 (From Henle's Anatomie). *Diagram of the Spinal Cord and its Membranes.* 1, the dura mater; 2, the arachnoid, and (3) the pia mater; 4, the cortical layer of the neuroglia.

embedded in this substance. These fibrils have a longitudinal direction, except in the septa, where they form transverse networks, and in the grey substance, where they extend uniformly in all directions (Klein). Flat, branched, nucleated connective-tissue corpuscles are found in connection with the network of the neuroglia fibrils. The neuroglia, therefore, is composed of neuroglia-matrix, neuroglia fibrils, and branched cells, the latter being named *Deiter's cells* (*Fig.* 103).

FIG. 103.

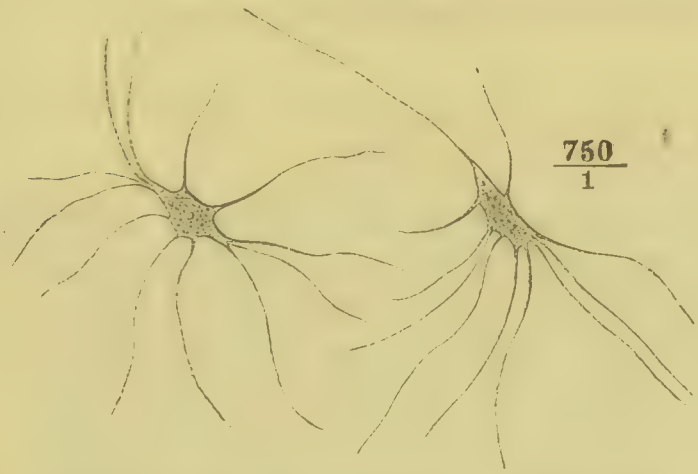

Fig. 103 (From Henle's Anatomie). *Deiter's Cells.*

Distribution of the Neuroglia.—The neuroglia is abundant in the following parts :—

(*a*) On the external surface of the cord, where it forms a peripheral crust beneath the intima piæ, the latter being easily separated from the former.

(*b*) In the septa which pass between different sections of the white matter ; the posterior fissure being, indeed, only a septum of this kind (Klein).

(*c*) It forms the ground substance of the anterior and posterior nerve roots.

(*d*) A layer of neuroglia of considerable thickness surrounds the epithelial lining of the central canal, named the *central grey nucleus* of Kölliker.

(*e*) A peculiar form of neuroglia is found in the posterior portion of the posterior grey horns forming the *substantia gelatinosa* of Rolando.

The neuroglia is always more abundant near the grey matter and in the peripheral crust than in the parts between them.

§ 351. *The grey matter* occupies the central parts of the cord in the well-known shape of an H. The median part contains the central canal, and the "central grey nucleus" of Kölliker the anterior grey and white commissures lying in front and the posterior commissure behind it. The lateral parts or columns consist of an anterior, middle, and posterior part; the first of these representing the *anterior*, and the last the *posterior grey horn;* while the middle portion on each side of the central canal consists of the vesicular column of Clarke, and what may be called the *central column.* The central grey nucleus of Kölliker may indeed be regarded as a portion of the central column.

The grey matter consists of a (1) matrix of neuroglia, (2) ganglion cells, and (3) nerve fibres.

(1) *The neuroglia* of the grey matter is similar to that of the white. It is looser in texture and more spongy in the central grey column than in either the anterior or posterior horns, and in this situation it also contains a relatively larger number of Deiter's cells.

(2) *The ganglion cells* of the anterior horns are relatively large, branched cells, containing in some animals masses of yellow pigment (§ 13). These cells are surrounded by a lymph space, through which the processes of the cell pass. The ganglion cells of the posterior horns are much smaller and less branched than those of the anterior horns. Some of the latter appear spindle-shaped, but each extremity is branched into several processes.

(3) *The nerve fibres* of the grey matter are of different kinds. The great bulk of the grey matter is composed of a minute and dense network of fine fibrils, named Gerlach's nerve network. The nerve network surrounding the central grey nucleus of Kölliker is less dense than in other parts. The branched processes of the ganglion cell attach themselves to Gerlach's nerve network; while the unbranched processes pass into a medullated nerve fibre of the anterior root. The cells of the posterior horns are not directly connected with any nerve fibres, but anastomose with them indirectly through Gerlach's nerve network (Klein).

§ 352. *The white matter* is composed of medullated nerve fibres, by far the greater number being arranged in a longitudinal direction. A vertical section of the spinal cord is represented in *Fig.* 104, showing the longitudinal disposition of the fibres in the anterior and lateral columns. Each nerve fibre possesses an axis cylinder, and a medullary sheath, but there is no definite evidence of the presence of a sheath of Schwann, or of nerve corpuscles, as in the medullated fibres of the cerebro-spinal nerves. The nerve fibres are embedded in neuroglia as previously described; they vary much in size, some being broad, some of medium size, while others are very fine.

FIG. 104.

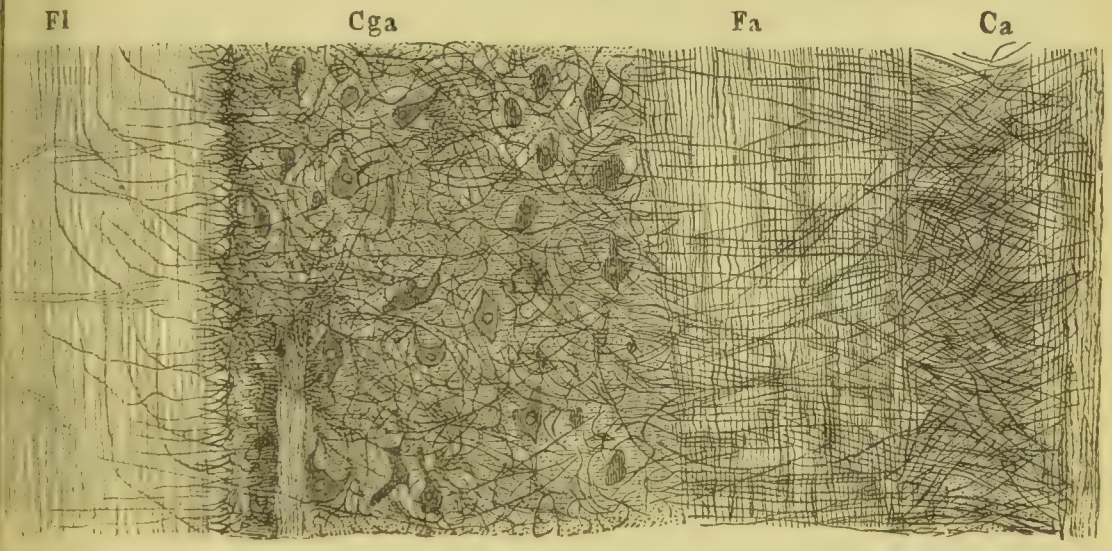

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Fig. 104 (From Henle's Anatomie).—Fl, Anterior column; Cga, Anterior grey horn; Fa, Lateral column; Ca, Posterior grey horn.

The white matter also contains nerve fibres that have an oblique or horizontal direction. The following may be distinguished:—

(1) The fibres of the posterior roots pass into the grey matter of the posterior horns as horizontal fibres. These fibres on entering the cord spread out laterally in the form of a fan, so that an external fasciculus, an internal fasciculus, and a median portion may be distinguished. The fibres of the *external fasciculus* wind forwards round the external margin of the posterior horn, and at least some of them pass forwards through the anterior commissure, a few even passing between the longitudinal fibres of the anterior column, so as to reach the internal and anterior groups of ganglion cells of the anterior grey horn of the opposite side

(*Fig.* 134, *p''*). The fibres of the *internal fasciculus* pass between the longitudinal fibres of the posterior root-zone to gain the posterior horn (*Fig.* 134, *pr'*). Some of them then wind round the vesicular column of Clarke, but it is not known whether they are connected with the cells of that column. A few of these fibres appear to pass behind the vesicular column of Clarke and to decussate with the corresponding fibres of the opposite side in the posterior commissure. The *median portion* of the posterior root enters the white matter of the posterior column, and its fibres pass for a longer or shorter distance in a longitudinal direction, either upwards or downwards, before joining the posterior grey horns.

(2) The medullated nerve fibres of the anterior nerve roots pass in an oblique direction from the grey matter of the anterior horns through the white matter.

(3) The anterior commissure is said by Gerlach to be composed of medullated nerve fibres that pass from the grey matter of the anterior horn of one side into the white matter of the anterior tract of the opposite side. Some of the fibres, however, pass from the anterior horn of one side to the pyramidal tract of the opposite side, while others, as already described, pass from the internal fasciculus of the posterior roots of one side to the anterior grey horn of the opposite side.

(4) Medullated nerve fibres emerge from the sides of the grey matter of the anterior horns, and after a short course enter the white matter of the lateral tracts (Klein).

(5) Nerve fibres emerge from the posterior grey horns, and after a longer or shorter horizontal course enter the white matter of the posterior column (Gerlach). It is probable that they leave the posterior tracts again as the nerve fibres of the posterior roots (Klein).

(6) Fibres emerge from the cells of the vesicular column of Clarke, which pass obliquely outwards and upwards to enter the direct cerebellar tract (Flechsig). These fibres form round bundles at the junction of the grey substance and the lateral column, and are cut transversely in horizontal sections. These bundles are represented in *Figs.* 134 to 140 as dark round spots near the *formatio reticularis* (*fr*).

§ 353. *Distribution of the Vessels of the Spinal Cord, Medulla Oblongata, and Pons.*

The vertebral artery is the first and largest branch of the subclavian artery. It arises from the posterior aspect of the trunk, and ascends through the foramina in the transverse processes of all the cervical vertebræ, except the last. It winds backwards around the articulating process of the atlas, pierces the dura mater, enters the skull through the foramen magnum, and terminates at the lower border of the pons Varolii by uniting with the corresponding vessel of the opposite side to form the *basilar artery*.

The basilar artery runs forward in the groove on the anterior surface of the pons Varolii, and divides at the anterior border of the pons into two terminal branches, one to either side.

FIG. 105.

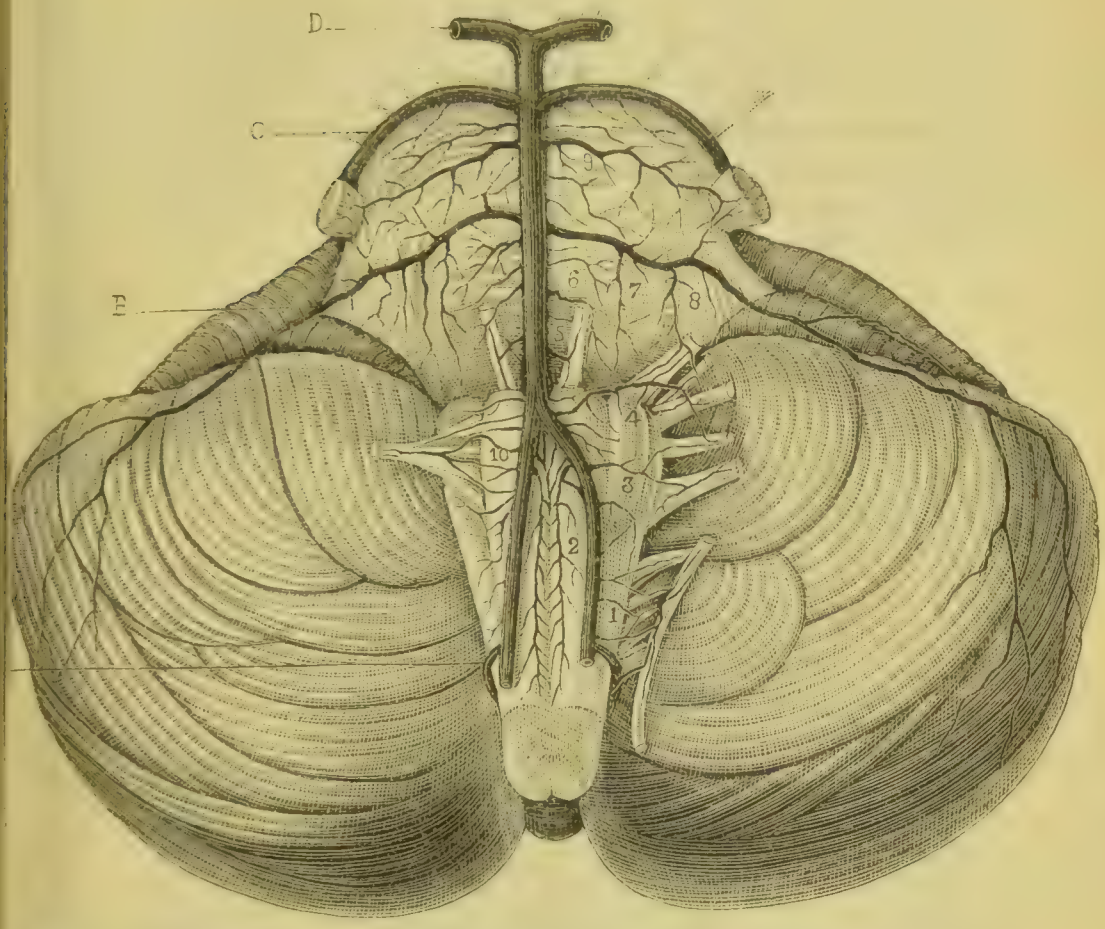

FIG. 105 (After Duret). *Arteries of the Medulla Oblongata, Pons, and Inferior Surface of the Cerebellum.*

1, Root arteries of the spinal accessory nerve.
2, Anterior spinal arteries.
3, Arteries of the pneumogastric and glosso-pharyngeal nerves.
4, Inferior arteries of the auditory and facial nerves (vertebral branches).
5, Root arteries of the sixth nerve.
6 and 7, Arteries of the sub-olivary fossa.
8, Superior arteries of the auditory and facial nerves (branches of the middle cerebellar artery).
9, Arteries of the trigeminal nerve.
10, Arteries of the hypoglossal nerve (branches of the vertebral and anterior spinal arteries).
A, Inferior cerebellar artery.
B, Middle cerebellar artery.
C, Superior cerebellar artery.
D, Posterior cerebral artery.

Branches.—The branches of the vertebral and basilar artery are the following :—

Vertebral.	*Basilar.*
Lateral spinal,	Transverse,
Muscular branches,	Middle cerebellar,
Posterior meningeal,	Superior cerebellar,
Anterior spinal,	Posterior cerebral.
Posterior spinal,	
Inferior cerebellar.	

The lateral spinal branches enter the intervertebral foramina, and taking the course of the roots of the spinal nerves, are distributed to the spinal cord and vertebræ. Where the vertebral artery curves round the articular process of the atlas, it gives off several *muscular* branches.

The posterior meningeal arteries are small branches which enter the cranium through the foramen magnum, to be distributed to the dura mater of the cerebellar fossæ, and to the falx cerebelli.

FIG. 106.

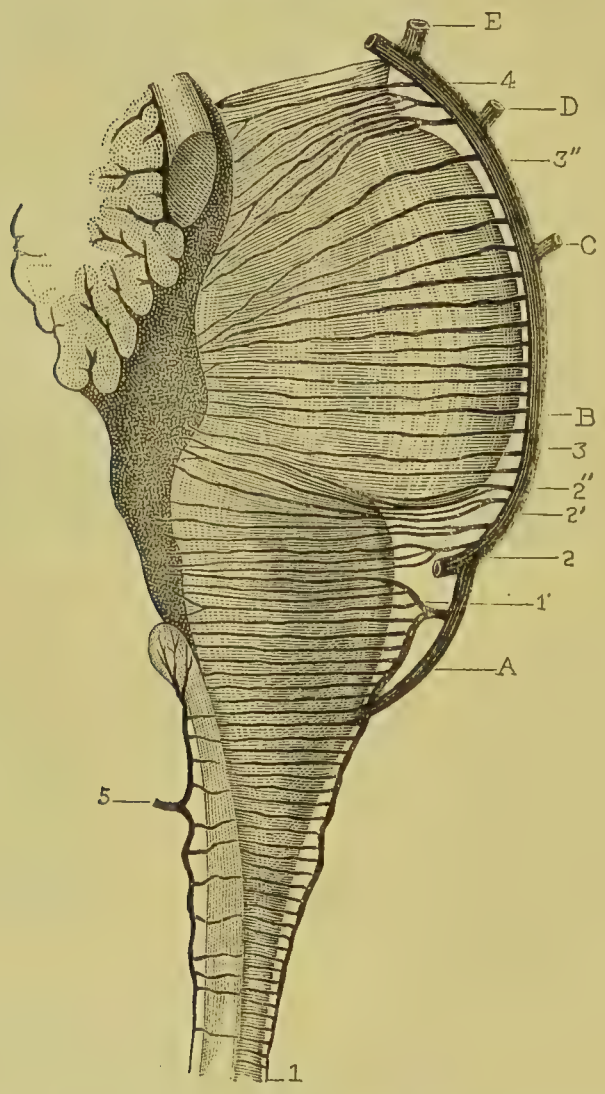

FIG. 106 (After Duret). *Arteries of the Pons and Medulla.*

1 1′, Anterior spinal artery, the bulbar branches.
2 2′ 2″, Inferior arteries of the pons.
3 3″, Median arteries of the pons.
4, Superior arteries of the pons.
5, Posterior spinal arteries, median branches.

A, Left vertebral artery.
B, Basilar artery.
C, Middle cerebellar artery.
D, Superior cerebellar artery.
E, Posterior cerebral artery.

The anterior spinal artery is a small branch which unites with its fellow of the opposite side, on the front of the medulla oblongata. The artery formed by the union of these two vessels descends along the anterior aspect of the spinal cord, to which it distributes branches, and forms the commencement of the anterior median artery.

The posterior spinal artery winds around the medulla oblongata to reach the posterior aspect of the cord, and descends on either side to the cauda equina. It communicates very freely with the spinal branches of the intercostal and lumbar arteries, and near its origin sends a branch upwards to the fourth ventricle.

The inferior cerebellar arteries wind around the upper part of the medulla oblongata to reach the under surface of the cerebellum, to which they are distributed. They pass between the filaments of origin of the hypoglossal nerve in their course, and anastomose with the superior cerebellar arteries. Small branches derived from these trunks pass to the choroid plexus of the fourth ventricle.

FIG. 107.

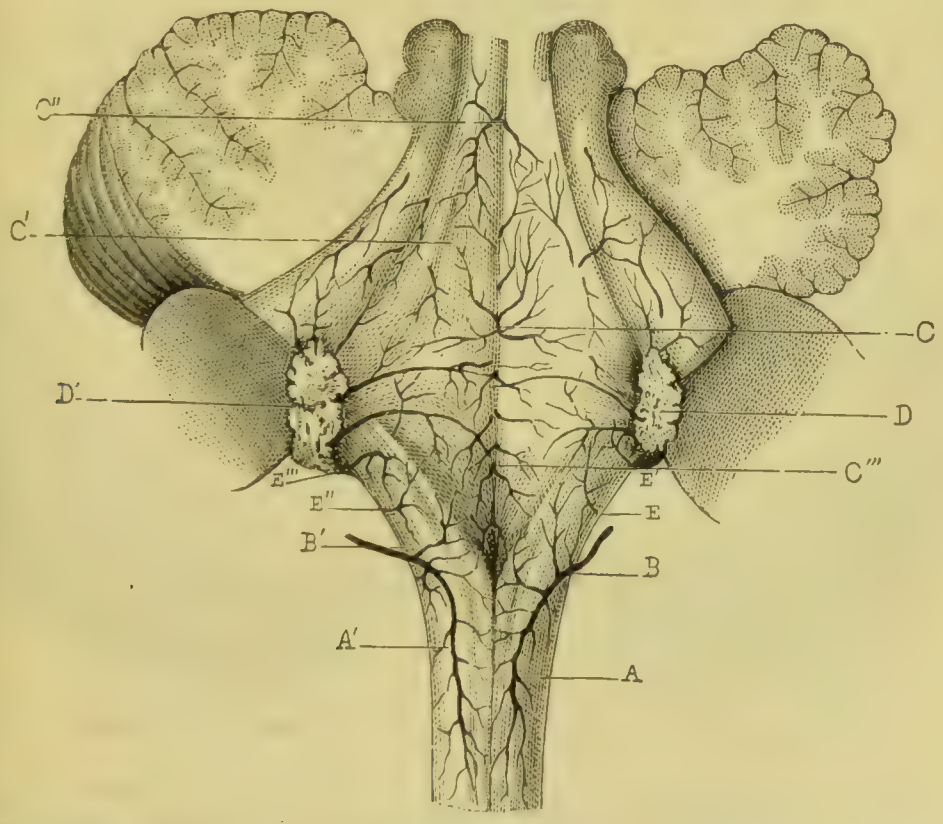

FIG 107 (After Duret). *Distribution of the Arteries on the Floor of the Fourth Ventricle.*

A A′, Posterior spinal artery.
B B′, Its pyramidal branches.
C C′ C″ C‴, Emergence of the median arteries.
D D′, Choroid plexus drawn to one side. (Two or three arteries may be seen to emerge from it.)
E E′ E″ E‴, Arteries of the restiform bodies coming from the inferior cerebellar artery.

The transverse branches of the basilar artery supply the pons Varolii, and adjacent parts of the brain.

The middle cerebellar artery arises from the trunk of the basilar, about its middle. It runs parallel to the transverse branches, and passes along the middle peduncle to be distributed to the anterior part of the under surface of the cerebellum. It gives off a small branch, *auditiva interna*, which accompanies the auditory nerve into the meatus auditorius internus, and to the labyrinth of the ear. The auditory branch is frequently derived directly from the basilar.

The superior cerebellar arteries wind around the crus cerebri on each side, lying in relation with the fourth nerve, and are distributed to the

FIG. 108.

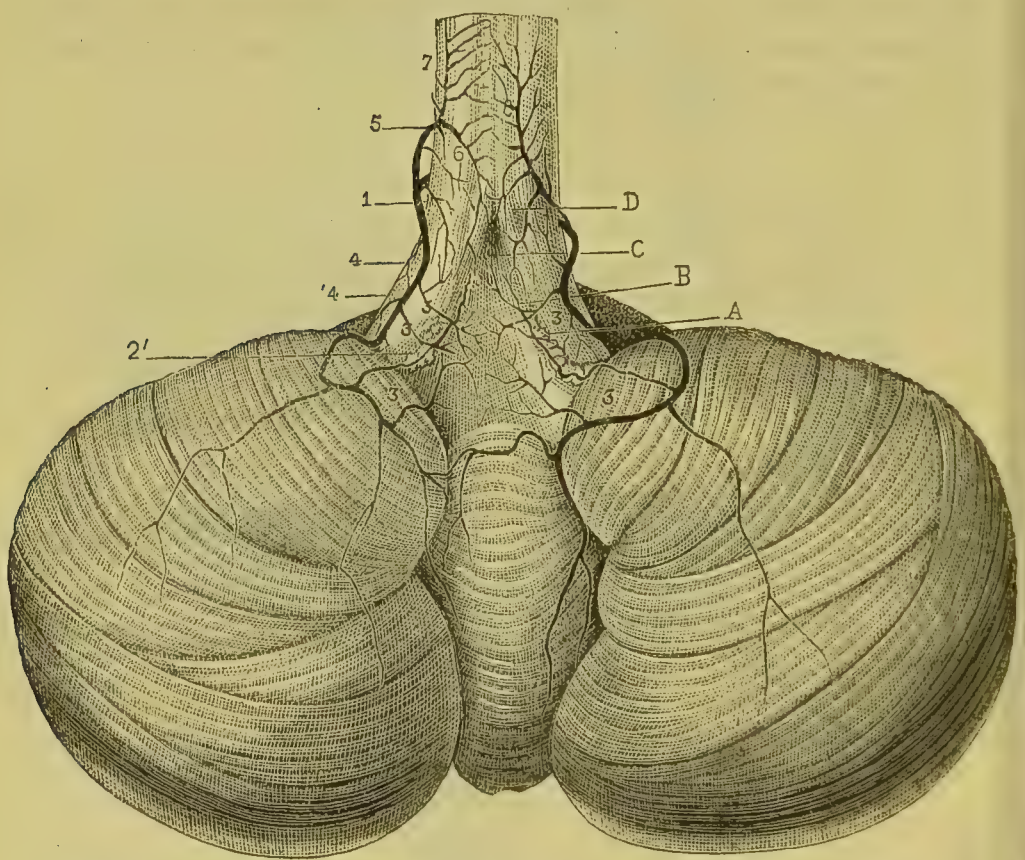

FIG. 108 (After Duret). *Arteries of the Posterior Part of the Medulla and the Cerebellum.*

A, Choroid plexus. B, Choroid velum. C, Posterior opening, forming a communication between the fourth ventricle and the posterior subarachnoid space. D, Posterior pyramid.

1, Inferior cerebellar artery.

2 2″, Artery of the choroid plexus.

3 3 3 3, Arteries of the choroid velum. Some proceed to the floor of the fourth ventricle; they are capillary.

5, Posterior spinal artery.

6, Its ascending or pyramidal branch.

7, Its descending branch.

8, Its median branch.

upper surface of the cerebellum anastomosing with the inferior cerebellar. Branches of the superior cerebellar arteries run inwards to supply the valve of Vieussens and the posterior part of the velum.

The ascending cervical branch of the inferior thyroid artery gives off one or two branches (*spinal branches*) which enter the intervertebral foramina along with the cervical nerves, and assist in supplying the bodies of the vertebræ and the spinal cord and its membranes.

The spinal branches of the aortic intercostal arteries enter the intervertebral foramina of the dorsal region, and supply the vertebræ, spinal cord, and membranes.

The spinal branches of the lumbar, ilio-lumbar, and lateral sacral arteries enter the spinal canal through the intervertebral foramina; they are distributed like the other spinal arteries, and anastomose with them.

§ 354. The following arteries are distributed to the medulla oblongata and pons :—

1. The Root Arteries.—These arteries are directed laterally towards the roots of the nerves, which they penetrate near their point of emergence. They subdivide into an *ascending* branch, which is directed towards the nuclei of origin of the nerves, and a *descending* branch which descends towards the periphery.

(*a*) *Anterior Root Arteries* (*Fig.* 109, *ar*).

(1) The arteries of the hypoglossal nerve are derived from both the anterior spinal and vertebral arteries.

(2) The arteries of the sixth nerve are derived from the basilar.

(3) The arteries of the third nerve are derived from the trunk of the basilar near its termination.

(*b*) *Lateral Root Arteries* (*Fig.* 109, *lr*).

(1) The arteries of the spinal accessory nerve are derived from the inferior cerebellar and vertebral arteries.

(2) The arteries of the pneumogastric and glosso-pharyngeal nerves arise from the vertebral artery.

(3) The arteries of the auditory, facial, and portio intermedia (nerve of Wrisberg) are derived from the vertebral a little before its termination, and from a branch of the basilar. Branches may also descend perpendicularly from the middle cerebellar artery.

(4) The artery of the trigeminus is comparatively large and constant, and is derived directly from the basilar about its middle. Another branch is derived from the middle cerebellar artery.

(5) The fourth nerve, as well as the optic and olfactory nerves, receives its arterial supply from the branches of the circle of Willis.

FIG. 109.

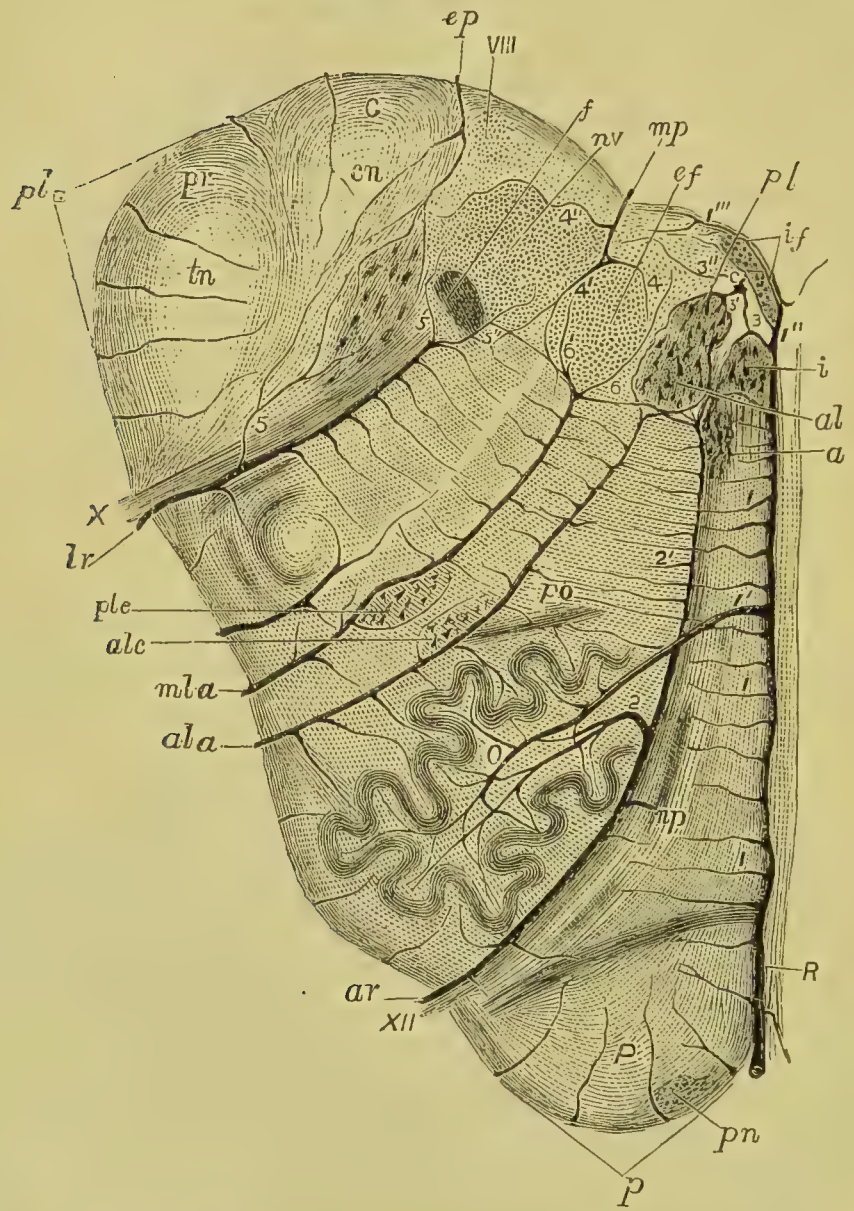

FIG. 109. *Section of the Medulla Oblongata, showing the Distribution of the Vessels.*

R, Artery of the Median Raphé.
1 1 1, Branches to the *formatio reticularis.*
1′, Branch to the olivary body.
1″, Branches to the hypoglossal nucleus.
1‴, ,, ,, floor of the fourth ventricle, and to the internal inferior nuclei of the facial (*if*).
p, Pyramidal arteries.
ar, Anterior root artery (hypoglossal).
2′, Branch to olivary body.
2″, Branches to the *formatio reticularis.* It terminates in branches to the hypoglossal nucleus.
lr, Lateral root artery (vagus).
5, Branch to the restiform body and the inner division of the inferior cerebellar peduncle.
5′, Branches to the nucleus of the vagus. Also gives branches to the ascending root of the fifth and the *formatio reticularis.*

2. Arteries of the Median Raphé (*Fig.* 109, R).

(*a*) Bulbar arteries derived from the anterior spinal artery (*Fig.* 106, 1).

(*b*) Inferior arteries of the pons derived from the lower end of the basilar (*Fig.* 106, 2 2′ 2″).

(*c*) Median arteries of the pons, derived from the trunk of the basilar (*Fig.* 106, 3 3″).

(*d*) Superior arteries of the pons, derived from the superior end of the basilar (*Fig.* 106, 4).

The annexed diagram (*Fig.* 110) shows that a double row of vessels enter the Raphé, the vessels on each side of the middle line entering at different levels. A vertical section of the olivary body shows that the vessels enter the hilus in a similar manner; so that the branches from the anterior root artery and the artery of the Raphé are never seen in the same horizontal section as represented in *Fig.* 109.

3. The Lateral Arteries of the Medulla Oblongata.

(*a*) *Anterior lateral artery* (*Fig.* 109, *ala*) passes into the substance of the medulla behind the olivary body. It gives branches to the

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ala, The anterior lateral artery of the medulla oblongata. It supplies branches to the formatio reticularis, olivary body, anterior nucleus of the lateral column (*alc*), and terminates in branches to the hypoglossal nucleus.

mla, The middle lateral artery of the medulla oblongata. It supplies branches to the *formatio reticularis*, the posterior nucleus of the lateral column (*plc*), and terminates in branches which are distributed to the external accessory nucleus of the facial (*ef*).

pla, The posterior lateral arteries of the medulla oblongata. They supply the restiform bodies.

C, Central artery.

3 3′ 3″, Branches to the hypoglossal and external accessory facial nuclei.

mp, Median posterior artery.

4 4′ 4″, Branches to the external accessory facial and pneumogastric nuclei.

ep, External posterior artery. It supplies branches to the internal division of the inferior peduncle of the cerebellum and restiform body.

i, Internal group of cells of the hypoglossal nucleus.

al, Antero-lateral ,, ,,

pl, Postero-lateral ,, ,,

a, Anterior ,, ,,

alc, Anterior nucleus of the lateral column.

plc, Posterior ,, ,,

VIII, Inferior portion of the posterior median acoustic nucleus.

if, Internal accessory facial nuclei.

ef, External accessory facial nucleus.

f, Fasciculus rotundus.

XII, Hypoglossal nerve.

X, Pneumogastric nerve.

G, Column of Goll.

pr, Posterior root-zone. The direct cerebellar tract forms a thin band lying external to the column of Goll and posterior root-zone.

cn, Clavate nucleus.

tn, Triangular nucleus.

o, Olivary body.

po, Parolivary body.

np, Nucleus of the pyramid.

pn, Nucleus of the arciform fibres.

P, Anterior pyramid.

olivary body, the anterior lateral nucleus, and terminates between the groups of ganglion cells of the hypoglossal nerve.

(*b*) *Middle lateral artery* (*Fig.* 109, *mla*) passes into the substance of the medulla in front of the restiform body. It gives branches to the posterior lateral nucleus, and terminates between the group of cells, which give origin to the lateral mixed system of nerves.

(*c*) *The posterior lateral arteries* (*Fig.* 109, *pla*) enter the substance of the restiform body behind the roots of origin of the mixed lateral system of nerves.

4. The Central Artery (*Fig.* 109, *c*) of the medulla oblongata is a continuation of the central artery of the spinal cord. It subdivides into internal, middle, and external branches (*Fig.* 109, 3 3′ 3″), which are distributed between the groups of cells of the hypoglossal nucleus.

5. The Median Posterior Artery (*Fig.* 109, *mp*) enters the substance of the medulla oblongata on the floor of the fourth ventricle. It is probably derived from the choroid plexus. It is mainly distributed to the groups of cells which give origin to the nerves of the lateral mixed system.

6. The External Posterior Artery (*Fig.* 109, *ep*) enters the substance of the medulla at the junction of the grey substance with the restiform body.

Fig. 110.

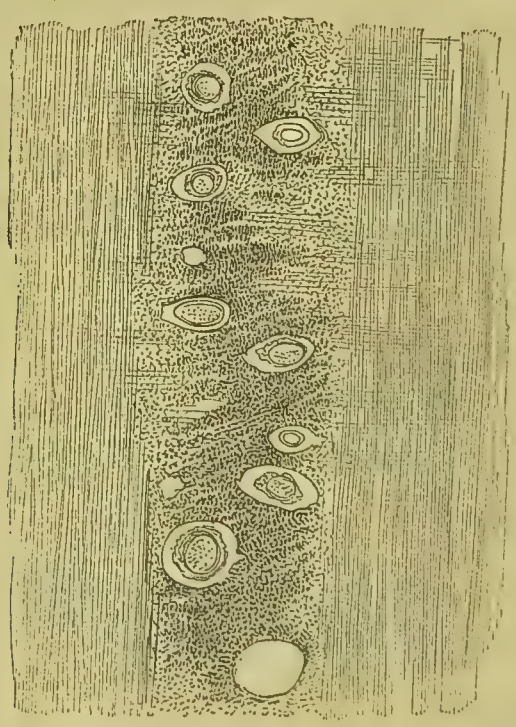


Fig. 110 (From Henle's Anatomie). *Vertical Section of Raphé of the Medulla Oblongata, showing the entrance of the vessels.*

§ 355. *Arteries of the Spinal Cord.*

The anterior median artery gives off a series of small branches, which pass backwards in the anterior median fissure, and reach the anterior commissure, hence these vessels may be called the arteries of the anterior median fissure (*Fig.* 111, *af*). Each of these vessels on reaching the anterior commissure divides into two main trunks, which enter the grey substance of the anterior horns; these may be called the arteries of the anterior commissure (*Fig.* 111, *ac*).

FIG. 111.

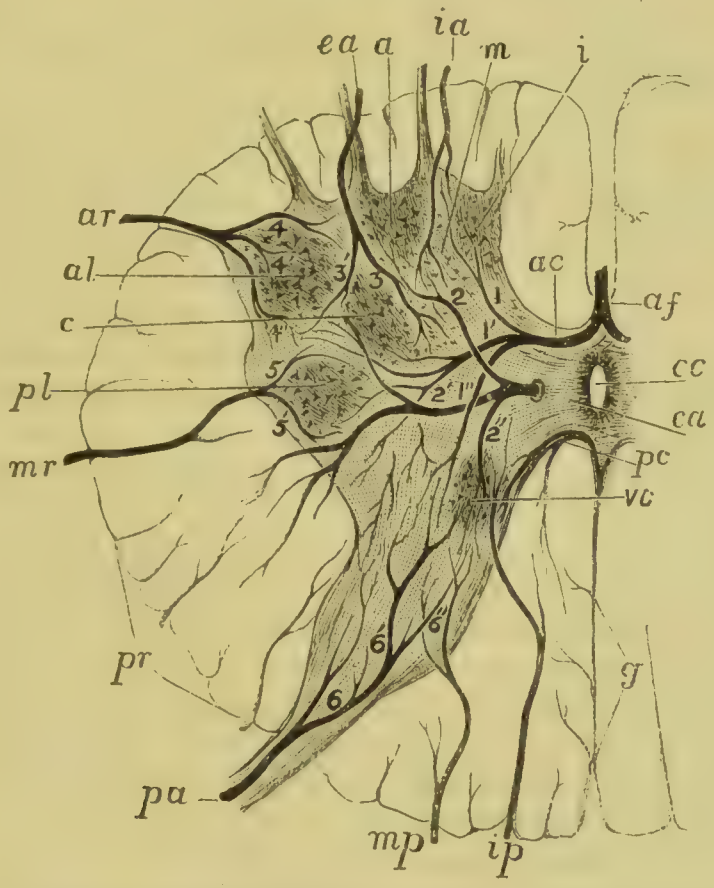


FIG. 111. *Diagram of the Distribution of the Blood-vessels in the Spinal Cord.*

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

The artery of the anterior commissure subdivides into three branches, which, from their position, may respectively be named the anterior (*Fig.* 111, 1), median (*Fig.* 111, 1′), and posterior (*Fig.* 111, 1″) branches. The anterior branch curves forwards, and is distributed to the anterior and internal portion of the grey substance; the median is distributed to the lateral portion of the anterior horn, while the posterior is directed backwards to the posterior horn.

The central artery also gives off an anterior (*Fig.* 111, 2), median (*Fig.* 111, 2′), and posterior (*Fig.* 111, 2″) branch, which are distributed respectively to the anterior, lateral, and posterior portions of the grey substance. The median branches of the two main vessels, besides supplying the grey substance, are also distributed to the pyramidal tract of the lateral column.

The posterior spinal artery (*Fig.* 111, *pa*) gives off branches, which pass by the side of the posterior roots to enter the grey substance of the posterior horns, where they subdivide into a variable number of small branches (*Fig.* 111, 6 6′ 6″), which may be called arteries of the posterior horns. In addition to the vessels just described, a large number pass from the pia mater into the substance of the cord, and some of these are so large and so constant as to deserve special mention; two run by the side of the bundles of fibres which constitute the anterior roots of the nerves, hence they may be called the anterior root arteries. The branch nearest the median fissure may be called the internal anterior root (*Fig.* 111, *ia*), and the other the external anterior root (*Fig.* 111, *ea*) artery.

The internal anterior root artery (*Fig.* 111, *ia*), on entering the grey substance, joins the anterior branches of the first subdivision of the artery of the anterior median fissure and of the central artery.

The external anterior root artery (*Fig.* 111, *ea*), on entering the grey substance, subdivides into two branches, the inner (*Fig.* 111, 3) of which is distributed along with the vessels just mentioned; while the outer branch (*Fig.* 111, 3′) passes between what we may call the antero-lateral (*Fig.* 111, *al*) and central groups (*Fig.* 111, *c*) of cells.

A very constant vessel passes to the grey substance from the pia mater, at the point of junction of the anterior and lateral columns of the cord, and it may therefore be called the *antero-lateral artery* (*Fig.* 111, *ar*). On reaching the grey substance it frequently divides into three branches, one of which passes in front (*Fig.* 111, *ar*, 4 4′ 4″), another behind, and another into the substance of the antero-lateral group of cells. Another constant vessel (*Fig.* 111, *mr*) passes from the lateral aspect of the cord, and on reaching the grey substance it subdivides into two branches, the one of which passes in front and the other behind the postero-lateral group of cells (*Fig.* 111, *pl*), and this vessel may from its position be called the *median-lateral artery*. Small branches (*Fig.* 111, *pr*) pass at short intervals through the posterior part of the lateral column, and, together with the median branches of the first subdivision of the artery of the anterior median fissure, and of the central arteries, supply the posterior part of

the lateral columns; hence these vessels may be called *posterior lateral arteries.*

Two vessels pass from the pia mater into the substance of the posterior column; the one nearest the posterior median fissure, and which may therefore be called the *internal posterior artery* (*Fig.* 111, *ip*), passes between the column of Goll and the posterior root-zone; and after passing through about two-thirds of the depth of the posterior column, it curves outwards to reach the posterior grey horn. The other vessel may be named the *external or median posterior artery* (*Fig.* 111, *mp*); it passes into the substance of the posterior column at the middle of the posterior root-zone, and on reaching about one-third the depth of the posterior column, it curves outwards to reach the posterior grey horn, where it terminates. Small vessels (*Fig.* 111, *g*) pass from the pia mater of the posterior median fissure into the substance of the column of Goll. Another vessel, which may be called the *artery of the posterior commissure* (*Fig.* 111, *pc*), passes from the pia mater along the posterior margin of the posterior commissure, and winds backwards along the internal edge of the posterior horn.

§ 356. *Grouping of the Ganglion Cells.*—The ganglion cells of the anterior horns are arranged in groups which are pretty constant for the same portions of the cord, although the arrangement varies considerably when sections at different elevations are compared. A diagram of the topographical distribution of these groups is given in *Fig.* 111. Beginning at the posterior and lateral aspect of the anterior horn, a group is observed which from its position is called the *postero-lateral group* (*pl*). It is bounded behind by the posterior and in front by the anterior twig of the median branch of the central artery; while on its external aspect it receives branches from the median lateral artery, one of which passes behind and another in front of it. Anterior to this group is another, which from its position is called the *antero-lateral group* (*al*). On its external aspect the group receives branches from the anterior lateral artery, one of these passing behind and another in front of it, while a median branch of the artery may often be seen to pass into its substance. A branch from the external anterior root artery winds round its inner border to gain the posterior aspect; while the anterior branch of the central artery passes along its internal and anterior aspects.

It has already been mentioned that the internal and external anterior root arteries, on reaching the grey substance, divide

into two branches; and the external branch of the former and internal of the latter converge so as to meet at a point like the limbs of the letter V. In the small area of grey matter which lies between these vessels several distinct cells are so constantly observed as to deserve a special name. These cells may from their position be called the *anterior group* (*a*). Another group of large cells, which may be called the *internal group* (*i*), is bounded anteriorly and internally by white substance, and on the external aspect by the anterior branch of the first subdivision of the artery of the anterior median fissure. Another group of cells may be observed towards the centre of the anterior horns, and it may therefore be termed the *central group* (*c*). It is bounded in front and on its internal and external borders by the external and internal branches of the external anterior root artery; and behind and also on its internal border by the median and anterior branches of the central artery. A very important area lies between the internal group on the one hand and the antero-lateral and central groups on the other, while the anterior group passes into its anterior border, like a small wedge, so as to divide it into the form of the letter Y. The cells of this *median area* (*m*) are much smaller than those of the other groups, and the area itself is exceedingly vascular, being supplied by the two anterior root arteries, the anterior branch of the first division of the artery of the anterior median fissure, and the anterior branch of the central artery. A final group of cells lies near the internal border of the posterior grey horn near the posterior commissure called the vesicular column of Clarke (*vc*). We must again direct attention to the fact that *Fig.* 111 is only a diagram; and although it is more like the upper part of the lumbar and lower portion of the cervical enlargements than any other part of the cord, yet it is not a strictly accurate representation of any one section. The distribution of these groups at various elevations of the cord will be better understood after the history of the development of the grey substance has been sketched.

§ 357. *Development of the Central Grey Tube.*

The parts which subsequently correspond to the anterior grey horn are the first portions of the cord to be developed. These are soon suc

ceeded by lateral masses, and somewhat later by the posterior horns. The anterior grey commissure is then formed, and this is soon followed by the development of the posterior commissure, and it is only at a considerably later period that the white commissure appears. When the tube which forms the rudiment of the cord has closed, it is seen to be somewhat oval on section, and at this period it consists almost entirely of grey substance.

The grey substance is at first composed of small round cells, not much larger than lymphoid corpuscles, with a distinct nucleus, and no difference can be detected between one portion and another; the whole is simple and indefinite in its structure. A section of the cord at the third month of embryonic life (*Fig.* 112) shows that the central canal has contracted to a small oval opening, covered by columnar epithelium, while the grey substance has assumed the general outline characteristic of the grey substance of the adult cord. The grey substance is also surrounded by a mantle of white substance, but we shall entirely neglect the history of the development of the latter in the meantime.

§ 358. *Development of the Anterior Grey Horns.*

The most noticeable feature about the grey substance at the third month is that the anterior grey horns are distinctly differentiated from the posterior horns, not simply in their general outline, but in their intimate structure. The groups of ganglion cells are now beginning to be distinctly recognisable. Of these, the antero-lateral group is the most advanced in its development. Large, mostly round, cells, with a distinct nucleus, are observed embedded in the embryonic tissue; but the cells have not yet assumed distinct processes. The small internal group is also well represented by several distinct large cells, but the cells are more elongated, and not quite so large or so distinct as in the antero-lateral group. A few cells may be observed in the anterior group. The postero-lateral group is represented by four or five large round cells, but the central group is not yet represented. The areas in which the median and central groups are subsequently developed, and the area which separates the antero-lateral and postero-lateral groups, are composed entirely of embryonic tissue, with small round cells. The vesicular column of Clarke can also be distinguished at this period, by a slight increase in the size of the cells in comparison with those of the surrounding tissue, but the group does not appear in the portion of the cord from which this section was taken.

Fig. 112.

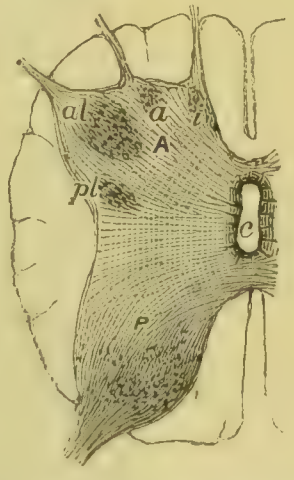
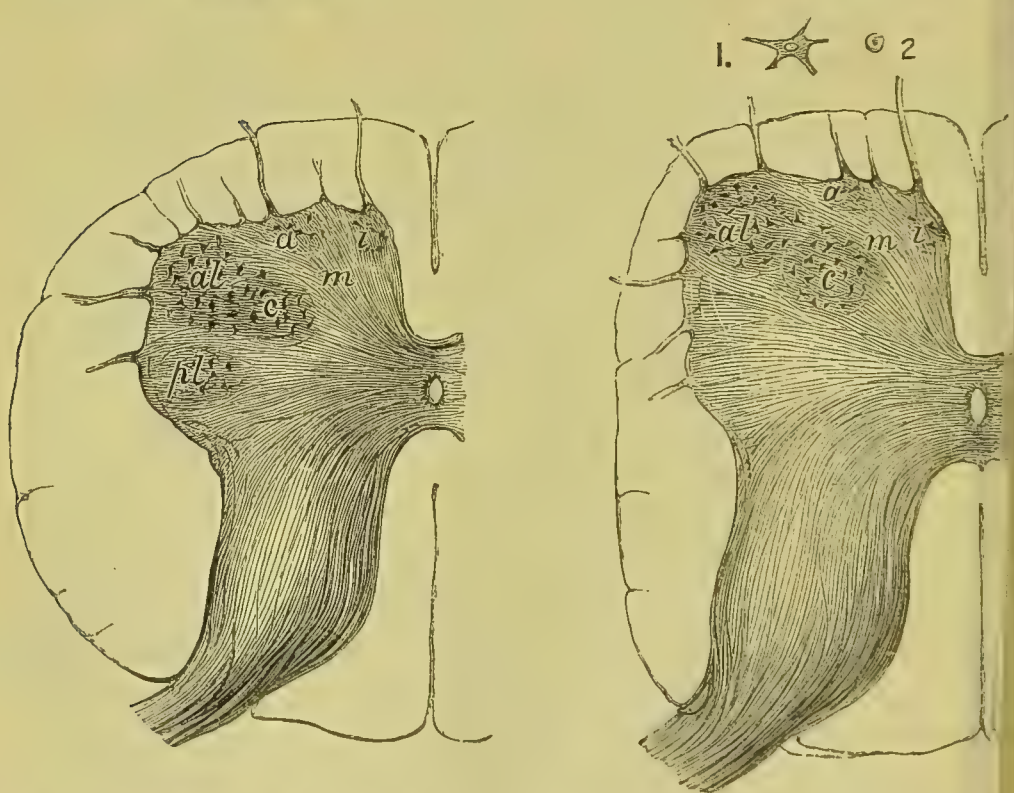


Fig. 112. *Section from the middle of the Cervical Enlargement of the Spinal Cord at the third month of Embryonic Life.*—C, Central canal. The other letters indicate the same as the corresponding letters in *Fig.* 111.

A still further advance in development is recognisable at the end of the fifth month of embryonic life (*Figs.* 113 and 114). The cells of the antero-lateral group have not only increased still further in size, but their processes are now well developed (*Fig.* 114, 1), and each may be seen to lie in a distinct cavity. Those of the anterior and internal groups are also well developed, and the same may be said with respect to the cells of the centre of the postero-lateral group; and even those of the central group are fairly well developed, although only two or three of them have as yet developed processes. The area in which the median group is subsequently developed, and the margins of the postero-lateral and central groups still consist of embryonic tissue. The larger cells of these areas are represented in *Fig.* 114, 2. The section represented in *Fig.* 113 was taken from the middle of the cervical enlargement, and the vesicular column of Clarke is not represented; but the cells of this column are fairly well developed at the fifth month in the lower end of the cervical enlargement and in the dorsal region and upper end of the lumbar enlargement. The section represented in *Fig.* 114 was taken from the middle of the lumbar enlargement, and no trace of the postero-lateral group could be discovered; but in the upper portion of the lumbar enlargement it occupies a similar position to

FIG. 113. FIG. 114.



FIGS. 113 and 114 (Young). *Sections of Spinal Cord of a Five Months Human Embryo*, from the middle of the cervical and lumbar enlargements respectively.—*i*, internal; *a*, anterior; *al*, antero-lateral; *pl*, postero-lateral; *c*, central, *m*, median, and groups of ganglion cells: 1, ganglion cell of the centre of the antero-lateral group; 2, ganglion cell of median group

hat which it occupies in the cervical enlargement, as represented in *Fig.* 113. The vesicular column of Clarke does not appear in the lumbar nlargement.

The ganglion cells of the various groups have become still further deve-oped at the ninth month (*Figs.* 115 and 116); while by the development

FIG. 115.

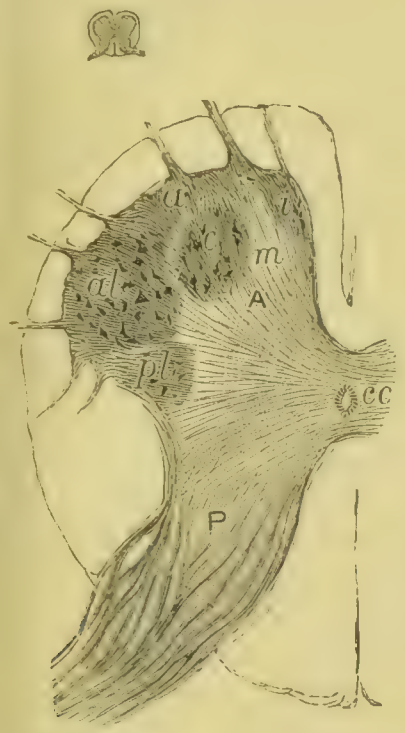
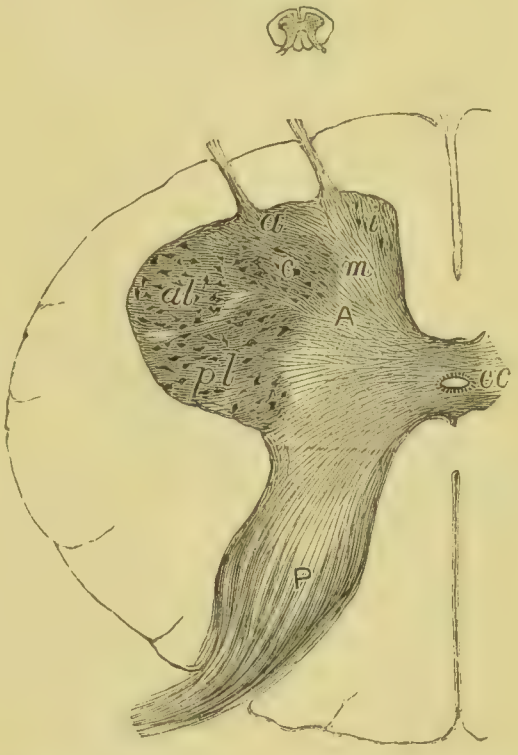


FIG. 116.



FIGS. 115 and 116 (Young). *Sections of Spinal Cord of a Nine Months Human Embryo*, from the middle of the lumbar and cervical enlargements respectively.—A, anterior, and P, posterior horns. The small letters indicate the same as in Figs. 113 and 114. The normal size of the section from which the drawing was made is shown above each figure.

of caudate cells in the central and postero-lateral groups the various groups have became so approximated as not to be so distinctly recognisable from each other as they were at the fifth month of embryonic life. The section represented in *Fig.* 115 was taken from the middle of the lumbar enlargement, and the postero-lateral group is not so well represented as it is in the upper part of the lumbar region. The median area now contains distinct ganglion cells instead of consisting entirely of embryonic tissue. These cells are, however, not much larger than those of the antero-lateral group at the third month; while they are by no means so well developed as those of the latter at the fifth month. The cells of the median group are small, angular masses with a distinct nucleus, but only a relatively small number of these have developed processes. It is not necessary to say much at present with respect to the adult cord. The most noticeable feature in which the cervical and lumbar enlargements of the adult cord differ from the corresponding parts of the cord of a nine

months embryo is in the fact that the ganglion cells of the median group have developed processes like those of the other groups. The cells of the median group, however, especially in the cervical enlargement, are not only much smaller than those of the other groups, but they are much thinner and more transparent. These cells are so transparent that they may be very readily overlooked altogether in sections cleared by oil of cloves, and mounted in Canada balsam; while the cells of the antero-lateral group not only intercept the light, but require considerable change of focus, in order to bring their anterior and posterior surfaces clearly into view.

The relationship which the developing cells bear to the distribution of the blood-vessels is exceedingly interesting. The earlier-developed cells appear to be thrust further and further away from the vessels as development advances. The postero-lateral group, for instance, first shows itself by the development of four or five large cells, which appear about the centre of the spot in which the completed group is subsequently situated; and, as ganglion cell after ganglion cell becomes developed around this centre, the area becomes increased in size by the growth of additional embryonic tissue around the circumference of the group in the part which is in relation with the arterioles (*Fig.* 111). The ganglion cells of the centre of the group are the first to be developed, and the group increases in size by the gradual development of new cells around the central ones. The marginal cells of the group are consequently the last to be developed. Similar remarks apply to the ganglion cells of the central group, as well as to the antero-lateral, anterior, and internal groups, except that the last three groups, instead of being surrounded on all sides by grey substance, are on one of their sides in contact with white substance.

§ 359. *The Accessory Nerve Nuclei of the Spinal Cord.*

(1) *Median Area.*—The comparatively late period in the development of the cord at which the ganglion cells of the median area of the anterior horns assume processes shows that this area must be regarded as an accessory structure (§ 33). The relatively large size of this area in the cervical, as compared with the lumbar enlargement, shows that it is a much more important structure in the former than the latter region. In the fifth month of embryonic life the median area is not larger in the cervical than in the lumbar region, as shown in *Figs.* 113 and 114, where it will be seen that there is scarcely any difference in the general outline of the anterior horns in the sections from the middle of the cervical and lumbar enlargements respectively. In the embryo of the ninth month, however, the median area in the cervical is decidedly larger than in the lumbar enlargement (*Figs.* 115 and 116), and consequently the anterior grey horn in the former region is extended laterally to make room for this area. The relative increase in the size of the median area in the cervical enlargement of the human adult cord, as compared with that of the lumbar enlargement, is still more

narked than in the cord of a nine months embryo, as may be seen in *Figs.* 117 and 118, where the median area occupies a large space, and the ateral outgrowth of the anterior grey horn of the cervical region is very ecided.

On observing the large relative size of the median area in the cervical nlargement of the adult human cord, as compared with that of the lumbar nlargement, and even as compared with that of the cervical enlargement f the cord of the embryo, it occurred to me that this area might not

FIG. 117.

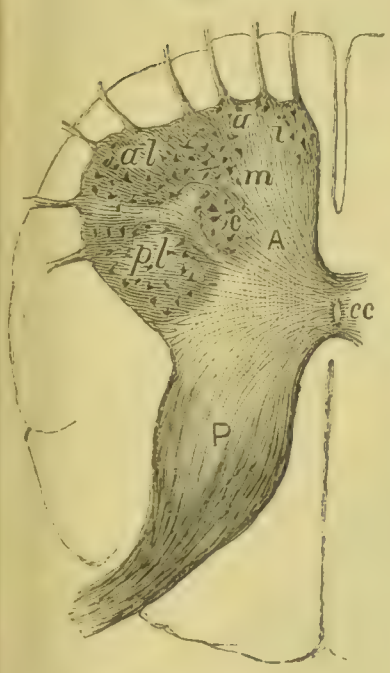
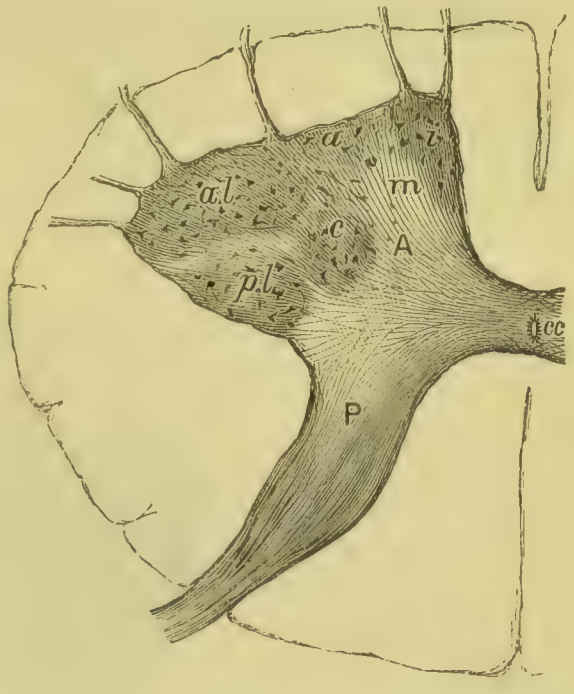


FIG. 118.



FIGS. 117 and 118 (Young). *Sections of the Adult Spinal Cord from the middle of the Lumbar and Cervical Enlargements respectively.*—The letters indicate the same as those in *Figs.* 113 and 114.

possess any relative importance in the cervical enlargement of the spinal cord in animals. In order to test this conclusion I applied to Mr. Larmuth, of the Owens College, whose beautiful sections of the spinal cord are well known in Manchester, and asked him if he would be kind enough to let me have sections of the lumbar and cervical enlargements, as well as from the middle of the dorsal region and the upper portion of the cervical region of the spinal cord of the ox. Mr. Larmuth, in kindly consenting to let me have what I wanted, volunteered the statement that it was quite unnecessary to have a section of both the cervical and lumbar regions, as the two were so alike as to be indistinguishable, and both were like the lumbar enlargement of the human cord. This was, to a large extent, the very fact I was in search of. I have had an opportunity since that time of examining these sections more minutely A section from the cervical enlargement of a calf is represented in *Fig.* 119, and it

will be at once seen that the general outline of the grey substance is very like that of the grey substance of the lumbar enlargement in man, and the median area occupies a still smaller area in the former than in the latter. The median area, indeed, can scarcely be said to exist in the spinal cord of the calf, and this is also true with respect to the cord of the ox.

(2) *The medio-lateral area* lies between the antero-lateral and postero-lateral groups of ganglion cells, and it will be hereafter seen that it is a very important structure in the dorsal and upper cervical regions of the cord (*Figs.* 120 and 121, *ml*). The cells of this area are not well developed at the ninth month of embryonic life in these regions of the cord, and it is entirely unrepresented in the spinal cords of the ox and dog.

FIG. 119.

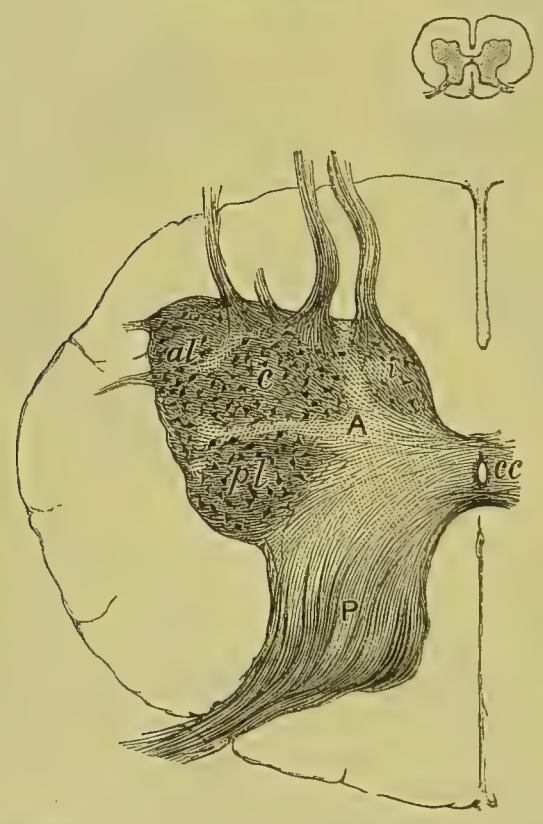


FIG. 119 (Young). *Section of Cervical Enlargement of Calf.*—The letters indicate the same as *Fig.* 113.

We have just noticed that the cells of the median and medio-lateral areas are not only developed at a comparatively late period of embryonic life, but that they are also much smaller in size than those of the other groups of the anterior horns. It might, therefore, be concluded that the size of a ganglion cell may be accepted as a true test of the time at which it began to develop. This test can, however, be relied upon only within certain very narrow limits. The cells at the margins of the poster

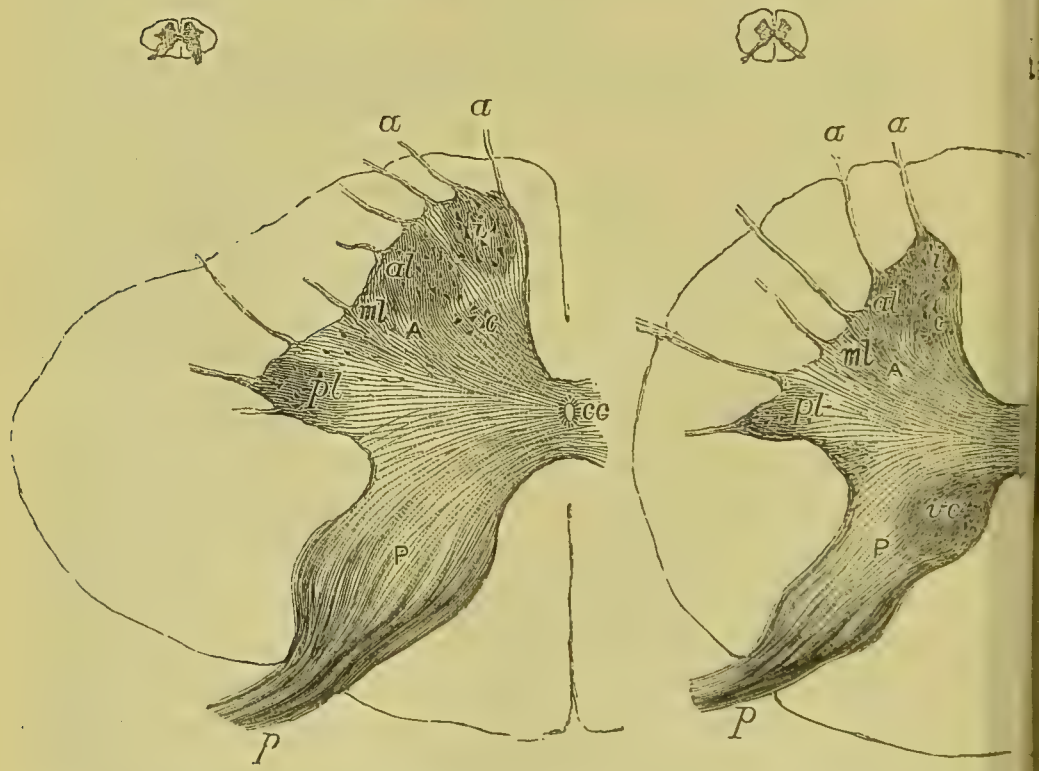
teral group in the lumbar and cervical enlargements are nearly if t quite as large as those of the centre of the group, although the tter began to develop at a much earlier period than the former; while e cells of the nuclei of origin of the third and fourth nerves are small, though they have begun to develop at a comparatively early period. he size of the cell may be accepted as a rough test of its age during the eriod of development, and no longer, just as the size of a growing uman being may be accepted as a rough test of age until the adult ondition is attained, when it ceases to be a test any longer. The ze of the ganglion cells of the anterior horns of the cord of the dult appears to depend mainly if not entirely upon the size of the uscle over whose function it presides; hence the cells of the nuclei of ne third and fourth nerves are small, while the greater number of the ells of the cervical enlargement are large, and those of the lumbar nlargement are still larger. It frequently happens that the later-eveloped cells of the cord are small in the adult condition, but this is ecause the most special muscular adjustments are effected by the con-ractions of small muscles.

§ 360. *Development of the Neuroglia.*

So far we have spoken only of the development of the ganglion ells, but we must now briefly refer to that of the neuroglia. In the arly weeks of fœtal life the neuroglia consists of small round nucleated ells, or rather of a nucleus surrounded by a layer of soft protoplasm, and vith scarcely a trace of basis substance. As development advances, he protoplasm contracts round the nuclei, and the latter become mbedded in a fibrillated, some say granular basis substance. The euroglia becomes denser and more compact in proportion as it acquires nore and more of the basis substance and loses its cellular character. This change does not occur in every part of the grey substance at the ame time. Speaking broadly, the neuroglia assumes a fibrillated texture n the very portions of which the ganglion cells are earliest developed; while it maintains its embryonic condition in the margins of the groups of ganglion cells of the anterior horns and along the line of the blood-vessels. And when a section of the adult cord is held up to the light the groups of large ganglion cells may be seen as dark spots intercepting the light, and strongly contrasting with the transparency of the median area and of the margins of the antero-lateral and postero-lateral groups along the lines of the vessels. The transparent portion also embraces the anterior and posterior grey commissures and the central column of the grey substance as far back as the substantia gelatinosa, with the exception of the area occupied by the vesicular column of Clarke. The transparency of the area just described is no doubt due in some measure to the fact that the small ganglion cells themselves are more transparent than the large ganglion cells, but it is also in great measure due to the loose and spongy character of the neuroglia in the former areas as com-

On passing now to the upper cervical region of the cord, it will be observed that a transposition of the ganglion groups takes place somewhat similar to that which occurs in the dorsal region. Above the level of the fifth cervical vertebra the area of the median group of small cells rapidly diminishes in size, so that the antero-lateral approximates the internal group, and the small anterior, and probably also the central groups disappear. An area of small cells is, however, interposed between the antero-lateral and postero-lateral groups, which begins to show itself as low down as the sixth cervical nerve, and gradually increases in size to the upper end of the cord. There the median group disappears entirely, so that the internal and antero-lateral groups are only separated by a small vessel, while a considerable area of small cells lies between the antero-lateral and postero-lateral groups (*Fig.* 120 *ml*). The

FIG. 120. FIG. 121.



FIGS. 120 and 121 (Young). *Sections of the Adult Human Spinal Cord*, fro the upper cervical and dorsal regions respectively.—A, anterior, and posterior horns; *aa*, anterior roots; *cc*, central canal; *ml*, the medio-later area. The other letters indicate the same as the corresponding ones Figs. 113 and 114. The size of the sections from which the drawing w taken is indicated above each. In Fig. 121, *vc* represents the vesicul column of Clarke.

istribution of the different groups in the upper cervical region s, indeed, very similar to that which occurs in the dorsal region. t will, however, be seen, on comparing *Figs.* 120 and 121, that he central group is unrepresented in the upper cervical, while it s represented in the dorsal region, but this difference is unimportant since I am not sure that the group is always absent in he upper cervical, or always present in the dorsal region. One important difference, however, exists between the dorsal nd upper cervical regions of the cord, and that is the presence f the vesicular column of Clarke in the former and its absence n the latter. The vesicular column of Clarke begins at the pper end of the lumbar enlargement, where it consists of a group of large bipolar cells ; it is continued upwards throughout he whole of the dorsal region, although its cells are considerably smaller here than in the upper lumbar region, and it terminates in the lower part of the cervical enlargement.

§ 364. *The Grey Substance of the Medulla Oblongata, Pons, and Crura Cerebri.*

In the upper segment of the spinal cord both the grey and white substances undergo extensive rearrangement. One important alteration of the white substance is that the column of Goll increases in size by the addition of grey matter—the clavate nucleus—in its interior (*Fig.* 122, G, *cn*), and at a little higher level the posterior root-zone also increases in size by the formation of the triangular nucleus in its substance (*Fig.* 122, *pr, tn*). The increased size of the posterior columns displaces the gelatinous substance of the posterior grey horns (*Fig.* 122, *sg*), so that they extend in a lateral direction instead of posteriorly as in the cord. Another rearrangement of the white substance is produced by the crossing of the lateral columns, so as to form the anterior pyramids of the medulla (*Fig.* 122, *x*, P). These fibres by their crossing cut off the anterior grey horns from the rest of the grey substance, while they thrust the commissures, the central grey column, and the central canal further towards the posterior surface of the medulla. The principal alterations of the grey substance, therefore, consist in the lateral displacement of the posterior horns, the slight posterior displacement of the central canal, central grey column, and commissures, and the

detachment of the anterior horns from the central grey column. A careful examination, however, shows that one or two other minor but exceedingly important alterations have taken place. The triangular nucleus and, at a little higher level, the clavate nucleus (*Fig.* 122, *tn*, *cn*) give off *arcuate fibres*, which are

FIG. 122.

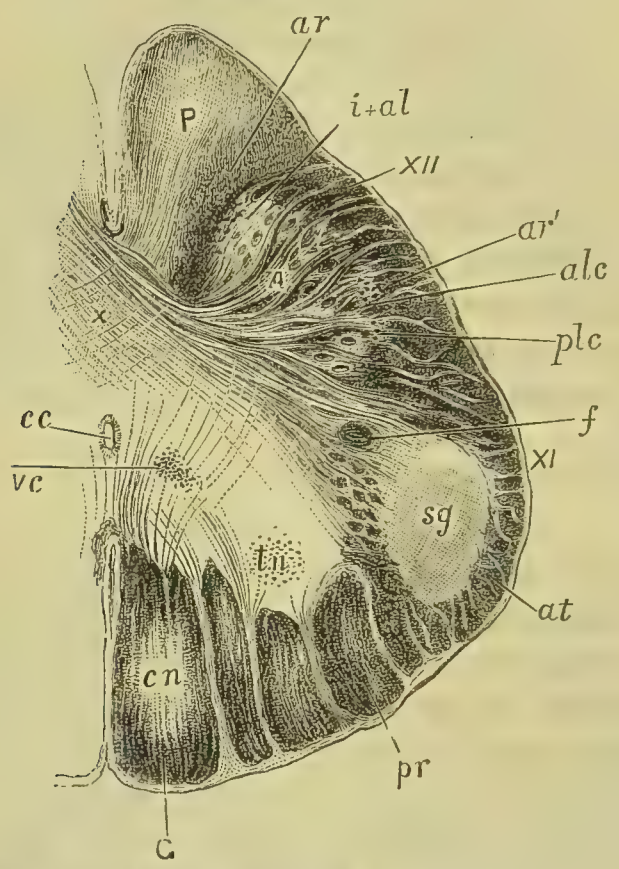


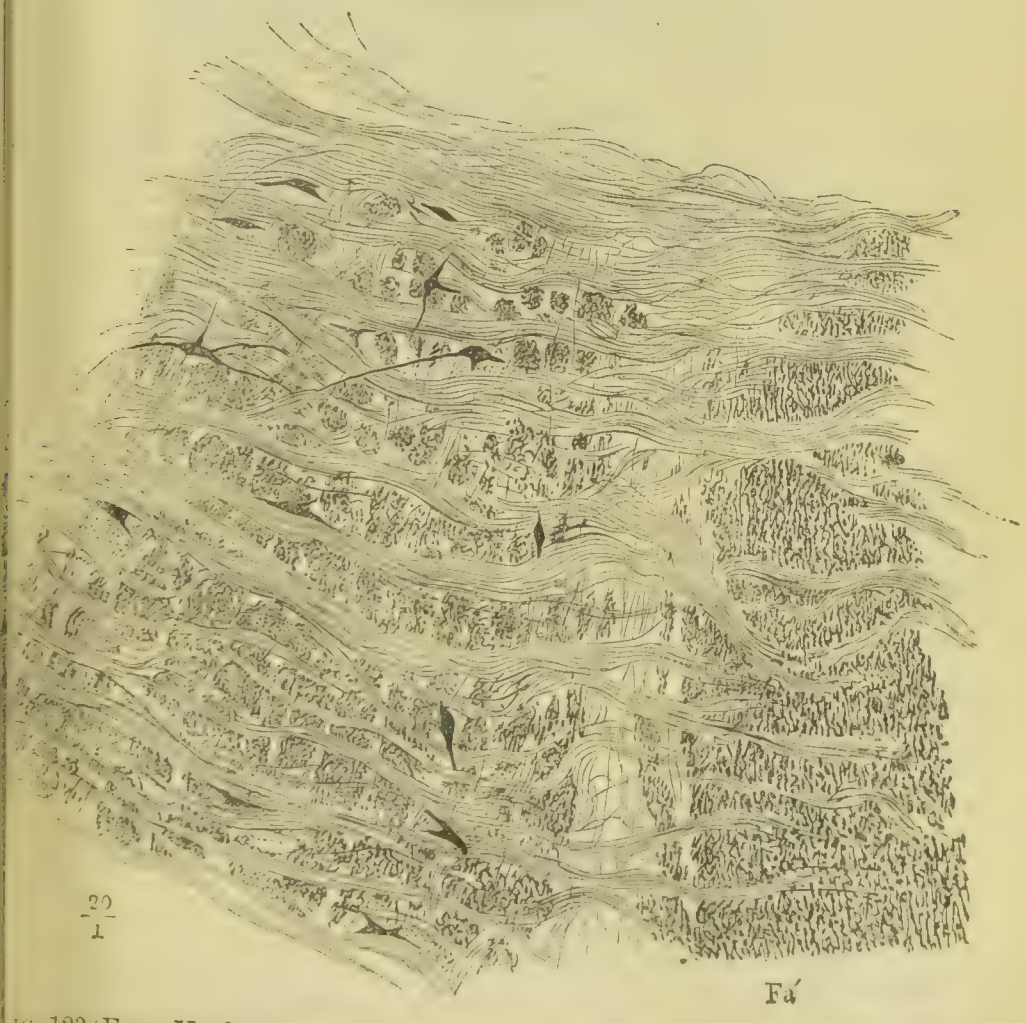
FIG. 122 (Young). *Section of the Lower End of the Medulla Oblongata on a le with the crossing of the fibres of the lateral column.*

A, Anterior grey horns, showing that the grey matter has become mixed up wi the white substance of the anterior root-zone, and with arcuate fibres.
i × *al*, Internal group and a portion of the antero-lateral group.
alc, Anterior nucleus of the lateral column, being a portion detached from t antero-lateral group.
plc, Posterior nucleus of the lateral column, being a portion detached from t postero-lateral group.
sg, Substantia gelatinosa displaced laterally.
at, Ascending root of the trigeminus.
f, Fasciculus rotundus.
vc, Vesicular column of Clarke?
P, Pyramidal tract.
x, Crossing of the fibres.
ar, Internal portion of the anterior root-zone.
ar', External portion of the anterior root-zone.
XII, Hypoglossal nerve.
XI, Spinal accessory nerve.
G, The column of Goll—the slender fasciculus.
cn, The clavate nucleus.
pr, The posterior root-zone—the cuneate fasciculus.
tn, The triangular nucleus. *cc*, The central canal.

rected forwards and upwards in a semicircular manner to ach the olivary body of the same side. These fibres pass rough the posterior horns and thrust them still further in a teral direction, and, indeed, almost entirely separate the eater portion of each horn with its substantia gelatinosa from e grey substance which surrounds the central canal. The cuate fibres interlace with the fibres of the lateral columns the latter bend forwards to cross, and also detach a portion the antero-lateral and postero-lateral groups of cells, so that portion of these groups now extends into the white substance the anterior root-zones (*Fig.* 122, *alc*, *plc*).

The continuation of the anterior root-zones (*Fig.* 122, *ar* d *ar'*) of the cord through the medulla oblongata is broken into a reticulated formation—the *formatio reticularis*—first

FIG. 123.



IG. 123 (From Henle's Anatomie). *Formatio Reticularis of the Medulla Oblongata, showing the ganglion cells distributed through it.*

Fig. 124.

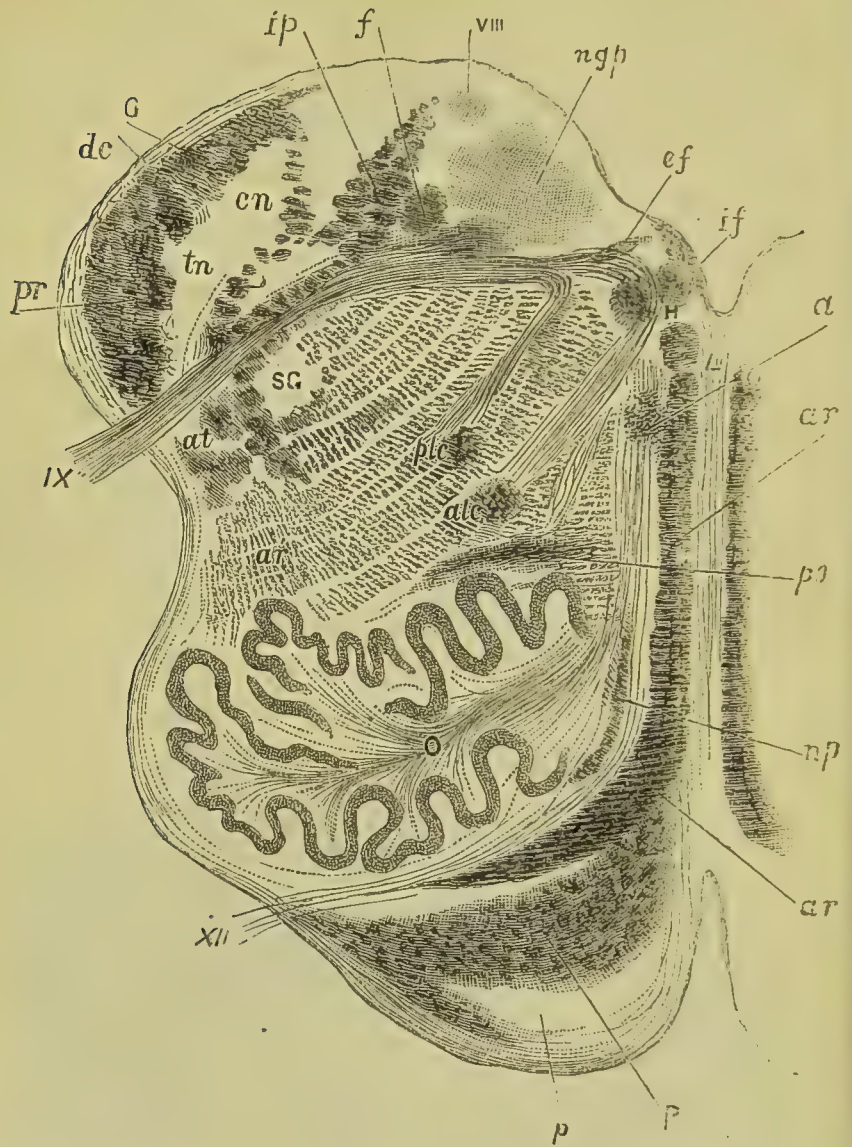


Fig. 124 (Young). *Section of the Medulla Oblongata on a level with the Gl pharyngeal Nerve.*

P, Pyramid.
p, Accessory portion of the pyramid.
XII, Hypoglossal nerve.
H, Nucleus of hypoglossal. The internal, antero-lateral, and postero-lat groups.
a, Anterior group of cells.
IX, Glosso-pharyngeal nerve.
ngp, Nucleus of glosso-pharyngeal.
VIII, Lower part of the posterior median acoustic nucleus.
if, Internal accessory facial nuclei.
ef, External accessory facial nuclei.
alc, Anterior nucleus of the lateral column.
plc, Posterior nucleus of the lateral column.
f, Fasciculus rotundus.
ip, Internal division of the inferior peduncle of the cerebellum.
G, Column of Goll.
cn, Clavate nucleus.

the arcuate fibres of the triangular and clavate nuclei, and
en by the arcuate fibres of the inferior peduncles of the cere-
llum, and the whole of this tissue is thickly studded with
udate ganglion cells, as represented in *Fig.* 123. Whether
l these cells are the representatives of those detached by the
cuate fibres from the antero-lateral and postero-lateral groups
the cord is not known. The cells detached from these
oups, however, aggregate into two more or less distinct groups
the lateral part of the *formatio reticularis* of the medulla.
ıese groups may from their position be called the anterior and
)sterior nuclei of the lateral column of the medulla (*Figs.* 122,
24, and 125, *alc, plc*); while the terms antero-lateral and
)stero-lateral may still be retained to designate what I
:lieve to be the upward continuations of the portions of the
ıtero-lateral and postero-lateral (*Fig.* 109, *al, pl*) groups of
e cord which have retained their connection with the grey
atter that may be considered as representing the anterior
rnua.

§ 365. *Continuation of the Anterior Grey Horns of the pinal Cord through the Medulla Oblongata, Pons, and Crura erebri.*—This slight digression into the examination of the arrangement of the white substance, which takes place in ıssing from the spinal cord to the medulla oblongata, appeared ecessary in order fully to understand the redistribution of ıe groups of ganglion cells occurring in the medulla. At ıe lower end of the medulla portions of the antero-lateral ıd postero-lateral groups may be seen to extend laterally ıto the substance of the anterior root-zone, or into the lateral)lumn of the medulla oblongata as it may now be called.

', Posterior root-zone.
tn, Triangular nucleus.
', The direct cerebellar tract lying on the surface of the posterior root-zone, and the ascending root of the trigeminus.
, Ascending root of the trigeminus.
sg, Substantia gelatinosa.
, Posterior longitudinal fasciculus.
', The portion of the *formatio reticularis*, which represents the *internal* division of the anterior root-zone of the spinal cord.
', The portion of the *formatio reticularis*, which represents the *external* division of the anterior root-zone of the spinal cord.
Olivary body.
np, Nucleus of the pyramid.
po, Parolivary body.

From the anterior nucleus of the lateral column (*Fig.* 122, *alc*) fibres may be observed proceeding inwards and passing betwee the antero-lateral and postero-lateral groups. Some of thes fibres cross over and appear to be connected with the spina accessory nerve of the opposite side. Others wind round th postero-lateral group to get to the spinal accessory nerve the same side. From the posterior nucleus of the lateral colum (*Fig.* 122, *plc*), fibres proceed inwards to reach the grey sul stance, and wind backwards along the boundary line between th white and grey substance to reach the spinal accessory nerve the same side. The nuclei of the lateral column, therefor appear to give origin to some at least of the fibres of th spinal accessory nerve; and we have only to suppose that th same arrangement is carried out as we ascend the medulla an pons in order to understand the origin of the motor fibres the pneumogastric and glosso-pharyngeal nerves, those of large part of the facial nerve, and of the motor root of the fift The arrangement of the fibres from the nuclei of the later column which pass out along with the glosso-pharyngeal nerve represented in *Fig.* 124. The fibres from the anterior nucle (*Fig.* 124, *alc*) proceed backwards and inwards, and pass betwe what will be afterwards described as the antero-lateral a postero-lateral groups. I have not been able to assure myse that any of these fibres cross over to the opposite side, althou this is probable; but some of them may be distinctly observ to wind round the postero-lateral nucleus to proceed in t direction of the glosso-pharyngeal nerve. The fibres from t posterior nucleus (*Fig.* 124, *plc*) proceed backwards and inwar and on reaching the grey substance bend abruptly outwar along the edge of the white substance to reach the nerve. similar arrangement may be observed, at a lower level, wi respect to the pneumogastric and spinal accessory nerves. a higher elevation the fibres from the nuclei of the late column proceed backwards and inwards, the majority of th (genu nervi facialis) wind round the nucleus of the sixth ner and proceed outwards to join the facial nerve. The fibres fr the posterior nucleus of the lateral column (*Fig.* 126, *p* appear to me to pass backwards and to the outside of the nucl of the sixth nerve to join the facial. The anterior nucleus

ıe lateral column appears to terminate on a level with the rigin of the facial nerve. Fibres, however, seem to pass upwards ıd backwards from this nucleus to join the motor root of the fth nerve. In *Fig.* 127 the anterior nucleus of the lateral olumn of the medulla is not represented, but the fibres transersely cut at (*r*) shows that these have joined the others from

FIG. 125.

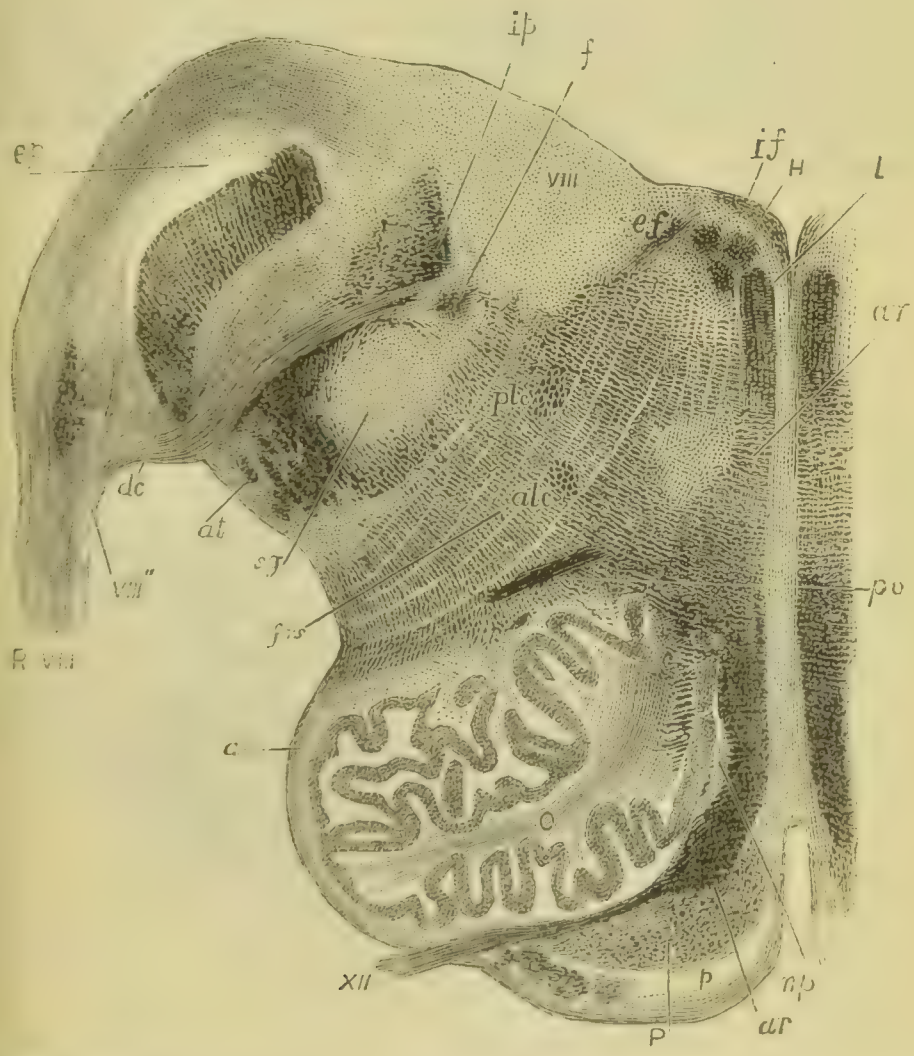


FIG. 125 (Modified from Flechsig). *Section of the Medulla Oblongata on a level with the superficial origin of the Acoustic Nerve.*

R VIII, Root of the acoustic nerve.
VIII, Posterior median acoustic nucleus.
VIII″, Posterior lateral acoustic nucleus.
H, Nucleus of the hypoglossal nerve.
ip, Internal division of the inferior peduncle of the cerebellum.
ep, External division of the inferior peduncle of the cerebellum.
frs, Formatio-reticularis.
a, Arciform fibres. The remaining letters indicate the same as the corresponding letters in Fig. 124.

a different level, and I believe that these fibres have ascended from the anterior nucleus of the lateral column. The motor nucleus of the fifth (*Fig.* 127, v) appears to be the continuation upwards of the posterior nucleus of the lateral column. The nucleus now lies close to the sensory fibres of the nerve, and its fibres, instead of winding backwards at first, as they do at a lower level, appear to pass outwards at once by the side of the sensory fibres.

The groups of cells of the anterior horns may be traced upwards more or less distinctly to the nucleus of origin of the hypoglossal nerve. The hypoglossal nerve begins on a level with the upper limit of the crossing of the fibres of the pyramidal tract. The crossing of the fibres had detached the

FIG. 126.

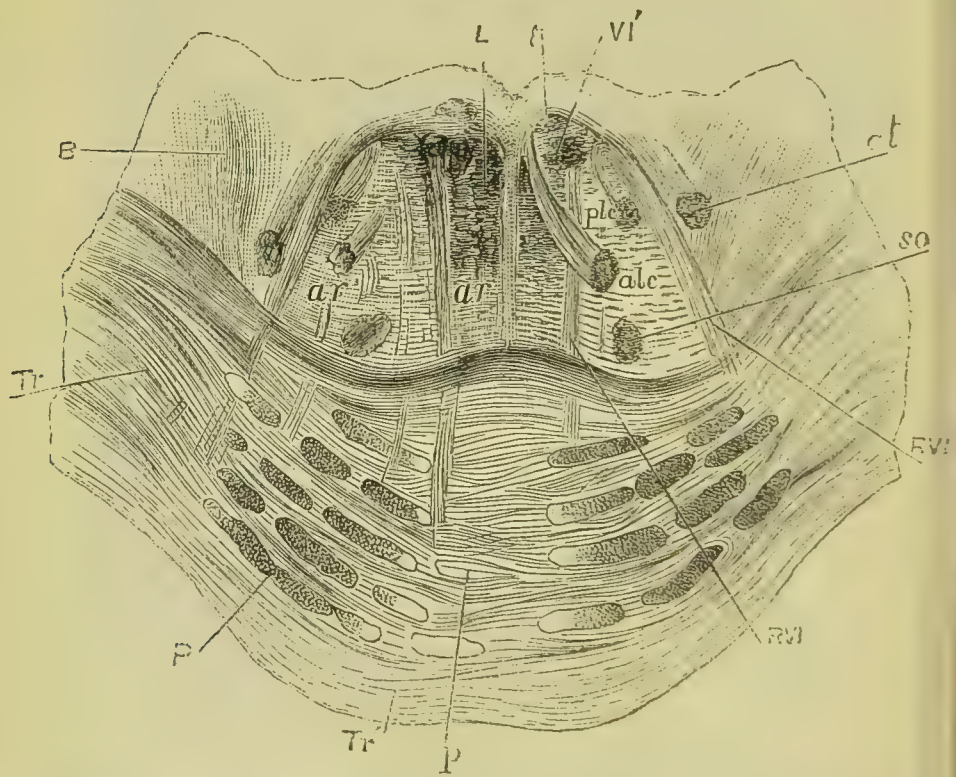


FIG. 126 (Modified from Erb). *Transverse Section of the Pons on a level with the Abducens and Facial Roots, from a nine months embryo.*—The right half represents a section made a little lower than the left. *P*, Pyramidal tract; accessory portion of the pyramidal tract; *Tr* and *Tr'*, transverse fibres of the pons; *so*, superior olivary body; *alc* and *plc*, anterior and posterior nuclei of the lateral column respectively, representing the nucleus of the facial nerve; RV root of the facial nerve; VI', nucleus of the sixth nerve; RVI, root of the sixth nerve; *at*, ascending root of the trigeminus. *B*, The internal division of the peduncle of the cerebellum as it passes from the cerebellum; *L*, posterior longitudinal fasciculus; *ar* and *ar'*, the upward continuation of the internal and external divisions of the anterior root-zone of the spinal cord; *t*, fasciculus te

anterior horns from the grey substance surrounding the central canal; but when the crossing is completed, these two portions again become united. The olivary body is, however, interpolated, the whole of the grey matter is thrust further and further back until the posterior commissure disappears, and the central canal opens at the calamus scriptorius on the floor of the fourth ventricle. The nucleus of the hypoglossal nerve is often described as if its cells constituted one group. These cells are, however, distinctly arranged into several groups which correspond so closely with the arrangement of the groups in the anterior horns of the cord that I have no hesitation in regarding those of the former as continuations of the latter. An

FIG. 127.

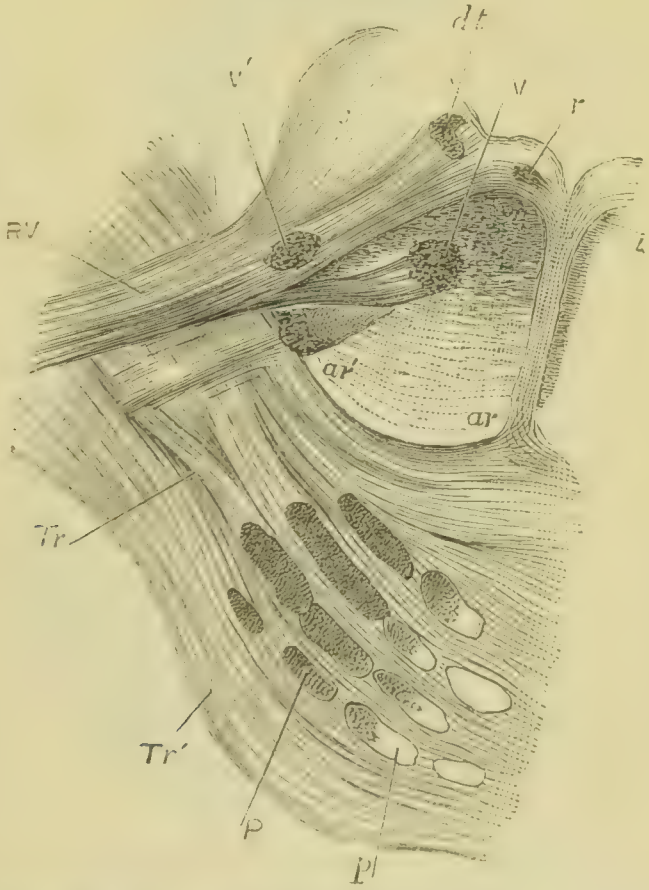


FIG. 127 (Modified from Erb). *Transverse Section of the Pons on a level with the origin of the Trigeminus, from a nine months human embryo.*—*P*, pyramidal tract; *p*, accessory portion of the pyramidal tract; *Tr*, *Tr'*, transverse fibres of the pons; *at*, ascending root of the trigeminus and gelatinous substance; *dt*, descending root of the trigeminus; *r*, root-fibres of the trigeminus cut transversely; V, motor nucleus of the trigeminus; V', middle sensory trigeminal nucleus; RV, root of trigeminus; *C*, roots of the fifth proceeding from the cerebellum; L, Posterior longitudinal fasciculus; *ar* and *ar'*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord.

internal, antero-lateral, and postero-lateral (*Fig.* 109, *i, al, pl*) group may be distinguished, and these appear to correspond to the groups of the same name in the cord; while a large number of cells may be observed at the roots of the hypoglossal nerve (*Fig.* 109, *a*), which may be called the anterior group, and which corresponds to the anterior group in the cord. All that has been previously said with regard to the development of the groups of cells in the anterior horns of the cord applies equally to those of the hypoglossal nucleus. The central cells of the latter groups develop first, while the marginal cells develop last and close to the blood-vessels which ramify between the groups as they do in the cord.

FIG. 128.

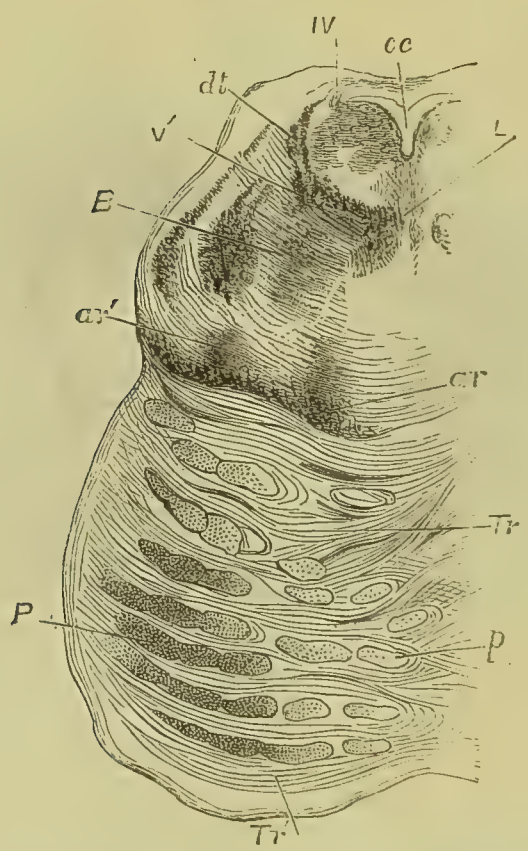


FIG. 128 (Modified from Meynert). *Transverse Section of the Pons on a level with the upper end of the Fourth Ventricle, from a nine months human embryo.*—*P*, pyramidal tract; *p*, accessory portion of the pyramidal tract; *Tr*, *Tr'*, transverse fibres of the pons; *B*, superior brachium of the pons; *L*, posterior longitudinal fasciculus; *ar* and *ar'*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord; V', middle sensory trigeminal nucleus; *dt*, descending root of the trigeminus; IV, nucleus of the fourth nerve; *cc*, aqueduct of Sylvius.

It is not easy to trace the continuation of the groups of cells of the anterior horns of the cord beyond the nucleus of the hypoglossal nerve, inasmuch as the groups become separated longitudinally by the transverse fibres of the pons. It is, however, probable that the nucleus of the sixth nerve (*Fig.* 126, VI′) represents the postero-lateral group, and the bending of the fibres of the facial nerve round the nucleus corresponds to the similar bending of the fibres which issue from the anterior nucleus of the lateral column in the lower part of the medulla round the

FIG. 129.

FIG. 129 (Modified from Krause). *Transverse Section of the Crus Cerebri on a level with the anterior pair of Corpora Quadrigemina, from a nine months embryo.*—*cc*, crusta; *P*, pyramidal tract; *p*, accessory portion of the pyramidal tract; *LN*, locus niger; *RN*, red nucleus of the tegmentum; *L*, posterior longitudinal fasciculus; *ar* and *ar′*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord; III, third nerve; III′, nucleus of the third nerve; IV, fourth nerve; IV′, nucleus of the fourth nerve; IV″, crossing of the fibres of the fourth nerves to opposite sides; *dt*, descending root of the trigeminus; *cc*, aqueduct of Sylvius; *x*, crossing of the fibres of the superior peduncles of the cerebellum; *pf*, fasciculus of medullated fibres proceeding to the anterior pair of corpora quadrigemina.

postero-lateral group to join the spinal accessory nerve. The postero-lateral group cannot be traced beyond the nucleus of the sixth nerve, and probably ceases there. The internal, anterior, and antero-lateral groups are dislocated upwards, as a result probably of the longitudinal extension of the central grey tube, which is rendered necessary in order to provide accommodation for the large mass of the transverse fibres of the pons. These groups reappear in front of the aqueduct of Sylvius, and form the nuclei of the third and fourth nerves (*Fig.* 129, III′, IV′). The fourth nerve is in my opinion merely a portion detached from the third by the decussating fibres of the superior peduncles of the cerebellum, and thus compelled to seek its destination by an independent route. The fourth nerve, therefore, appears to belong to the system of anterior motor nerves represented by the hypoglossal, sixth, and third nerves, and not to the "mixed lateral system" represented by the spinal accessory, vagus, glosso-pharyngeal, and fifth nerves. Although the facial is a purely motor nerve, it appears to belong at least in part to that lateral system. That the nucleus of the sixth on the one hand and that of the third and fourth on the other really belong to the same nucleus, and are only separated from one another by some structure being intercalated in the course of evolution, is rendered probable by the fact that the nucleus of the sixth is connected with a portion of the nucleus of the third of the opposite side by a distinct bundle of fibres (Duval). The fact that these nerves are so closely related in their functions affords further corroborative evidence in favour of this opinion.

§ 366. *Continuation of the Posterior Grey Horns of the Spinal Cord through the Medulla Oblongata, Pons, and Crura Cerebri.*—We have already seen that the substantia gelatinosa of the posterior horns was not only thrust out laterally, but also almost detached from the rest of the grey substance by the arcuate fibres, and we must now observe that it maintains this lateral and superficial position as high as the level of the point of emergence of the fifth nerve (*Figs.* 124 to 127, *at*). It may, indeed, be said that this structure is continued upwards to the level of the opening of the aqueduct of Sylvius into the

third ventricle, since the descending root of the fifth nerve appears to be a somewhat similar structure to the ascending root and gelatinous substance (*Figs.* 127 to 129, *dt*). The white substance of the ascending root appears to be the analogue of the posterior root-zones of the cord—a mere continuation upwards of these zones, after what belongs to the spinal portion of the central grey tube has terminated in the clavate nucleus.

§ 367. *Continuation of the Central Column and the Vesicular Column of Clarke through the Medulla Oblongata.*

In the lower end of the medulla the central column becomes separated from the anterior horn by the decussating pyramidal fibres, and almost separated from the posterior grey horns by the lateral displacement of the latter. A bundle of transverse fibres still connect the central column and the posterior horns, and these separate so as to leave an interspace in which longitudinal fibres may be observed to ascend towards the medulla. These form a round bundle (*Figs.* 122 and 124, *f*), which reaches as far as the upper end of the glosso-pharyngeal nucleus, and has been called the "ascending root of the lateral mixed system" by Meynert, and the "respiratory fascicle" by Krause. In the dorsal region of the spinal cord the middle portion of the grey substance is represented by two columns on each side of the central canal—the vesicular column of Clarke, and the central column—but the column of Clarke is unrepresented in the lumbar and cervical regions of the cord. It appears to me, however, that the vesicular column of Clarke again becomes represented in the lower end of the medulla. A group of cells may be observed near the posterior and internal margin of the central column in the lower end of the medulla (*Fig.* 122, *vc*), corresponding to the position occupied by the vesicular column of Clarke in the dorsal region; and the cells of both groups manifest a tendency to be bipolar instead of multipolar, like those of the anterior horns. Assuming, therefore, that the group of cells in the middle portion of the grey matter in the lower end of the medulla is the upward continuation of the vesicular column of Clarke, and that the remaining portion represents the central column in the cord, we shall have no difficulty in tracing the disposition of these portions of grey substance in the medulla.

Immediately above the crossing of the pyramidal fibres, where the anterior horns are pressed backwards towards the central canal, the central column lies posterior to the groups of cells representing the anterior horns, and close to the central canal, while the representative of the vesicular column of Clarke lies external to the central column, and posterior to the groups representing the anterior grey horns (*Fig.* 130, XI). The nucleus which represents the vesicular column of Clarke contains pigmented bipolar cells, and constitutes the posterior nucleus of the spinal accessory nerve (*Fig.* 130, XI′). And when the central canal has opened into the floor of the fourth

FIG. 130.

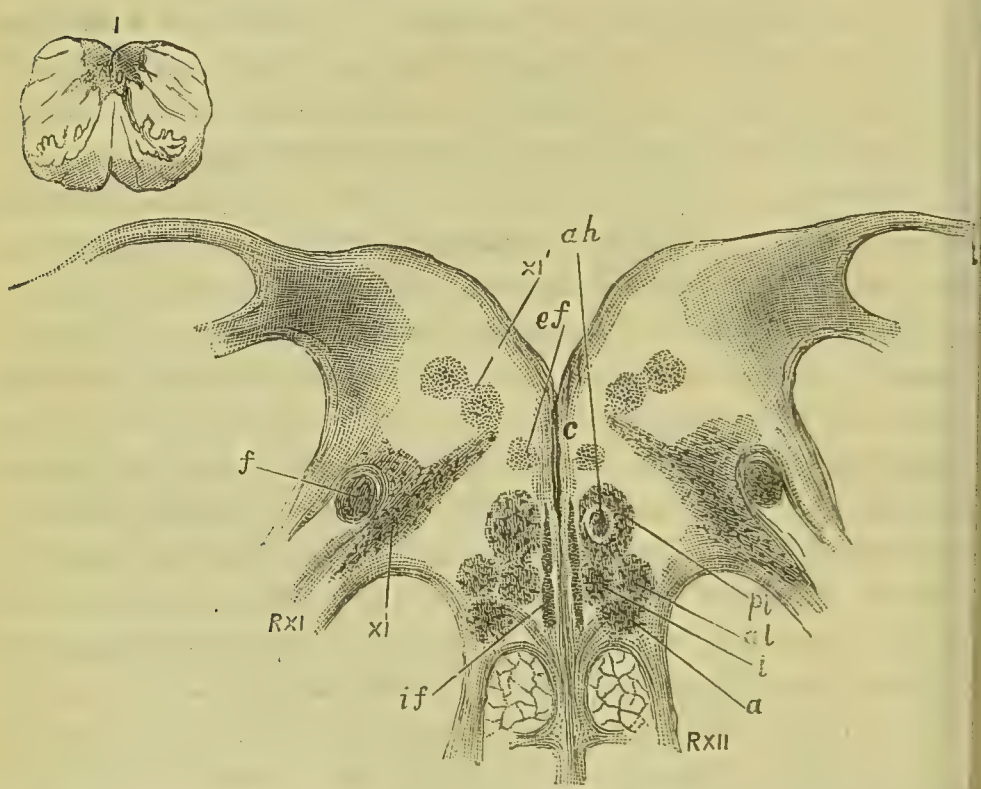


FIG. 130 (Young). *Section of the Medulla Oblongata, a little below the point of t Calamus Scriptorius, showing the groups of cells of the grey substance.*

RXI, Fibres of origin of the eleventh or spinal accessory nerve.
XI, Posterior nucleus of the eleventh nerve.
XI′, Accessory nucleus of the eleventh nerve.
RXII, Fibres of origin of the twelfth or hypoglossal nerve.
a, *i*, *al*, *pl*, Anterior, internal, antero-lateral, and postero-lateral groups c cells respectively.
ah, Accessory hypoglossal nucleus.
if, Internal accessory facial nuclei.
ef External accessory facial nucleus.
C, Central canal.
f, Fasciculus rotundus.

ventricle, the representative of the vesicular column of Clarke is thrust backwards, and laterally so as to form the principal part of the nuclei of origin of the spinal accessory, vagus, and glosso-pharyngeal nerves, while the central column winds round the groups representing the anterior horns (*Fig.* 124, H), so as to lie internal, posterior, and external to them. The posterior portion of the central column is elevated into a ridge (*funiculus teres*) close to the median fissure in the inferior part of the floor of the fourth ventricle (*Fig.* 124, *if*). The central column is continued upwards, as a thin film of grey substance, on the floor of the fourth ventricle, and lying behind the fibres of origin of the facial (*Fig.* 126, *t*) and the fifth (*Fig.* 127, *r*); while in the upper end of the pons and crura it is represented by the grey matter which immediately surrounds the aqueduct of Sylvius (*Figs.* 128 and 129, *cc*).

The characteristics of the central column are, as we have already seen, that its texture is spongy, rendering it transparent in section, and that its cells are comparatively late in their development. We saw reason, indeed, to regard the central column as being the embryonic part of the central grey tube, and that the portions of it which are first developed are thrust outwards as new layers grow about the central canal. If this be true, we may expect to find that any additional nuclei which may form in the medulla oblongata in the course of development will grow in the representative of the central column. This expectation is realised. Whether the spongy portion of grey substance, which lies internal, posterior, and external to the hypoglossal nucleus, be or be not the continuation upwards of the central column, several groups of cells may be observed in it which do not become developed until subsequently to the ninth month of embryonic life, and which do not appear to be represented in the spinal cord; they may, therefore, be called the *accessory nuclei* of the medulla oblongata. These nuclei must be carefully distinguished from the nuclei of origin of the spinal accessory nerve.

§ 368. *Accessory Nuclei of the Medulla Oblongata.*

(1) *Accessory Nuclei of the Facial Nerve.*—The first of these which I shall mention is what has been described by Dr. Lock-

hart Clarke as the inferior facial nucleus. This nucleus consists really of several small nuclei. Two of these, which may be called the internal accessory facial nuclei (*Fig.* 130, *if*), appear as two small round nuclei close to the inner side of the hypoglossal nucleus and the central canal; and when the canal opens on to the floor of the fourth ventricle, they are situated immediately beneath the ependyma of the ventricle, and close to the middle line (*Fig.* 124, *if*). Fibres from these nuclei ascend in the funiculus teres and enter the fasciculus teres (*Fig.* 126, *t*) through which they join the other fibres of the facial nerve.

Another somewhat larger group of small cells is situated at first posterior (*Fig.* 126, *ef*) and then external (*Fig.* 12 *ef*) to the nucleus of the hypoglossal. The fibres which issue from it also join, I believe, the fasciculus teres, and the group may, therefore, be called the external accessory facial nucleus (*Fig.* 109, *ef*). The cells of these nuclei are small, and destitute of processes in a nine months embryo.

(2) *Accessory Nuclei of the Eleventh Nerve.*—Two groups of small cells, which develop at a comparatively late period, may be observed lying behind the posterior nucleus of the eleventh nerve (*Fig.* 130, XI′). Meynert thinks that the cells of these groups are connected with commissural fibres which run behind the central canal, before it opens into the fourth ventricle.

(3) *Accessory Nucleus of the Hypoglossal Nerve.*—The next most important nucleus of this category is one which I have constantly observed in the hypoglossal nucleus of one side only (*Fig.* 130, *ah*). As I have not marked my sections, I am at present unable to say whether it is found on the right or left side. This nucleus is of a round form, and appears as if it were surrounded by a layer of white fibres, arranged longitudinally, which separates it from the surrounding tissue. It contains a large number of very small caudate cells, each being not one-fifth the diameter of the cells of the hypoglossal nucleus. The nucleus in some sections lies between the internal and external convolute of the nucleus of the hypoglossal; while at other times it is embedded in the substance of the internal convolute, being then situated near the margin of the group (*Fig.* 130). This nucleus is almost entirely limited to one side, although faint traces of it may occasion-

e observed in the opposite side; it is scarcely recognisable on ther side of the medulla at the ninth month of embryonic fe. The most reasonable supposition with regard to it is that regulates the movements of articulation, and that it is con-ected with the third left frontal convolution of the brain.

§ 369. *Special Nuclei of the Medulla Oblongata and Pons.*

(1) *The acoustic nuclei* can scarcely be said to be repre-nted by any portion of the grey substance of the cord. These uclei are four in number:—

(*a*) *The posterior median nucleus of the acousticus* (*Fig.* 125, VIII) comes in contact with the nucleus of the agus, but is more superficially situated than that of the tter, and somewhat to the outer side of the glosso-haryngeal nucleus. It occupies the whole space between ne ala cinerea and inferior peduncle of the cerebellum up the anterior border of the striæ medullares. The posterior oot of the acoustic nerve takes its origin chiefly from this ucleus, and passes out partly in superficial fasciculi (striæ oousticæ) and partly through the body of the medulla.

(*b*) *The posterior lateral acoustic nucleus* (*Fig.* 125, VIII″) a grey nodule lying in the peduncle of the cerebellum, etween the deep and superficial fibres of origin of the acoustic erve.

(*c*) *The anterior median acoustic nucleus* belongs to the nterior roots of the acoustic nerve, and is situated anterior the striæ medullares. It occupies the external angle of the urth ventricle, about the middle of the cerebellar peduncle.

(*d*) *The anterior lateral acoustic nucleus* appears like a rolongation of the posterior lateral acoustic nucleus, and is edged in between the middle peduncle and the flocculus. gives origin to the portio intermedia Wrisbergii. Some natomists believe that the fibres which pass in the chorda mpani, and which confer taste on the anterior two-thirds of ne tongue, are derived from the nerve of Wrisberg (Bigelow). is also probable that one of the other nuclei—perhaps the osterior lateral acoustic nucleus—gives origin to the fibres pplied to the labyrinth, and is not connected with the purely oustic fibres.

(2) *The corpora quadrigemina* and geniculate bodies are the nuclei of origin of the second or optic nerve; but we are unable to say, in the present state of our knowledge, what structures constitute the nuclei of origin of the first or olfactory nerve.

§ 370. *Superadded Grey Matter of the Medulla Oblongata and Pons.*

(1) *The Clavate Nucleus.*—The columns of Goll contain in the lower part of the medulla a nucleus of grey matter, which is from its form called the clavate nucleus (*Figs.* 122 and 124, *cn*). It is a longitudinal pillar of grey substance, and produces the enlargement in the fasciculus gracilis, known as the clava.

(2) *The triangular nucleus* (*Figs.* 122 and 124, *tn*) is a grey nucleus enclosed in the cuneate fasciculus, the latter of which is the continuation upwards of the posterior root-zones of the cord. It is a longish grey body on the inner border of the cuneiform column, and enlarging as it ascends. The clavate and triangular nuclei extend to the posterior end of the postero-lateral acoustic nucleus.

(3) *The olivary body* (*Fig.* 124, *o*) is situated in the lateral columns of the medulla, close to the anterior pyramid. In form it is like a bean or an almond, with the hilus directed inwards. It contains a number of small ganglion cells, and is in substance very similar to the corpus dentatum of the cerebellum.

(4) *The parolivary body* (*Fig.* 124, *po*) is a band of grey matter which bounds the internal half of the posterior border of the olivary body.

(5) *The nucleus of the pyramid* (*Fig.* 124, *np*) (internal parolivary body) lies opposite the pyramid, in front and to the inside of the olivary body.

(6) *The superior olivary body* (*Fig.* 126, *so*) is a longish, grey column, situated in the pons in front of the facial nucleus.

(7) *The red nucleus of the tegmentum* (*Fig.* 129, *RN*) of Stilling, or *superior olive* of Luys, is situated in the crus cerebri, between the crus and tegmentum, and is similar in structure to the olivary body.

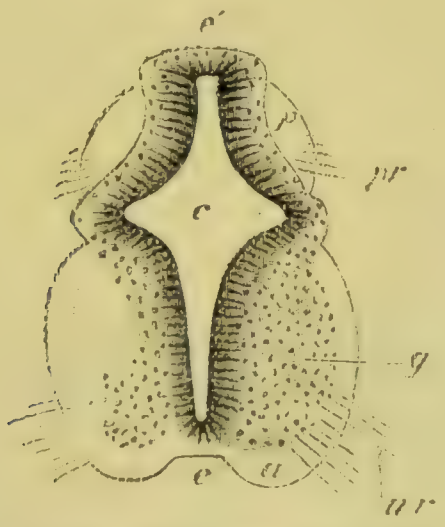
(8) *The middle sensory nucleus of the trigeminus* (*Figs.* 127 and 128, V′) is also a superadded structure. This nucleus is situated in the substance of the afferent fibres of the trigeminus,

ot far from their entrance into the pons. In structure it s somewhat similar to that of the ganglia of the posterior oots, and it may represent the ganglion of the descending oots, while the Gasserian ganglion represents that of the scending roots of the nerve.

§ 371. *Development of the White Substance of the Cord.*

The white substance is formed on the surface of the deeper grey ubstance. Soon after the tube which forms the rudiment of the cord as closed, it is seen to be somewhat oval on section, with a central canal. t this period the cord consists almost entirely of grey matter; and by ie appearance of lateral slits each lateral half becomes imperfectly ivided into two parts, the anterior and posterior. In the human embryo zone of white substance appears towards the end of the first month on ie exterior of each of these parts; and these may respectively be called ie anterior and posterior root-zones (*Fig.* 131, *a*, *p*). The anterior portions

FIG. 131.



IG. 131 (From Kölliker). *Transverse Section of the Cervical Part of the Spinal Cord of a Human Embryo of six weeks.*—*c*, Central canal; *e*, *e'*, Its epithelial lining; *g*, Grey substance; *ar*, Anterior roots; *pr*, Posterior roots; *a*, Anterior root-zones; *p*, Posterior root-zones.

what are afterwards the lateral columns of the cord develop as parts of ie anterior root-zones, but the posterior portions do not begin to develop ıtil about two weeks later. The portions last developed appear to belong the posterior root-zones, and join them in the medulla to form the stiform bodies; and Flechsig thinks that they pass directly to the cortex the cerebellum, hence they may be called the direct cerebellar fibres of ie lateral columns.

At the end of the eighth week, then, the grey substance of the cord in ie human embryo is covered anteriorly, posteriorly, and laterally by a

layer of white substance; but at this period very remarkable changes take place. Two bundles of longitudinal fibres, one for each side, are intercalated between the direct cerebellar fibres of the lateral columns and the posterior horns of grey matter. These bundles on being traced upwards are found to pass forwards at the lower end of the medulla, and after decussating with one another they push aside the anterior columns, and form the inner and larger portion of the anterior pyramids of the medulla; hence the fibres may be called the pyramidal fibres of the lateral columns (*Fig.* 132, *P*, *P'*). About the same time analogous formations appear in the anterior columns, one on each side of the median fissure which separates the anterior root-zones. These bundles are very variable in size and form, but are generally wedge-shaped or elliptical; they form the outer and lesser portion of the anterior pyramids of the medulla, but do not decussate with one another. They are called the columns of Türck or of Lockhart Clarke; and they may also be called the pyramidal fibres of the anterior columns (*Fig.* 132, *T*). At the same period at which these bundles begin to develop, somewhat similar formations appear between the posterior root-zones, one on each side of the posterior median fissure, and these are called the columns of Goll (*Fig.* 132, *G*). The anterior white commissure (*Figs.* 134 to 140, *ac*) also appears at the same time, that is, about the eighth week. A most important point to notice in connection with the development of the white substance is that the fibres when first developed are destitute of a medullary sheath, and only become

FIG. 132.

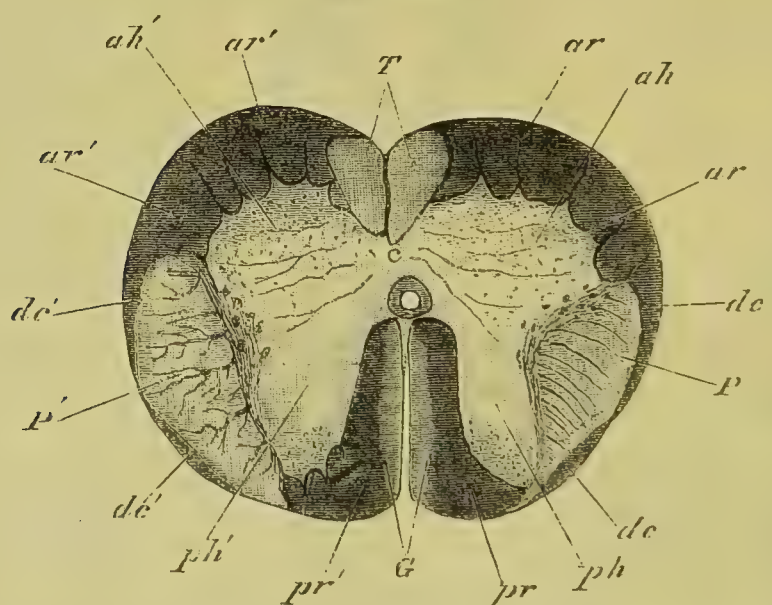


FIG. 132. *Cord of Human Embryo at five months.*—*ah*, *ah'*, anterior horns of gr substance; *ph*, *ph'*, posterior horns of grey substance; *ar*, *ar'*, anterior ro zones; *pr*, *pr'*, posterior root-zones; *P*, *P'*, pyramidal fibres of lateral column *T*, columns of Türck; *G*, columns of Goll; *dc*, *dc'*, direct cerebellar fibre *c*, anterior commissure.

nedullated at a later period of development. The law of development already tated might, indeed, have led us to anticipate that such would be the case. correlated fact is that the fibres of the bundles which are first formed evelop a medullary sheath at a time when the fibres of the later-formed undles are non-medullated. When the cord of a human embryo is xamined at the end of the fifth month it will be found that the pyramidal bres of the lateral columns, the fibres of the columns of Türck and of the olumns of Goll, are non-medullated; while the fibres of the anterior and osterior root-zones, and those of the cerebellar fibres of the lateral olumns, are medullated. When a transverse section of the cord is xamined in glycerine after hardening in chromic acid, the bundles composed of the non-medullated fibres will be found to transmit the light nore readily than those composed of the medullated fibres, so that the ection exhibits the appearances represented (*Fig.* 132) in the cervical egion of the cord of a human embryo at the fifth month. Even when xamined by the naked eye after hardening in chromic acid the bundles mposed of non-medullated fibres are seen to be of a much darker colour nan the bundles constituted of medullated fibres; and the former also ecome much more deeply stained with carmine than the latter. The undles composed of the non-medullated fibres are, indeed, to the naked ye and in their reactions to staining fluids, more like the grey than the hite substance of the adult cord.

The Accessory Portion of the White ubstance.—Inasmuch as the greater art of the fibres of the anterior and osterior root-zones, as well as those the direct cerebellar tract, are medullated as early as the fifth month of mbryonic life, it may be presumed nat all of them are fully developed birth. The case, however, is different with regard to the fibres of the yramidal tract, some of them being nedullated and fully developed at ne ninth month of embryonic life, hile others are not. The fibres of ne columns of Goll are probably also ot all fully developed at birth. The bres of the pyramidal tract in the ord are separated by the septæ of euroglia and the branching vessels into nall lozenge-shaped spaces (*Fig.* 133). he later-formed fibres appear to innuate themselves from above downards along the margins of these paces, so that the earlier-formed fibres

FIG. 133.

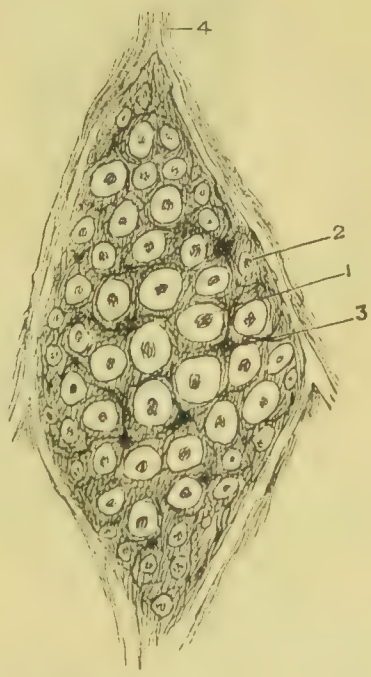


FIG. 133. *Transverse Section of a portion of the Pyramidal Tract magnified.*—1, Fibres of large diameter; 2, fibres of small diameter; 3, Deiter's cells; 4, twig of the median branch of the central artery of the spinal cord.

occupy their centres ; the older being therefore further removed from the blood-vessels than the younger fibres. It may be assumed that the earlier-formed fibres connect the cortex of the brain with the earlier-formed or fundamental ganglion cells of the anterior horns, while the later-formed fibres connect the cortex with the accessory cells. What has already been said with regard to the size of the ganglion cells as a test of the stage of

FIG. 134.

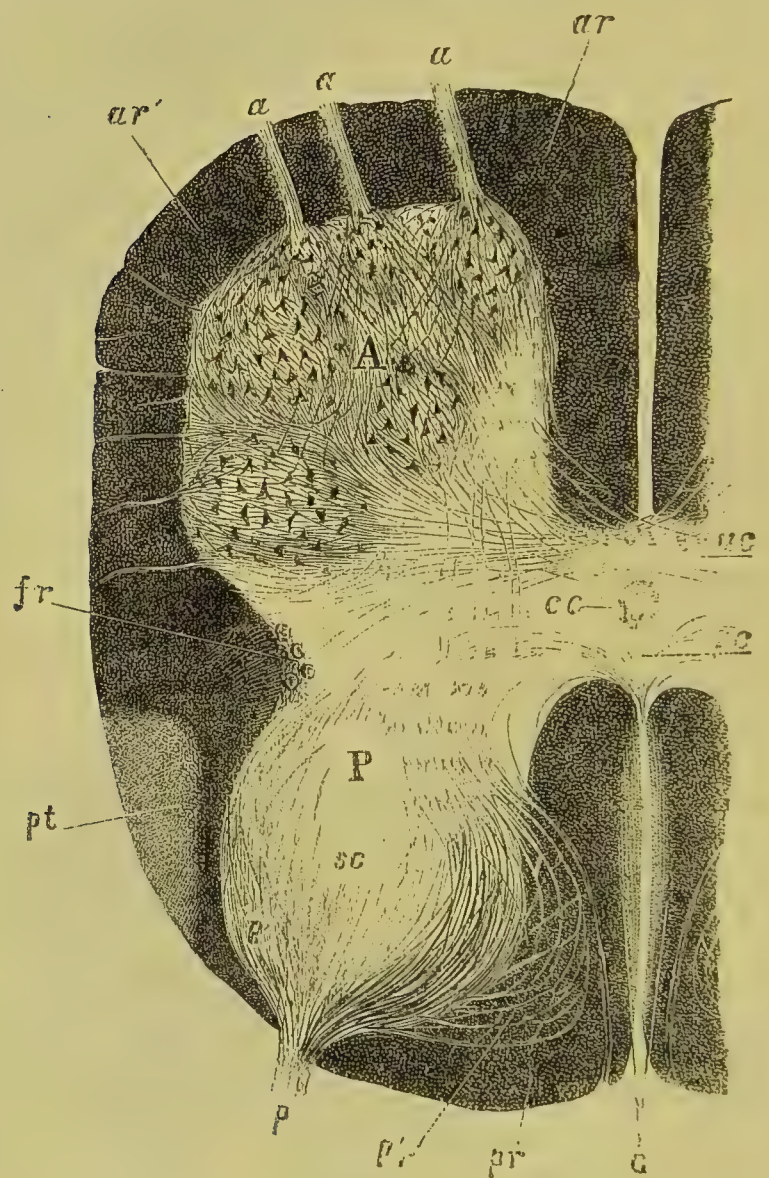


FIG. 134. *Middle of Lumbar Enlargement. Section of Spinal Cord from the middle of the Lumbar Enlargement.*—A P, anterior and posterior grey cornua respectively; *SG*, substantia gelatinosa; *cc*, central canal; *ac*, *pc*, anterior and posterior commissure respectively; G, column of Goll; *pr*, posterior root-zone; *p*, posterior root; *p'*, external radicular fasciculus; *p'r*, internal radicular fasciculus; *a*, *a*, *a*, anterior roots; *ar*, *ar'*, anterior root-zone; *fr*, formatio reticularis; *pt*, pyramidal tract; T, column of Türck.

levelopment of the cell is equally true with respect to the diameter of the nedullated fibres. The diameter of these fibres may be accepted as a ough test of the age of the fibres during the period of development, but o longer. It is very probable that the small medullated fibres of the yramidal tract connect together the small cells of the anterior horns and elatively small cells in the cortex of the brain ; while on the contrary the hick fibres connect the large ganglion cells of the anterior horns and large ells of the cortex. The largest cells of the spinal cord, for instance, are ound in the lumbar region, and the largest in the cortex of the brain in the aracentral lobule—the centre of the movements of the leg—and it is pro-able that these cells are connected with each other by thick fibres. We

FIG. 135.

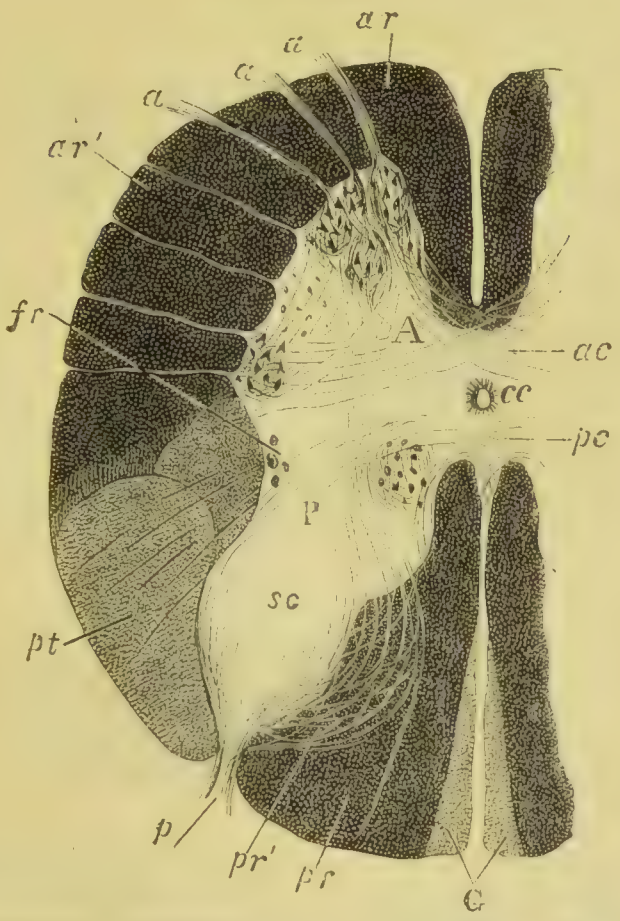


FIG. 135. *Upper end of Lumbar Enlargement.*—The letters indicate the same as the corresponding ones in *Fig.* 134.

ave already seen that, as a rule, the accessory are smaller than the funda-nental ganglion cells of the anterior horns, and it may therefore be in-erred that the accessory fibres of the pyramidal tract are as a rule smaller han the fundamental ones. The smaller fibres are found in greater numbers in the internal and posterior part of the lateral column, the portion of the white column which adjoins the grey substance. At this spot the septa of connective tissue are larger, the neuroglia is more

spongy, and the lozenge-shaped spaces already described (*Fig.* 133) are more distinctly marked than in the more external layers of the white substance. The *formatio reticularis* of the spinal cord appears indeed to owe its structural peculiarities mainly to the fact that it consists in great part of longitudinal fibres of small diameter separated into bundles by comparatively large septa of loose neuroglia. This portion of the cord also transmits fibres which issue from the grey substance to ascend in the pyramidal tract, and from the vesicular column of Clarke to pass out to the direct cerebellar tract. But the longitudinal fibres of small diameter, which are so abundant in this portion of the cord, would appear to belong to the accessory portion of the pyramidal tract. Indeed, the

FIG. 136.

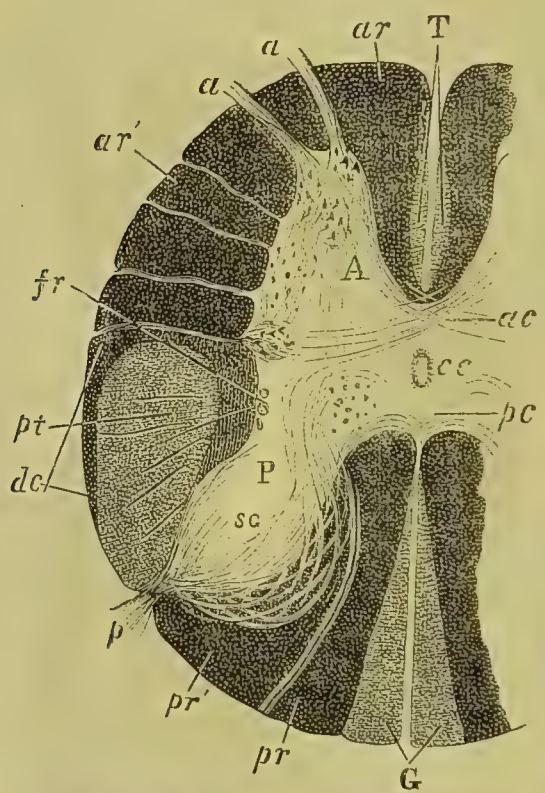


FIG. 136. *Lower end of Dorsal Region.*—T, column of Türck; *dc*, direct cerebellar tract. The other letters indicate the same as the corresponding ones in *Fig.* 134.

spongy character of the neuroglia and the vascularity of this area render it peculiarly adapted for the growth of new fibres. The fibres of the columns of Goll are also separated by the distribution of the blood-vessels and septa of connective tissue into lozenge-shaped spaces. The fibres at the margins of these spaces are not medullated at nine months of embryonic life, and they are as a rule less in diameter in the adult cord than the fibres which occupy the centres of the spaces. These small fibres must therefore be regarded as belonging to the accessory system. The fibres of the posterior root-zones are smaller than those of the anterior and lateral

lumns, with the exception of some of the accessory fibres of the pyramidal act. The reason of this appears to be that the fibres of the posterior ot-zones connect the cells of the posterior horns with each other, and .e latter being themselves small the intercommunicating fibres are also nall.

§ 372. *Longitudinal Distribution of the White Substance.*

These, then, are the component parts of the spinal cord, onsidered with reference to its transverse section; the longi- udinal distribution of these parts must now be described.

Fig. 137.

Fig. 137. *Middle of Dorsal Region.*

The grey matter extends the whole length of the cord, and its ize maintains a constant relation to the number and variety f the movements to be co-ordinated; hence it is larger in the umbar and cervical regions, where the movements of the imbs are co-ordinated. The anterior and posterior root-zones lso extend the whole length of the cord, and, speaking roadly, their size maintains a pretty constant relation to the

size of the grey matter, although there is probably a slight increase of size from below upwards. The most noticeable feature with regard to the remaining bundles of fibres is, that they increase steadily in size from below upwards. The fibres of Goll (*Figs.* 134 to 140, G) extend the whole length of the cord, but they gradually diminish in size from the medulla, so that mere traces of them are to be found in the lumbar region. The pyramidal fibres of the lateral columns (*Figs.* 134 to 140, *pt*) also extend the whole length of the cord, but steadily diminish

Fig. 138.

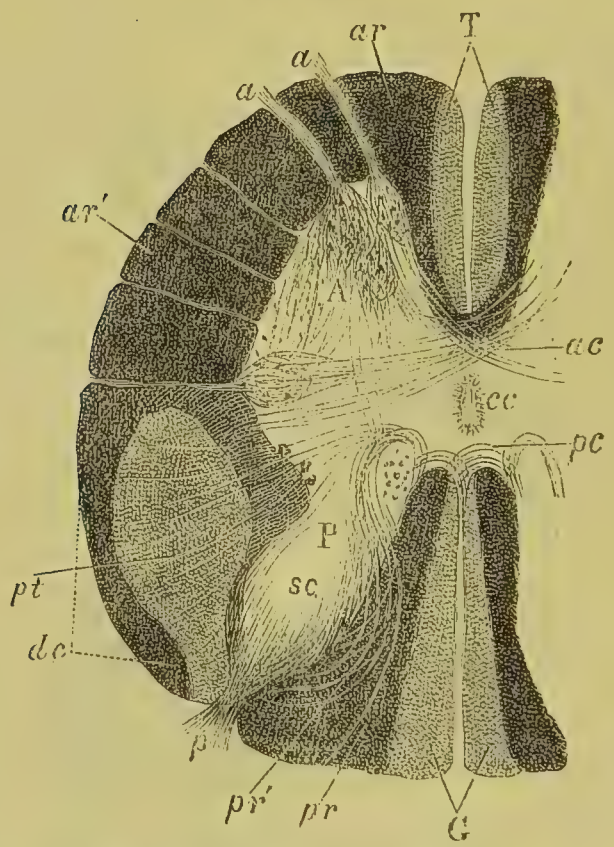


Fig. 138. *Upper end of Dorsal Region.*

in size from above downwards, so that they are reduced to comparatively small bundles in the lumbar region. The direct cerebellar fibres of the lateral columns (*Figs.* 136 to 140, *dc*) appear in the cervical region as thin lamellæ of fibres, one on each side, external to the pyramidal fibres. They diminish in size from above downwards, and disappear somewhat below the middle of the dorsal region, so that in the lower dorsal and

mbar regions the pyramidal fibres come to the surface of the rd. The fibres of Türck (*Figs.* 136 to 140, T) also diminish size from above downwards, and disappear about the middle the dorsal region.

The relative size and position of the different segments of e white substance may be seen in *Figs.* 134 to 140, which

Fig. 139.

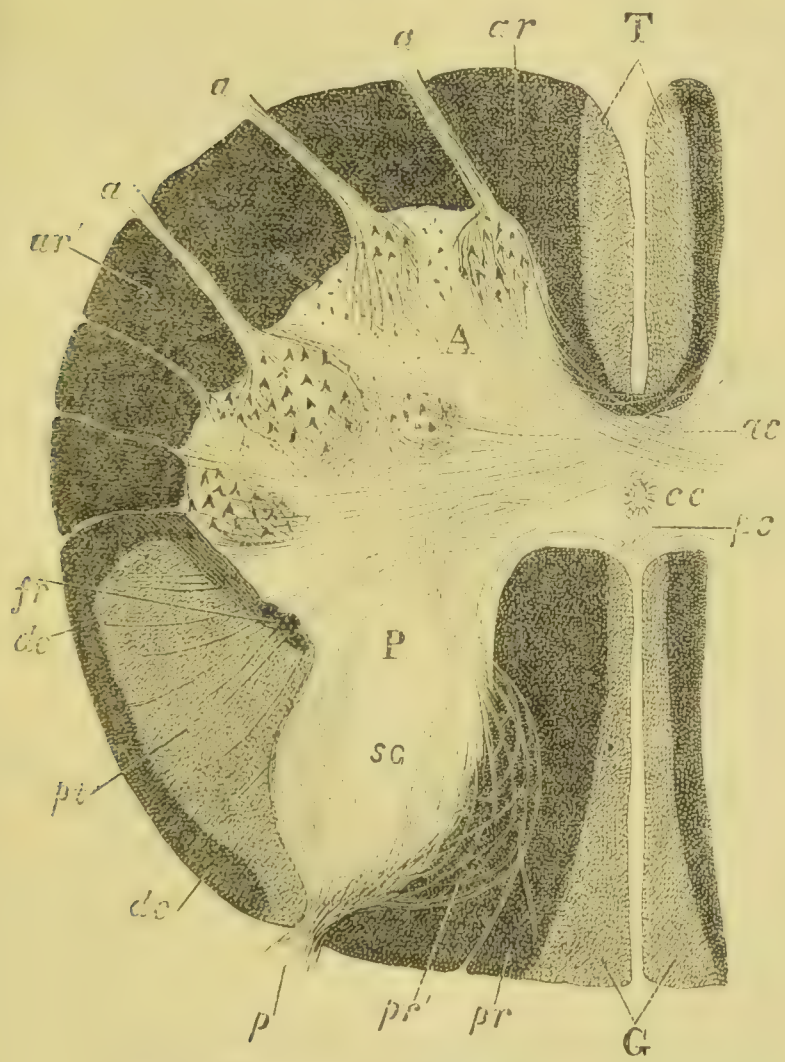


Fig. 139. *Middle of Cervical Enlargement.*

epresent sections of the spinal cord of a nine months human mbryo at different elevations. The fibres of the pyramidal racts (*pt*) of the lateral columns, and of the columns of Goll G) and of Türck (T), have assumed a medulla at the ninth nonth, and are not, therefore, so distinctly marked off from he remaining portions of the white substance as they are

represented in the figures, the latter being in this respect more like the appearances presented by the cord between the fifth and sixth months of embryonic life, at a time when the fibres of the anterior and posterior root-zones and the direct cerebellar tract are alone medullated.

FIG. 140.

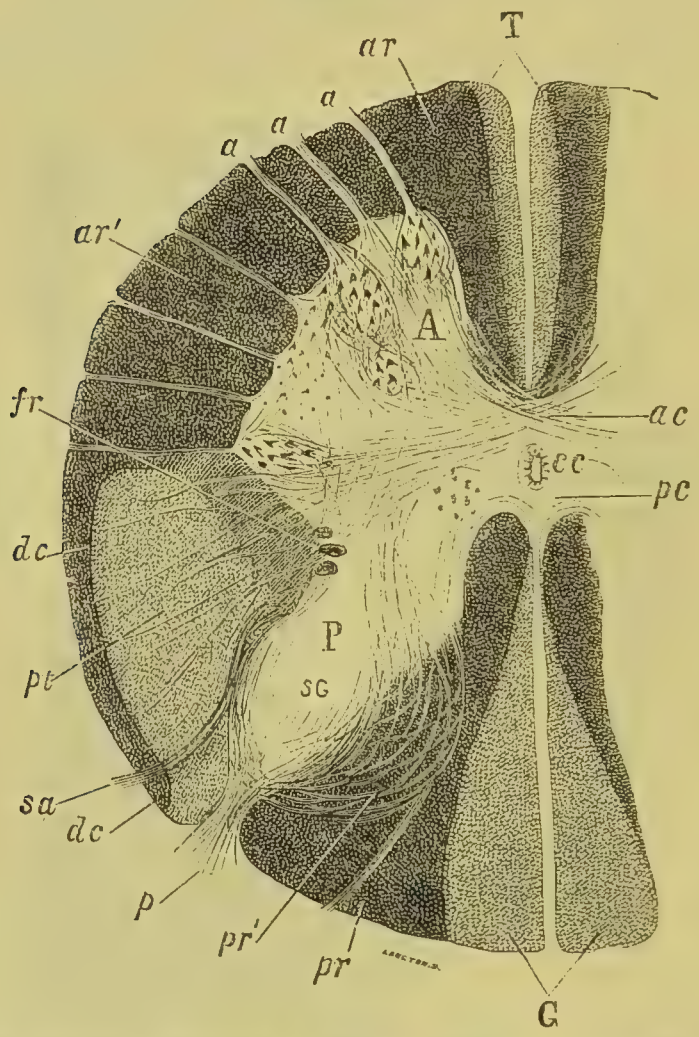


FIG. 140. *Section on a level with the Second Cervical Nerve.*—*sa*, Spinal accessory nerve. The other letters indicate the same as the corresponding ones in *Figs.* 134 and 136.

§ 373. *Continuation upward of the various Segments of the White Substance of the Cord through the Medulla Pons, and Crus Cerebri.*

(1) *Columns of Goll and Posterior Root-zones.*—A transverse section of the lower half of the medulla shows that the columns of Goll are continued upwards into the medulla

the form of two bundles of fibres, one on each side of the sterior median fissure. Each bundle contains a nucleus of ey matter, which from its form is called the clavate nucleus, d the bundle itself is called the pyramidal column, or sciculus gracilis (*Fig.* 122, G, *cn*). External to this fasciculus placed a wedge-shaped bundle, called the fasciculus cuneatus, lding in its interior a grey nucleus, called from its form the angular nucleus (*Fig.* 122, *pr*, *tn*). The greater portion of e fibres of the posterior root-zone of the cord terminates in e cuneate fasciculus and its enclosed grey nucleus. The nder and cuneate fasciculi of the medulla are much larger in e than the column of Goll and posterior root-zone of the rd, owing to the interposition of the grey nuclei; hence the sterior horn of grey matter is displaced outwards and forwards the medulla, so that the continuation of the gelatinous sub-nce forms a mass of grey matter on the lateral aspect of the edulla, known as the grey tubercle of Rolando (*Fig.* 122, *sg*). his mass of grey matter is continued upwards in the medulla d pons to the level of the point of emergence of the fifth rve, and gives origin to the ascending root of the latter. close relationship with the external surface of this grey ass is a bundle, the fibres of which are medullated in a ne months embryo. This bundle is the homologue in the edulla of the posterior root-zone of the cord, and is frequently und diseased in locomotor ataxy (*Figs.* 122 to 127, *at*).

One of the most remarkable rearrangements of fibres in the edulla arises from the fact that the cuneate fasciculus, through e intermediation of its nucleus, resolves itself into arcuate res, which pass forwards and upwards to be connected with e nucleus of the olivary body on the same side; and it is so probable that the slender fasciculus through its nucleus has similar termination.

A transverse section of the upper part of the medulla shows at the fibres have undergone a still further rearrangement, nd that they are greatly reinforced in number; but the course the additional fibres will be more readily traced if we follow hem from the cerebellum to the medulla, instead of from below pwards.

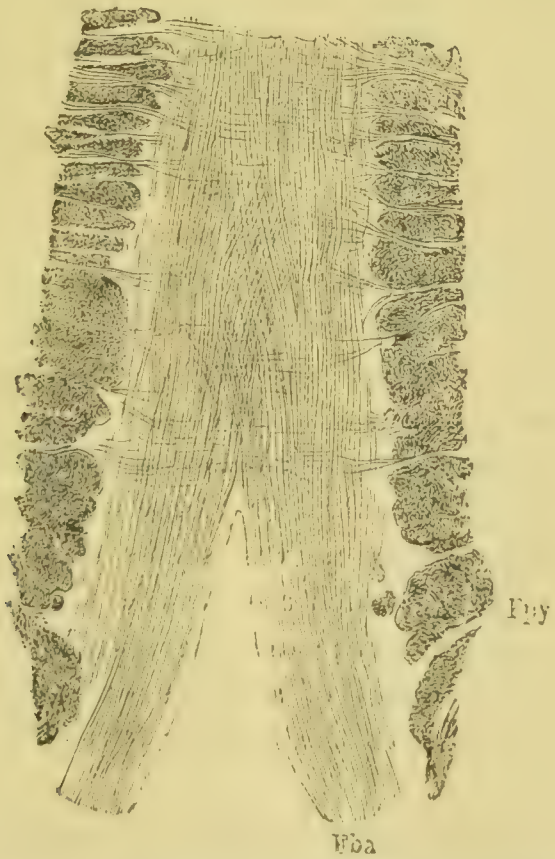
(2) *Connections of the Peduncles of the Cerebellum with*

the Medulla Oblongata, Pons, and Crura Cerebri.—The *ferior peduncle* of the cerebellum, according to Stilling, bre up, on entering the medulla, into an internal (*Fig.* 125, *ep*) a an external (*Fig.* 125, *ip*) division, the latter of which he cal the "restiform body." The fibres of the internal divis spring from the roof-nuclei of Stilling, and on reaching medulla resolve themselves into arcuate fibres, which p downwards and inwards, interlacing with the ascending fib of the anterior root-zone behind the olivary body of the sa side ; and some anatomists believe that they cross the medi raphé to reach the olivary body of the opposite side. T fibres of the restiform body are derived from the cortex of cerebellum, and from a layer of fibres surrounding the dent nucleus ; and this division, on descending to the medulla, s divides into two bundles, which are separated from one anotl by the direct cerebellar fibres of the lateral columns of cord in their ascent towards the cerebellum (*Fig.* 125, *dc*). a nine months human embryo the fibres of the restiform bo are non-medullated (*Fig.* 125, *ep*) ; while those ascending fr the lateral columns are medullated (*Fig.* 125, *dc*), so that the t sets can be readily distinguished from one another. The fib of the restiform body, like those of the internal division of peduncle, resolve themselves into arcuate fibres ; the exter bundle forming the zonular layer which passes in front of olivary body, and the fibres of which reach the median raphé passing both in front and behind the anterior pyramid. Th which pass in front of the anterior pyramid are called *arcifo fibres* (*Fig.* 125, *a*) ; they wind backwards to reach the medi raphé (*Fig.* 141), where, after decussating with the correspo ing fibres of the opposite side, they bend outwards to re the olivary body of the opposite side where they terminate. great part of the arcuate fibres of the internal bundle se to pass through the olivary body of the same side without be connected with its grey substance ; and after gaining the ra they also cross over to pass into the interior of the olivary b of the opposite side, in the grey substance of which all arcuate fibres of the restiform body terminate. The oliv body, therefore, is the medium of communication between cuneate fasciculus and probably also the slender fasciculus

same side on the one hand, and the restiform body and bably the internal division of the cerebellar peduncle of the posite side on the other hand.

The fibres of the *middle peduncle* of the cerebellum are rived from the cortex; they pass in front of and through the bstance of the pons (*Figs.* 126 to 128, *Tr* and *Tr'*), where y separate the ascending fibres of the anterior pyramids o bundles (*Figs.* 126 to 128, *P, p*), and interlace in the ddle line with the fibres of the middle peduncle of the posite side. On reaching the opposite side they are supposed terminate in the cells of interposed grey matter, by means which they are connected with fibres descending from the sta. The close relationship of the middle peduncles with lateral lobes of the cerebellum is well illustrated by the t that in those animals in which the latter are deficient or ent the transverse fibres of the pons are few or entirely nting.

Fig 141.



G. 141 (From Henle's "Anatomie"). *Diagram of a horizontal section of the anterior part of the median raphé of the Medulla Oblongata.*—Fpy, Anterior pyramid; Fba, Fibræ arciformes.

The fibres of the *superior peduncles* are derived from the dentate nuclei; they decussate with one another in the tegmentum, the fibres of one side passing over to be connected with the red nucleus of the opposite side (*Fig.* 129, *x*). The fibres of the superior peduncles are medullated in a nine months embyro; they may be seen surrounding, and even in, the substance of the red nucleus (*Fig.* 129, *RN*), and a considerable proportion of them pass upwards uninterruptedly into the tegmental portion of the internal capsule, and either end in the inferior or external surface of the thalamus, or, as I am inclined to believe, pass uninterruptedly along its external border upwards to be connected with the central convolutions of the cortex of the cerebrum.

Some anatomists think that part of the fibres of the anterior root-zones pass through the crusta to join the lenticular nuclei; but a very important fact has been ascertained by Flechsig which renders this doubtful. Flechsig found that in a nine months human embryo the pyramidal fibres in the crusta are the only ones which have acquired a medullary sheath; and my own sections confirm this (*Fig.* 129, *P*). But the fibres of the anterior root-zones in the cord are medullated at a very early period of development, and long before the pyramidal fibres have acquired a medullary sheath; hence it may be inferred that none of the fibres of the anterior root-zones pass up into the crusta or motor tract of the crura, although it is very probable that new fibres become developed, which connect the corpora striata and the cord, and that these pass through the crusta and become mixed with the fibres of the anterior root-zones. The close connection which is maintained between the anterior root-zones and that portion of the central grey tube which is in immediate relation with the efferent nerves, seems to indicate that the former consist of fibres which co-ordinate the various segments of the cord longitudinally; and there are other grounds for believing them to consist of a series of looped fibres which originate and terminate in the anterior part of the central grey tube.

(3) The *direct cerebellar* fibres are represented by a thin lamella of longitudinal fibres lying on the surface of the cuneate fasciculus and of the grey tubercle of Rolando (*Figs.* 124 and

, *dc*). They pass upwards to the cortex, and thus form an nterrupted connection between its grey matter and the cord, ere the fibres are supposed to pass inwards between the idles of the pyramidal fibres of the lateral columns, to ninate in the cells of the group known as Clarke's column. eir function, however, is not yet ascertained.

4) *The Pyramidal Tract.*—The pyramidal fibres of the eral columns at the upper end of the cervical region of the l pass forwards and inwards towards the anterior median ure. These fibres decussate with one another in the dulla, so that those of the right side pass to the left, and se of the left to the right. The decussation frequently begins the upper portion of the cord; while the homologues of pyramidal fibres, which arise from the nerve-nuclei of the oglossal and facial nerves, cross separately in the pons ve the decussation of the pyramids. The pyramidal fibres he lateral columns during and subsequent to their decus- on come forwards into the anterior median fissure, and h aside the columns of Türck (*Fig.* 140, T), so that the latter n a prismatic bundle of fibres external to the former, and nd without decussating with one another. These two sets bres constitute the anterior pyramids of the medulla (*Figs.* to 125, P); they can be traced through the pons (*Figs.* 126 28, P), where they receive a large accession to their size, the peduncles of the cerebrum. According to the researches Flechsig, which my own sections confirm, the pyramidal es, after being separated into distinct bundles in the s, come together so as to form one compact bundle in peduncle (*Fig.* 129, P). This bundle occupies about the dle third of the crust of the cerebral peduncle, and, rary to what has hitherto been believed, it passes into posterior segment of the internal capsule, lying between lenticular nucleus and optic thalamus opposite the middle d of the latter. The pyramidal bundle is separated from caudate nucleus by a layer of fibres, which ascend from the rnal surface of the optic thalamus to reach the corona ata, while it rests on the three successive segments of the icular nucleus, and reaches the corona radiata opposite the d quarter of the caudate nucleus (reckoning from before

backwards). Having emerged from between the basal gangl without anywhere communicating with them, the fibres of t pyramidal bundle radiate in all directions towards the surfa of the cerebrum, and are mainly distributed to the centr convolutions about the sulcus of Rolando, the so-called "mot area" of the cortex (*Fig.* 142, *P″*).

FIG. 142.

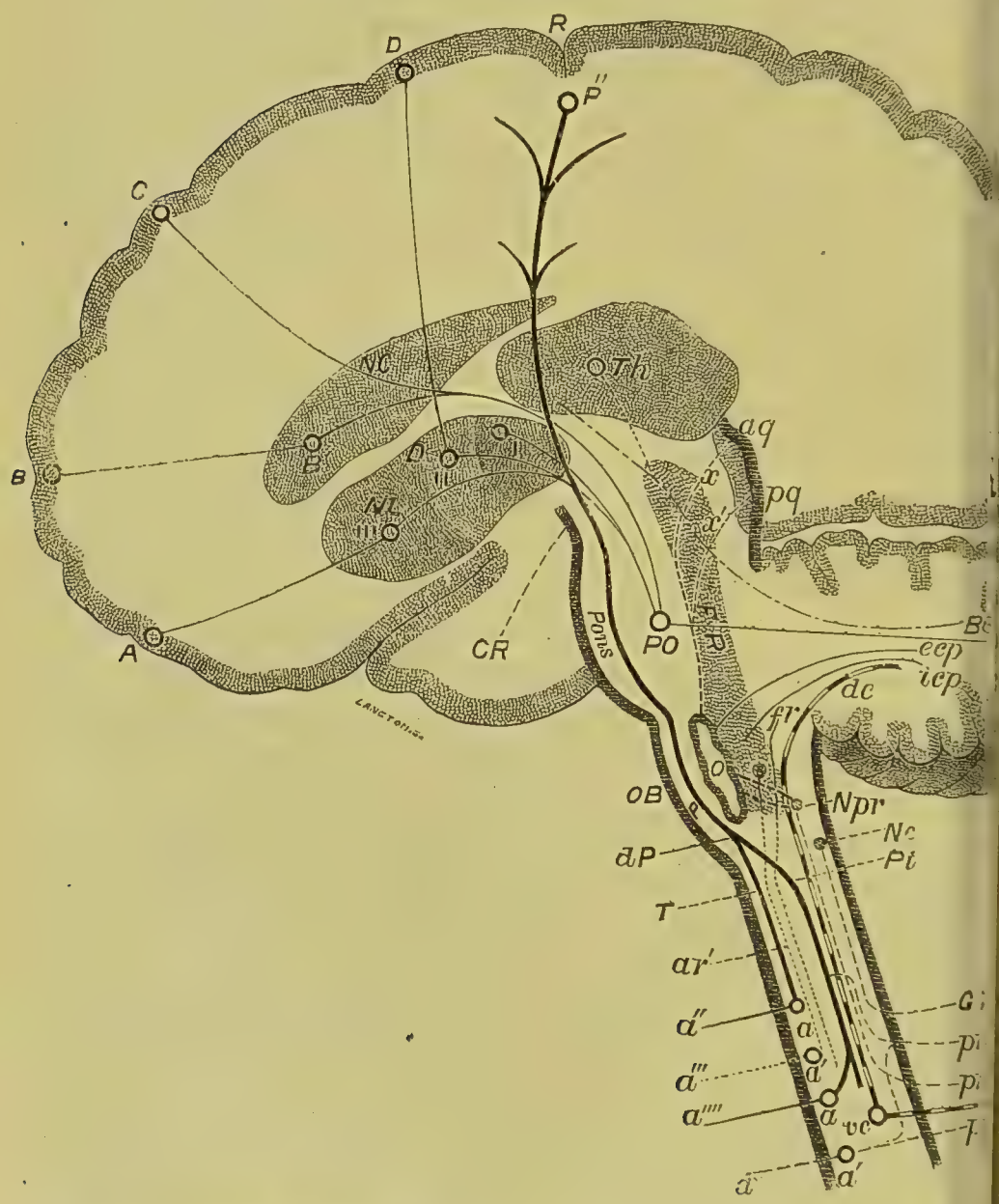


FIG. 142 (After Flechsig). *Diagram of the Grey Masses of the Spinal Cord Brain, showing the course of the Conducting Paths.*

R, Fissure of Rolando.
P″ P, *T* and *Pt*, Course of the fibres of the pyramidal tract from origin in the central convolutions to their termination in the anterior horns (*a*, *a′*).

The cardinal facts which concern us at present are, that res issue from the central convolutions of the cerebrum, ich pass through the internal capsules without communicating h the basal ganglia; that the same fibres pass through the ebral peduncles to enter the pons, where they at once begin diminish in number. The fibres of this kind, which pass ough the pons, collect together to form the anterior pyramids the medulla, which also diminish in size from above downrds, showing that some of these fibres are lost in the medulla elf. The internal and by far the larger portion of the ramids decussate with one another, and these portions pass ckwards so as to form in the cord the bundles of pyramidal res in the lateral columns—bundles which extend the whole gth of the cord, but gradually diminish from above downrds. The external and lesser portion of the pyramids pass ectly downwards to form the columns of Türck—columns ich dwindle gradually until they disappear, usually about the ddle of the dorsal region. It is not yet proved anatomically w these fibres end in the cord; but other considerations der it probable that they end in the grey matter of the terior horns and its continuation through the medulla, pons, d around the aqueduct of Sylvius. The pyramidal fibres, in e word, form an uninterrupted connection between the central nvolutions of the brain and the central grey tube of the rd.

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

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

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

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

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

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

dP, Decussation of the pyramids.

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

a, a', a'', a''', a'''', Anterior roots.

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

The Accessory Portion of the Pyramidal Tract.—We have seen that the accessory fibres of the pyramidal tract occupy the margins of the lozenge-shaped spaces into which the lateral column and column of Türck are divided (*Fig.* 133), and that they are very abundant in the portion of the lateral column which adjoins the grey substance, and especially in the *formatio reticularis*. But on ascending to the anterior pyramid of the medulla the accessory fibres become much more abundant, and

FIG. 143.

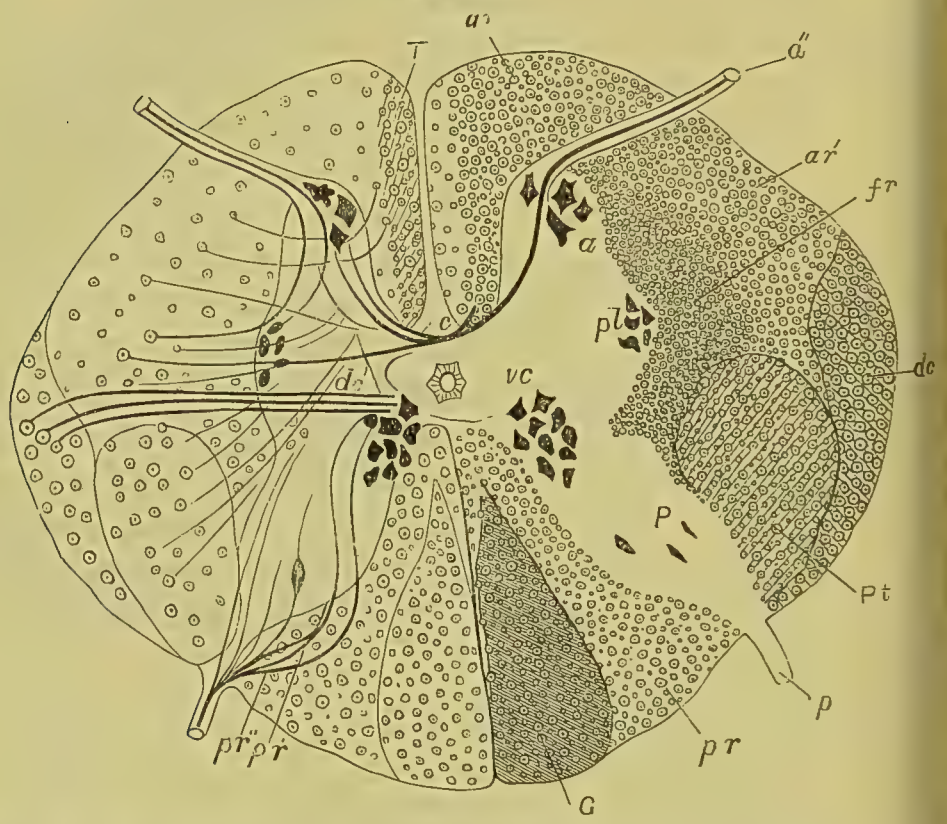


FIG. 143 (After Flechsig). *Diagram of Transverse Section of the Spinal Cord in upper half of the Dorsal Region.*

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

FIGS. 142 and 143 (After Flechsig).—Letters common to *Figs.* 141 and 142.

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

ıough some of them may still mix with the other fibres, y aggregate in the internal and anterior margin of the amid, so as to occupy a circumscribed area of the pyramid hout admixture with other fibres. This area is shown in *rs.* 124 and 125, *p*, which represent sections of the medulla of ine months embryo. On passing through the pons the non-dullated fibres occupy the inner portion of the longitudinal ɔiculi (*Figs.* 126 to 128, *p*), pass to the inner side of the dullated fibres in the crust of the crus cerebri (*Fig.* 129, *p*), l reach the cortex mainly by passing through the anterior f of the internal capsule.

5) *The Anterior Root-zones.*—The continuation of the erior root-zones through the medulla, pons, and crus, erves special attention. The course of the fibres of these es in the medulla is obscured by the fact that they do form a defined mass, as in the cord. They are separated) bundles by the arcuate fibres of the medulla, so as to n what is called from its reticular appearance the *formatio cularis* (*Fig.* 125, *frs*). The zones consist of two portions, *internal*, which lies between the anterior median fissure l the anterior roots (*Figs.* 134 to 140, *ar*), and an *external*, sisting of the remaining portion (*Figs.* 134 to 140, *ar'*). e *internal* portions of the anterior root-zones are pushed le in the lower part of the medulla by the decussating fibres the pyramidal tract, but above the level of the decussation, ere the olivary body is intercalated, the internal portion thrust backwards behind the pyramids and close to the dian raphé, while the fibres of the hypoglossal nerve separate internal from the external portion of the anterior root-zone. the spinal cord the internal portion of the anterior root-zone intains a close relation to the internal group of ganglion s, and this relation is apparently maintained throughout its rse in the medulla, pons, and crus. The portion which is led the *posterior longitudinal fasciculus* in the medulla *igs.* 124 to 128, *L*), pons, and crus, appears to be the con-uation upwards of the part of the internal portion of the terior root-zone which adjoins the grey matter, and this ɔiculus always lies to the inner side of the roots of the terior motor nerves, at their origin in the motor ganglion

cells. The upward continuation of the part of the internal portion of the anterior root-zone which is remote from the grey matter is represented by *ar* in *Figs.* 124 to 129.

In the crus the posterior longitudinal fasciculus is situated in front of the aqueduct of Sylvius, in close relationship with the nucleus of origin of the third nerve (*Fig.* 129, *L*). A portion of this bundle is continued forwards in the thalamus in the walls of the lateral ventricle, while the remaining fibres bend backwards to join the posterior commissure of the third ventricle. The fibres of the latter portion are the first to become medullated in the cerebrum of the human embryo.

The external portion of the anterior root-zone of the cord is continued upwards into the formatio reticularis of the medulla (*Figs.* 124 to 128, *ar'*). The continuation of the external portion of the anterior root-zone lies behind the olivary body, and comes to the surface of the medulla in its lateral column. It is bounded internally by the root fibres of the anterior motor nerves, and externally by the root fibres of the nerves of the lateral mixed system, and posteriorly by grey matter. The interlacing fibres of the pons (*Figs.* 126 to 128, *Tr*) pass in front of this portion (*ar'*), while in the crus the latter comes again further forwards, the locus niger lying between it and the crust (*Fig.* 129, *ar'*).

(III).—FUNCTIONS OF THE SPINAL CORD AND MEDULLA OBLONGATA.

It would occupy too much space to describe fully the functions of the spinal cord and medulla oblongata, and the reader is referred to physiological manuals for the usual information on the subject. My main object at present is to elicit a few points which will be of subsequent use to us in interpreting the phenomena of disease, and in connecting symptoms with morbid alterations of structure.

§ 374. *Voluntary Action.*—The special functions of the cord are those by which the spinal centres are subordinated to the motor centres of the cortex of the brain. It is probable that all the spinal centres are connected with the motor centres of the cortex of the brain, or are, in other words, under voluntary

trol; but the later-acquired movements of man are more roughly under voluntary guidance than the earlier-acquired undamental actions. Inasmuch as the observation of the elopment of the cord has enabled us to draw a broad dis-tion between the fundamental and accessory portions of the icture of the spinal cord, it will be well to endeavour first to nect the later-acquired or accessory functions with the later-uired or accessory structure. The earlier-acquired or funda-ntal functions will then be left as a residuum to be con-ted with the fundamental structure of the cord.

1) *The Accessory Functions of the Spinal Cord and Medulla Oblongata.* movements of the hand afford the best example of the accessory ctions of the spinal cord. These movements are peculiar to man, by far the greater number of them are acquired after birth. It , therefore, be expected that the development of the structure, which esents these movements in the spinal cord, must also take place after h.

The movements which are most characteristic of the upper ex-nity in man are those of pronation and supination of the forearm the complicated movements of the hand and fingers, and it is exceed-y probable that the structural representatives of some if not all of se movements are to be found in the median group of cells. These s appear at a late period of the development of the cord, hence they n a speciality of structure which corresponds to some speciality of ction; again they maintain a small size even in the adult cord, and sequently may be expected to preside over the action of small muscles, h of these conditions being realised in the hand.

The smaller median area in the lumbar enlargement of the cord sides probably over the movements of the lower limbs, which dis-guish the adult man from the lower animals and also from the human nt. These movements are mainly executed by the extensors of the on the thigh and probably also by the adductors, and by the flexors of foot on the leg. Indeed, the slight elevation of the ball of the toe, so o allow the passive leg to swing forwards by its own weight in walking, he last movement acquired by the child; and we shall subsequently that it is the first movement to be affected in disease. If, then, the dian area of small cells be the structural correlative of the later-acquired more special movements of the limbs, it must be absent in those por-ns of the cord which do not supply nerves to limbs, and we have already n that this area is absent in the dorsal and upper cervical regions of cord.

It must be remembered that the muscles of the hand are connected h the earlier-formed or fundamental cells of the anterior horns, and

that the small cells of the median area do not of themselves suffice for the regulation of their movements. The increased development of the median area in the cervical enlargement represents merely a complication on the previous structure of the cord corresponding to the complication of muscular adjustments which distinguishes the hand of man from the anterior extremity of animals.

The hypoglossal accessory nucleus, and the internal and external accessory facial nuclei, appear to be the homologues in the medulla oblongata of the median area in the cervical and lumbar enlargements of the spinal cord. The hypoglossal accessory nucleus seems to be the additional structural complication rendered necessary by the complicated movements executed in the production of articulatory speech; while the facial accessory nuclei are the structural counterparts in the medulla of the movements of facial expression.

The next accessory function which I shall mention is the muscular adjustments necessary for maintaining the erect posture in man. These adjustments are also acquired a considerable time after birth, hence it may be inferred that their structural counterpart in the cord is not well developed at birth. The medio-lateral area corresponds in my opinion to these adjustments in the dorsal region of the cord. The cells of this area are not well developed at birth, and the area is entirely absent in the lower animals. These cells are also of small size, even in the adult cord, and if, as we have already stated, the size of the ganglion cell is related to the size of the muscle with which it is connected, the erectores spinæ are the muscles of the trunk which best correspond to this description. The medio-lateral area appears also in the upper cervical region, and it may be presumed that the small muscles which extend the vertebral column in the neck, and draw back and rotate the head, are supplied from these cells. We have already seen that some of the fibres of the eleventh nerve (spinal accessory) are derived from the postero-lateral group in the cord, and it is very probable that the accessory nucleus of this nerve in the medulla is the homologue of the medio-lateral area in the upper cervical and dorsal regions of the cord. The accessory nucleus of the eleventh nerve is the additional organisation rendered necessary by the complicated movements of the human larynx.

The marginal cells of the postero-lateral, antero-lateral, and central groups appear late in the development of the cord, and these therefore must be regarded as belonging to the accessory system, even although the ganglion cells are of comparatively large size. The fact that these cells are of large size shows that they must be engaged in the regulation of the movements of large muscles. It is probable that these marginal cells in the lumbar region regulate the contractions of the large muscles of the lower extremity which are engaged in maintaining the erect posture. The great relative size of the gluteus maximus in man, as compared with the lower animals, would appear to render necessary a corresponding increase in the number of ganglion cells

the spinal nucleus which regulates its movements in the former, as compared with that in the latter. And inasmuch as the gluteus maximus is not much called into action until a considerable time after birth, these superadded cells must belong to the accessory system. These additional cells may probably be represented by the marginal cells of the postero-lateral group in the lumbar region. The alternate upward rotation of the pelvis which takes place in walking, and which is mainly effected by contraction of the gluteus medius and minimus, is likewise a very special movement; and it also may be regulated by the later-developed cells of one or other of these groups of ganglion cells in the anterior horns.

We have seen that the postero-lateral group in the upper cervical region gives off the spinal portion of the spinal accessory nerve, and that this portion forms the external branch of the nerve, which is distributed to the sterno-cleido mastoid muscle and the upper portion of the trapezius. But in man the sterno-cleido mastoid is in close relation with the clavicular portion of the pectoralis major, being only separated from it by the clavicle, and in those animals in which the clavicle is deficient it runs with the anterior part of the trapezius muscle into the deltoid, forming a mastoido-humeral muscle. All of these muscles are closely associated in their actions, and it is, therefore, probable that all are innervated from the postero-lateral group, while the latissimus dorsi, rhomboidei, and several other muscles may perhaps be added to this list. It is very probable indeed that the muscles which may be compendiously summed up with reference to their functions as the accessory muscles of inspiration are innervated from this group in the cervical and dorsal regions. These muscles are briefly the sterno-mastoids and scaleni, the pectoralis major and minor, the serrati postici et superiores, the subclavius, and the elevators of the head and spinal column.

The postero-lateral and medio-lateral groups of ganglion cells consist of a series of superimposed ganglionic centres, constituting a column of cells which extends from the lumbar region, through the dorsal and cervical regions of the cord to the medulla and pons. Speaking broadly, this column regulates the muscular contractions necessary for the maintenance of the erect posture, the contraction of the extraneous muscles of inspiration, in part at least that of the muscles supplied by the spinal accessory, vagus, glosso-pharyngeal, seventh, and by the motor branch of the fifth nerves. The portion of the facial nerve supplied by the continuation of the postero-lateral group in the medulla probably presides over the function of the facial muscles in their relation with mastication and respiration. The series of superimposed ganglionic centres of which the postero-lateral group consists cannot act independently of each other; and in order to secure harmony of action, some of these centres must become subordinate to other centres, either of the same column or of some other part of the nervous system. All of them are doubtless co-ordinated in the cortex of the brain, but it is not improbable that the inferior centres of the column are also subordinated

to one of the superior centres in the medulla oblongata. If such should be the case, there is no occasion for assuming the existence of a distinct respiratory centre in the medulla oblongata apart from the upward continuation of the postero-lateral column of cells. It is much more probable that the respiratory centre is merely an enlargement in the medulla of the postero-lateral column of cells. It is also quite likely that this enlargement is closely connected with the other groups of cells which have been continued upwards from the cord into the medulla.

(2) *Fundamental Voluntary Functions.*—With respect to the functions of the antero-lateral group, I must content myself by saying very little. The cells of this group always maintain the lead in the course of development. It is not only that they begin to develop and assume processes at an earlier period than the cells of the other groups, but the greater portion if not all of them appear almost simultaneously, and maintain an equal rate of growth during development. The antero-lateral differs in this respect from the postero-lateral and central groups, which increase in size by the gradual addition of new ganglion cells at their margins. It may be expected, therefore, that this group will regulate the fundamental actions, or the actions which are carried on in a reflex manner, and which are in great measure independent of the cephalic ganglia. In this connection the intercostal muscles, the diaphragm, abdominal muscles, and the muscles constituting the floor of the pelvis will immediately suggest themselves. In the lower extremity the most general movements may be expected to be regulated by the antero-lateral group. These movements are flexion of the thigh on the body, of the leg on the thigh, and elevation of the heel. It may be said that elevation of the heel is a movement almost peculiar to man, but this is rendered necessary during locomotion, owing to the depression of the heel which has been effected in the course of evolution, by the progressive increase in the strength of the flexors of the foot on the leg.

On watching the first movements of the human infant it will be seen that the power to elevate the heel is acquired early, while the elevation of the toe so as to allow the foot to swing forwards by its own weight is the last movement acquired; hence it is the most special movement, and it will be represented in the cord by the superaddition of new ganglion cells to those already existing. What the movements are which are regulated by means of the antero-lateral group in the cervical region I can only make a rough conjecture. They are no doubt the simplest movements, and those which man possesses in common with the lower animals. The most probable of these movements are flexion at the wrist, simple flexion and extension at the elbow, and the backwards and forwards movements at the shoulder, and flexion of the neck and head. Some of the muscles engaged in these actions we have already found reason to believe were innervated by the postero-lateral group; but this does not exclude the possibility of their being innervated also by the antero-lateral group. There is so much uncertainty, however, with regard to the function of the

ro-lateral group in the cervical region that it would be hazardous nake any assertion with regard to it. There is also quite as much ertainty with respect to the functions of the central, internal, and erior groups.

§ 375. *Reflex Action.*—The production of reflex action is one the earliest and most fundamental functions of the spinal d. As we have already seen, every reflex act requires for its formance an afferent and an efferent fibre, and a centre· e earlier-formed ganglion cells of the anterior grey horns con- ute the centres of reflex action; and it is probable that the lex afferent fibres pass to them directly, without the inter- ition of the grey substance of the posterior horns. Inasmuch the reflex afferent fibres are formed at an early period in the velopment of the cord, they must be thrust out laterally cing the development of the posterior grey horn, so that y will occupy an external position in the fan formed by spreading out of the fibres of the posterior roots. We ve already seen that there are grounds for believing that the erent fibres of the tendinous reflexes pass in the inner radi- ar fasciculus, and it is not improbable that the afferent res of the cutaneous reflexes pass in the outer radicular ciculus. The efferent reflex fibres pass out in the anterior ts, and the same fibres probably convey both reflex and luntary impulses.

§ 376. *Trophic Function of the Cord.*—It is well known that e ganglion cells of the anterior horns of the cord exercise a phic influence on the muscles; but whether there are trophic ls endowed with special functions, or whether all the cells e endowed with both motor and trophic functions, I am unable say. With some degree of qualification, I feel inclined to opt the latter view.

It is well known that within certain limits, increased func- nal activity of a muscle is followed by an increase in its lk, and, conversely, that a diminution of its activity is followed diminution of its bulk. When, therefore, the mechanism in e cord, which regulates the movements of the muscle, is in a ate of activity, this is followed by an increase in the function of e muscle, and consequently by an increase in its bulk. If, in

addition to an increase in its bulk, the muscle be called upon to make a new adjustment in response to altered circumstances, the new adjustment can only become permanent in the race when it is organised in the cord by the growth of new cells and fibres in addition to the original mechanism by which its movements were guided. But if the new cells and fibres become incapacitated from any cause, the muscle will soon lose the structural modification which corresponded to its recently-acquired functional adjustment, but no other change will take place in it. As long as the original mechanism is maintained in the cord, so long will the nutrition of the great bulk of the muscle go on as before. But the case is very different when the function of the original mechanism is destroyed; then the nutrition of the muscle is injured at its very foundation, and profound trophic changes occur. It is very probable, therefore, that the influence exerted by the later-developed ganglion cells of the anterior horns on the nutrition of the muscles is small, while that of the earlier-developed cells is very great.

§ 377. *Automatic Action.*—The spinal cord contains a considerable number of what are regarded as automatic centres, but it is probable that many of these act in a reflex manner. The lumbar portion of the cord contains centres for the regulation of the acts connected with micturition, defecation, erection and ejaculation, and parturition. The oculo-pupillary centres in the upper dorsal and cervical regions of the cord have already been described.

Vaso-motor centres exist in the cord by means of which the *tonus* of the muscular coat of the vessels is maintained. It has been thought that the spinal cord also exercises a tonic action over the skeletal muscles, but this opinion is doubtful. The tone of the sphincters of the bladder and rectum, however, is undoubtedly maintained by the lumbar part of the cord, and is probably reflex in character. The peristaltic movements of the œsophagus, stomach, and intestines are regulated by the central grey tube. Little is known beyond conjecture of the localisation of the centres of visceral innervation in the cord. That they are not situated in the anterior grey horns is rendered certain by the fact that the visceral movements, and

ıe automatic actions of defecation, micturition, erection, and arturition remain unaffected in disease limited to the anterior rey horns.

Several considerations may be adduced tending to show that ıe vesicular column of Clarke contains the spinal centres of isceral innervation. The cells of this column are bipolar, like ıose of the sympathetic, and not multipolar, like those of the nterior horns which regulate the complicated actions of the keletal muscles. This column is absent in the lumbar and ervical enlargements, the portions of the cord which supply erves to the limbs, and in the upper half of the cervical egion which supplies nerves to the muscles of the neck. It , on the other hand, present in the upper lumbar and the orsal regions of the cord—the portions from which the trunk innervated, and is again represented in the medulla oblongata ; the principal nucleus of origin of the vagus—the most im- ortant visceral nerve of the body. It may be assumed that all ıe actions regulated through the vesicular column of Clarke re subordinated to the highest expanded portion of it which onstitutes the nucleus of the vagus; hence there is no reason) assume that the medulla oblongata contains a circumscribed aso-motor centre distinctly separated from the nucleus of the agus.

378. *Functions of the Posterior Grey Horns and Posterior Roots.*

Afferent impulses are conducted to the spinal cord by the osterior roots. As already remarked, it is probable that the fferent impulses, which have undergone the highest organi- ıtion in the cord, are conducted by the fibres which occupy the eriphery of the fan, formed by the spreading out of the fibres the posterior roots as they enter the substance of the cord. ı the anterior horns the most specialised actions are repre- ented, partly by the development of new processes to the xisting ganglion cells, and partly by the growth of additional ells; but in the posterior horns the fibres, which conduct the ıost specialised impulses, have become adapted to their ınctions by the gradual development in connection with them f special peripheral terminal organs on the one hand, and

central terminal organs on the other. The stimulation of certain fibres in an early stage of development may give rise only to diffused and irregular contractions, while at a higher stage of development complicated and apparently purposive reflex movements are produced by a similar stimulation; again, a stimulation which at an early stage of development gives rise only to a diffused sensation of pain, may at a higher stage of development evoke intellectual sensations of touch and temperature. It may, therefore, be expected that the fibres which conduct reflex impulses, and those that conduct the impulses which on reaching the cortex of the brain give rise to the intellectual sensations, will occupy the periphery of the fan of the posterior roots; while those which conduct the impulses which on reaching the cortex give origin to the common or emotional sensations will occupy its centre. We have already seen reason for believing that the afferent fibres of the tendinous reflexes pass through the internal radicular fasciculus to reach the posterior horn, and it is probable that the afferent fibres of the sense of touch and locality also pass through the same fasciculus. We have also supposed that the cutaneous reflex fibres pass through the external radicular fasciculus, and it is probable that the afferent fibres of the sense of temperature likewise pass through this bundle. The afferent fibres of the common sensation of pain pass through the centre of the posterior roots directly into the grey matter of the posterior horns.

Section of the white posterior column destroys the sensation of touch permanently in the regions situated below the section, but leaves the sensation of pain unaffected; and, conversely, section of the entire grey substance, leaving the posterior columns intact, destroys the sense of pain and leaves that of touch (Schiff).

A retardation of the conduction of sensation occurs when the posterior grey horns are cut, and the more the grey substance is diminished the more marked is the retardation. The conduction of sensory impressions decussates in the cord soon after the root fibres enter it, but considerable difference of opinion exists as to the mode and extent of this decussation with regard to the conducting paths of the different kinds of sensation. The further course of the afferent fibres through the cord is not well known.

It is supposed that the sensory paths of the lower extremities lie at first in the lateral columns, and do not enter the posterior columns till they reach a higher level. The posterior column of the lumbar region is said to contain only the nerves of touch for the pelvic region, sexual organs, perinæum, and anal region (Erb).

§ 379. *Functions of the Central Grey Column.*—The central grey column is not supposed to be endowed with any active functions, yet, pathologically regarded, it is, as will hereafter appear, one of the most important portions of the grey substance of the spinal cord. The continuation of this column in the medulla oblongata contains, as we have seen, the accessory nuclei; and the median areas of the anterior horn in the cervical and lumbar enlargements, as well as the medio-lateral areas in the dorsal and upper cervical regions, may be regarded respectively as anterior and lateral outgrowths of the central column, instead of being regarded as portions of the anterior horns. These areas, indeed, constitute the border-land between the central column and anterior horn, and they are involved in the diseases of both structures.

§ 380. *Functions of the Special Nuclei of the Medulla Oblongata, Pons, and Crura.*—The functions of the special nuclei do not require extended consideration at present. All of them serve to transmit impulses received through the nerves of special sense, not only to the cortex of the brain, but probably also to the cortex of the cerebellum, while likewise ministering to complex reflex actions. The corpora quadrigemina, for instance, are anatomically connected, not only with the cerebrum, but also with the superior peduncles of the cerebellum; while they have been proved, both anatomically and experimentally, to form an important reflex centre between the retina and the internal and external muscles of the eye. It is, indeed, likely that still more extensive and complex reflex actions are regulated by the corpora quadrigemina, since they are known to be anatomically connected with the upward continuation of the anterior root-zones of the spinal cord. Two of the four nuclei of origin of the auditory nerve are intimately connected with

the inferior and middle peduncles of the cerebellum, and it is probable that one of them at least conducts labyrinthine impressions to the cerebellum.

The corpora quadrigemina are homologous with the optic lobes in fishes and the lower vertebrata—organs which are developed in connection with the sense of sight. These ganglia appear to be the centres for the reflex co-ordination of all the muscular actions concerned in the movements of the eyeballs and of the reflex contraction of the pupils caused by light falling on the retinæ. It is through these bodies, and not directly, that the optic tracts come into relation with the cerebellum ; hence it may be expected that they will be associated with the latter in its functions. We have already seen that the corpora quadrigemina are connected with the anterior root-zones, or the system of fibres which co-ordinate the actions of the cord longitudinally on the side of the outgoing currents ; hence the inferior segments of the body are to a considerable extent brought under the regulative influence of these ganglia. The corpora quadrigemina are, however, simple co-ordinating centres, and their regulative action on the inferior segments of the body is of a purely reflex character. The following may be taken as an illustration of the manner in which I believe them to act:—While a fish is swimming through the water a sudden impression is made on the right eye by the shadow of a large approaching object, and immediately the muscles of the tail on the left side contract, and the head is turned away from the object. Such a movement would tend to secure the safety of the fish from capture by a more powerful antagonist. If, on the other hand, the impression is made by a relatively small object, the muscles of the tail on the same side might contract, so as to turn the head towards the object—a movement which would tend to secure prey. In these movements the main regulative centres are the optic lobes, and there is no occasion to believe that the actions are in any way of a different character from the ordinary reflex movements of the spinal cord. It may, however, be remarked, in passing, that since a large approaching object would produce a greater impression than a small object, a rudimentary eye would be more useful to its possessor for avoiding capture than in securing prey; and, consequently, the primary and fundamental connection between the eye and the inferior segments of the body would be a crossed one. The most ready communication, therefore, would be between the right eye and the muscles of the left side of the body. And this helps to explain the crossing of the optic nerves, not only in the lower animals with rudimentary eyes, but in the higher organisms; since, during the development of the latter from the former, the primary and fundamental crossing, however much it may be modified, is still retained. It is, indeed, very probable that the crossed connection which may be supposed to exist in the lower vertebrata between the rudimentary eyes and the muscles of the

body was the main factor in determining during the course of development the crossed connection which exists between the cerebral hemispheres and the spinal cord in the higher vertebrata.

§ 381. *Functions of the Superadded Grey Substance of the Medulla Oblongata, Pons, and Crura.*—We have already seen that there are no grounds for believing that the centres of respiration, deglutition, mastication, and the regulation of the heart's action, the vaso-motor diabetic, and so-called convulsive centre of Nothnagel, are represented by grey matter in the medulla, apart from that which is the upward continuation of the grey substance of the spinal cord, and consequently the masses of grey matter which are superadded in the medulla, pons, and crura, must preside over other important functions. Little, however, is known with respect to these. The most reasonable supposition I can form is that all of them are connected with the cerebello-spinal system, and are, therefore, engaged in regulating the tonic muscular contractions rendered necessary to maintain the various attitudes of the body.

§ 382. *Functions of the White Substance.*—According to the fundamental law of development already mentioned, we may expect that the parts of the cord which begin to develop at an early period are engaged in the most general actions; while those which develop at a late period are engaged in the most special actions. The most general actions of the cord are those which it performs as a group of simple co-ordinating centres; and the most special are those which it performs in subordination to the compound and doubly compound co-ordinating centres. We may, therefore, expect to find that the anterior and posterior root-zones, which appear at a comparatively early period in the development of the cord, belong to the spinal system of simple co-ordinating centres, while the direct cerebellar fibres, the column of Goll, and the pyramidal tract, which appear at a comparatively late period of development, bring the simple co-ordinating centres of the cord under the control and guidance of the compound and doubly compound co-ordinating encephalic centres. So far as can be ascertained, this expectation is realised.

§ 383. *Functions of the Anterior and Posterior Root-zones.* These consist, as already stated, of looped fibres, which connect ganglion cells at different elevations in the cord. The anterior root-zone maintains a close relationship with the anterior grey horns, and its fibres probably assist in co-ordinating efferent impulses from above downwards. But although the anterior root-zone belongs primarily to the spinal system, it is not improbable that it may have become at a subsequent stage of development connected indirectly, if not directly, with some of the cephalic centres. The close relationship of the olivary body with the anterior root-zone in the medulla would seem to imply that the latter may be the medium of conveying efferent impulses from the cerebellum. The anterior root-zone is also probably connected with the corpus striatum, and may therefore be the channel through which the efferent impulses from the latter are conveyed downwards to the cord. It is also connected with the corpora quadrigemina, and may serve to convey reflex impulses originating in the retina down the cord. The posterior root-zone, on the other hand, maintains an equally close relationship with the posterior grey horns, and its fibres probably assist in co-ordinating afferent impulses from below upwards. We have seen that, with the exception of the part which belongs to the sensory roots of the fifth nerve and the fasciculus rotundus, the posterior root-zone terminates in the triangular nucleus, and that the latter is connected by arcuate fibres with the olivary body, which in its turn is connected with the opposite half of the cerebellum. This indirect connection with the cerebellum would appear to indicate that some at least of the fibres of the anterior root-zone belong to the cerebello-spinal system.

§ 384. *Functions of the Direct Cerebellar Tract.*—This tract belongs to the cerebello-spinal system, its fibres connecting the vesicular column of Clarke and the cortex of the cerebellum (Flechsig). Little is known with regard to the functions of these fibres, except that they appear to convey afferent impulses. This is presumed to be the case, because when the fibres of the tract are injured in any part of their course, the portions above the seat of injury undergo rapid degeneration.

§ 385. *Functions of the Column of Goll.*—This column must be regarded as a special structure from the comparatively late period at which it is developed. Its fibres also undergo rapid degeneration above the seat of injury; hence it may be inferred that they convey afferent impulses, but nothing further is known with regard to their functions.

§ 386. *Functions of the Pyramidal Tract.*—This tract is now well known to be the means of communication between the motor area of the brain and the anterior grey horns of the cord.

The fibres which pass into the lateral column connect the anterior grey horn of one side with the cortex of the opposite side; while those which constitute the column of Türck connect the anterior horns and cortex on the same side. When the fibres of the tract are injured in any part of their course the portions below the seat of injury undergo rapid degeneration, and this fact alone is sufficient to indicate that these fibres convey efferent impulses. This tract is, indeed, the channel by means of which voluntary impulses are conveyed from the cortex of the brain to the spinal cord. The crossed and direct connection which this tract forms between the cortex of the brain and the grey anterior horns, is rendered necessary by the fact that every movement of one side of the body alters the centre of gravity, and necessitates a new adjustment of the opposite side. I obtained this idea in a conversation with Dr. Hughlings Jackson, and he illustrated his meaning by showing that when a man stands on the ball of the right foot, and stretches his right arm upwards and forwards to reach an object, the body being also inclined forwards, the left leg is instinctively thrust backwards, and the left arm downwards and backwards, in order to keep the centre of gravity as far back as possible and so to prevent the line of gravity from passing in front of the ball of the right foot. The muscular contractions of the right side of the body may be supposed to be regulated in this action from the left cortex of the brain through the fibres of the pyramidal tract of the lateral column of the right side, while the movements of the left arm and leg are also regulated from the left cortex, but the impulses are conveyed to the same side of the cord and of the body by the fibres of the column of Türck.

CHAPTER II.

MORBID ANATOMY AND CLASSIFICATION OF THE DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

(I.)—MORBID ANATOMY OF THE SPINAL CORD AND MEDULLA OBLONGATA.

In the preceding chapter we have traced the operation of the law of evolution in the development of the spinal cord and medulla oblongata; we must now trace the operation of the law of dissolution in the breaking down of the structure of these organs by disease.

§ 387. *Histological Changes.* — The histological changes which occur in the various elements of the structure of the cord must first be briefly described.

1. *Morbid Changes of the Ganglion Cells.*

(*a*) *Hypertrophy.*—In acute inflammation of the cord the ganglion cells become swollen, their contents cloudy and granular, the processes also taking part in the changes (*Fig.* 144, 2). These cells often contain a large amount of yellow pigment, a condition which has been described by Dr. Allbutt as "yellow degeneration" (*Fig.* 144, 3).

(*b*) *Shrinking.*— In the acute diseases of the grey substance of the cord, the ganglion cells, especially the small cells of the median areas, become shrivelled, their fluid contents appear to have escaped, and the cell wall to have shrunk around the nucleus and a small quantity of yellow pigment (*Fig.* 144, 4). At a subsequent period the cells lose their processes and become converted into small angular masses, in which even a nucleus cannot be detected.

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

(*d*) *Vacuolation.* — Two or three large spherical air spaces, named

vacuoles, may sometimes be observed in ganglion cells which have undergone a granular degeneration (*Fig.* 144, 7).

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

(*f*) *Pigmentary Degeneration.*—The best examples of pigmentary degeneration are seen in the chronic diseases of the cord. The cell wall

FIG. 144.

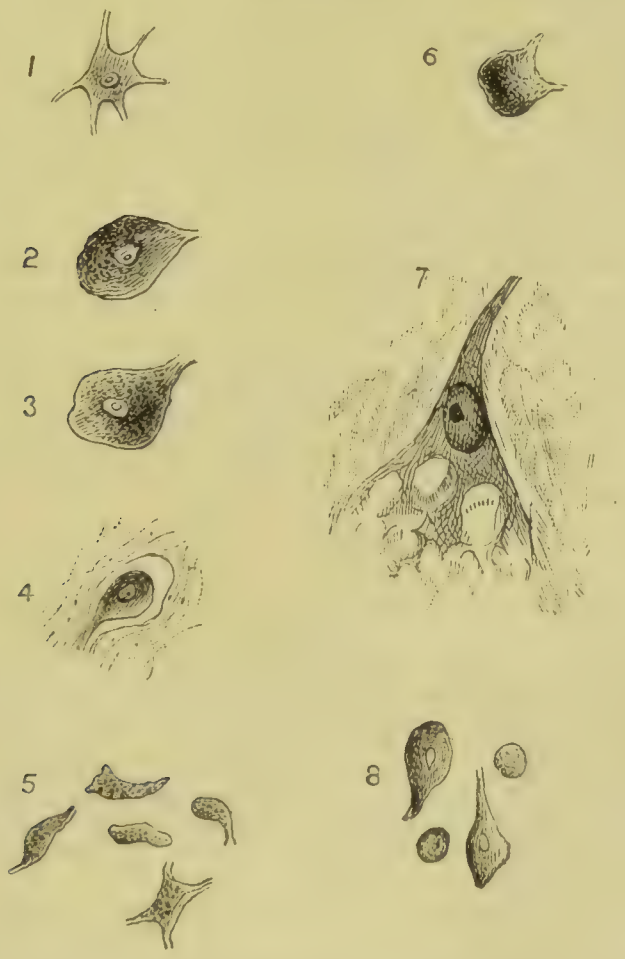


FIG. 144 (Young). *Ganglion Cells of the Anterior Grey Horns of the Spinal Cord.* 1, Healthy caudate cell; 2, Hypertrophied cell; 3, Yellow degeneration (the yellow colour cannot be represented here); 4, Shrivelled cell; 5, Chronic atrophy, a group of cells from a case of pseudo-hypertrophic paralysis; 6, Pigmentary atrophy; 7, Vacuolation, from a case of canine chorea (Gowers); 8, Chronic atrophy, from a case of progressive muscular atrophy—"yellow atrophy."

becomes contracted around a mass of dark granular pigment, the nucleus and nucleolus are indistinct or obliterated, the processes are atrophied, and many of them have disappeared (*Fig.* 144, 6).

(*g*) *Atrophy.*—In chronic diseases the cell wall becomes dense and contracted, the processes broken off, and the remnant of the cell converted into a small angular mass, without recognisable nucleus or nucleolus, and finally all traces of the cell may be lost (*Fig.* 144, 5 and 8).

(*h*) *Calcareous degeneration* of the ganglion cells of the cord is rarely observed (Förster).

2. *Morbid Changes of the Nerve Fibres.*

The medullated nerve fibres of the spinal cord undergo alterations more or less similar to those which have already been described in the case of the fibres of the peripheral nerves, and consequently these changes need not be described here in detail.

(*a*) *Hypertrophy of the Axis Cylinder.*—In myelitis it is not rare to observe on transverse section that the axis cylinders of many of the fibres have increased to two or three times their normal dimension. In longitudinal sections it is seen that the swelling does not extend the whole length of the axis cylinder; the latter presents a varicose appearance, so that its diameter is much diminished in size at some points.

(*b*) *Atrophy of the nerve fibres*, similar to that which occurs in the peripheral nerves when the fibres are severed from their trophic centres, may be observed in the medullated fibres of the spinal cord. This atrophy begins by coagulation of the myeline, which becomes granular and broken up into globular masses that are finally absorbed. The axis cylinder persists for a long time after the medullary sheath has disappeared, but by-and-by it also diminishes in size, and ultimately disappears.

(*c*) *Calcareous degeneration* of the fibres of the cord has been exceptionally observed (Förster, Virchow).

3. *Morbid Changes of the Neuroglia and Connective Tissue.*

(*a*) *Glüge's corpuscles* consist of large globular cells filled with granular contents. These cells may be observed in the spinal cord of the embryo, but are never met with in considerable numbers in the cord of the adult, except in cases of disease. They are supposed to derive their origin from fatty degeneration of the cells of the connective tissue and neuroglia, the white corpuscles of the blood, and the endothelial cells of the vessels and of the capsules of the ganglion cells.

(*b*) *Amyloid Corpuscles and Colloid Bodies.*—Amyloid corpuscles (corpora amylacea) are small, round, concentrically laminated bodies. Most of them are turned blue, or bluish grey, when acted on by iodine alone, and assume a beautiful bright blue tint on the addition of sulphuric acid. Colloid bodies are irregular masses, consisting apparently of changed myeline; they assume beautiful tints on being stained with logwood, or some of the aniline dyes. It is probable that these bodies may be the

result of post-mortem decomposition, and neither they nor the amyloid corpuscles afford trustworthy evidences of disease.

(*c*) *Deiter's cells* appear to be increased in number in inflammatory diseases of the cord.

(*d*) *Hypertrophy and Hyperplasia of the Connective Tissue.*—The septa of connective tissue become swollen, and the nuclei of the neuroglia largely increased in number. It is also probable that leucocytes, which have migrated from the vessels during inflammatory processes, may subsequently become organised, and thus increase the normal volume of the connective tissue of the cord.

(*e*) *Sclerosis and Retraction.*—When hyperplasia of the connective tissues has once taken place, the newly-formed tissue may subsequently undergo cicatricial contraction, and thus lead to the destruction of the nervous elements. The process which leads to sclerosis often begins in the nerve cells and fibres, and may be called *parenchymatous sclerosis.* At other times the morbid changes appear to begin in the connective tissue or neuroglia, the nerve cells and fibres being secondarily invaded; this form may be called *interstitial sclerosis.*

4. *Morbid Alterations of the Vessels.*

(*a*) *Intravascular Changes.*—The vessels are at times greatly distended with blood, but this is not a trustworthy evidence of disease, inasmuch as the distension may have occurred from the mode of dying, or from hypostatic congestion after death. The capillary arteries may at times be distended with emboli.

(*b*) Changes in the walls of the spinal vessels are observed in chronic Bright's disease, identical with those which occur in the vessels of the body generally in that disease.

(*c*) *Perivascular Changes.*—The most important perivascular changes observed in disease of the spinal cord are caused by migration of the white corpuscles of the blood into the perivascular lymph-spaces and surrounding tissues. The number of leucocytes surrounding a vessel may sometimes be so great as to constitute what has been called a *miliary abscess* (Plate V., *Fig.* 2). Rupture of a vessel may occur, giving rise to hæmorrhage into the tissues. Red blood corpuscles are at times localised in a perivascular space, but it is difficult to determine in these cases whether the red corpuscles have escaped by rupture, or have, like the white corpuscles, migrated through the wall of the vessel.

§ 388. Let us now pass from the details of the morbid changes of the cord to the general principles which underlie them. In accordance with the law of dissolution (§ 35) we may expect that the accessory portions of the cord will form parts of least resistance to the inroads of disease.

In the grey substance the least resistance to disease will be

offered by the central column, which is, as we have already seen, the embryonic area of the cord, and by the median area of the anterior grey horn in the lumbar and cervical enlargements and the medio-lateral area in the dorsal and upper cervical regions—areas which, as already remarked, may be regarded as outgrowths of the central column. These areas contain the accessory nuclei of the spinal cord, and since these ganglion cells not only are developed at a comparatively late period, but also frequently maintain a relatively small size in the adult, the resistance offered to the invasion of disease becomes still less. This law must necessarily be true whether the disease begin in the ganglion cells themselves, in the neuroglia, or in the vessels, or whether it be caused by a poison circulating in the blood, provided that the poison possess no special affinity for any one set of the ganglion cells more than for others.

The cell walls of the small and recently-developed cells are much thinner than those of the larger and earlier-developed cells, hence the exchange of materials, which is the necessary accompaniment of nutrition, takes place more readily in the former than in the latter. But this is not all. A large cell presents, in proportion to its bulk, a smaller surface to its environment for the absorption of nourishment than a small cell, and consequently the relative amount of nourishment absorbed by the large will be less than that absorbed by the small cell (§ 9). But high nutritive activity is associated with great instability, which declares itself in increased readiness to give out energy or to multiply, the latter process, of course, involving the disorganisation of a highly-organised tissue.

When, therefore, the ganglion cells of the anterior horns become diseased, it may be expected that the later-developed and small cells will be the first to suffer, and when a poison like strychnine circulates in the blood, the same cells will also be the first to be affected, supposing the drug not to possess a special affinity for one ganglion cell more than for another. The reason of this is, that the quantity of the poison which will enter the substance of the small cell will be much larger in proportion to its bulk than that which will enter into the substance of the large cell. If the disease begin in the neu-

roglia, it may be expected that the spongy and loose neuroglia of the later-developed portions of the grey substance will resist its inroads less effectually than the dense neuroglia surrounding the earlier-developed groups of ganglion cells. The central grey column possesses a loose and spongy neuroglia, and we have seen that it may be regarded as the embryonic area of the spinal cord, so that it may be expected to offer little resistance to the invasion of disease. We shall hereafter see that some of the most rapidly fatal diseases of the cord appear to ascend in the central grey column. It has been pointed out that the later-formed cells of the anterior horns grow close to the arteries, while the earlier-developed cells are pushed, in the course of development, away from them. When, therefore, rapid exudation takes place from the vessels, whether it consist of a fluid and granular exudation or of migration of white blood corpuscles, the cells in the neighbourhood of these vessels will suffer sooner and in greater degree than those more remote.

That the lines of least resistance to disease in the lumbar region are in the direction of the vessels is well illustrated by *Fig.* 145, which is taken from a section of the middle of the lumbar enlargement in a case of infantile paralysis, under the care of Dr. Humphreys, at the Pendlebury Hospital for Sick Children. This case is described in the "Transactions of the Pathological Society of London" for 1879, and will be subsequently mentioned. My present object is to show that even in an acute disease like infantile paralysis the cells near the vessels have become destroyed in preference to the

FIG. 145.

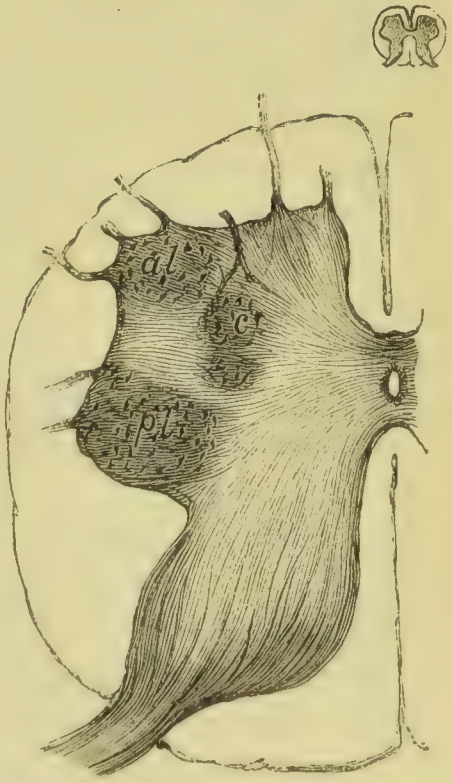


FIG. 145 (Young). *Section of the Lumbar Region of the Spinal Cord from a case of infantile spinal paralysis. pl*, postero-lateral group; *al*, antero-lateral group; *c*, central group. The internal and anterior groups have disappeared, and the marginal cells of the remaining groups are also destroyed.

others. If *Fig.* 145 be compared with *Fig.* 111, it will be seen at once that the disease is most marked in the vascular areas of the cord, and that the cells which have been last developed are, on the whole, those which have suffered most will be apparent by referring to the previous description and illustrations of the development of the cord. It is true that the earlier-developed cells of the internal and anterior groups have disappeared; but the cells of the antero-lateral, and those of the central portions of the postero-lateral and of the central group are well preserved; while the marginal cells of the two latter groups and all the cells of the median area are completely destroyed. It is not likely that this law will always be observed in a disease having such an acute and sudden onset as infantile paralysis; but an examination of the diagrams given by Clarke, Charcot, and Joffroy, shows so many indications of the fulfilment of this law that its occurrence cannot be regarded as accidental. The same law is observed, at least very frequently, in cases of acute and subacute ascending central myelitis, as well as in tetanus and hydrophobia. It was while examining cases of this kind that my attention was first directed to this subject. In all the acute diseases affecting the grey substance of the spinal cord I observed that, unless the destruction was so great as to involve the anterior horns in their entire extent, the small cells and those in the line of the distribution of the arteries manifested evidences of disease to a much greater extent than the large cells and those removed from the vessels.

The distribution of the disease in the cervical enlargement is similar to that in the lumbar region, except that the median area being much larger in the former than in the latter, injury to this area forms a more conspicuous feature of disease in the former than the latter. When the dorsal region of the cord is affected by acute disease of the grey substance, the most marked morbid changes are observed in the postero-lateral or rather the medio-lateral group; and the same is the case in the upper cervical region. A section of the middle of the cervical enlargement is represented in *Fig.* 146, taken from a case of subacute ascending spinal paralysis. The disease began after exposure to severe cold with sudden paralysis of the lower

extremities, without much disturbance of sensibility. This was followed by rapid wasting of the muscles, and loss of faradic contractility. The paralysis in the course of a few weeks gradually invaded the muscles of the trunk, the muscles of the upper extremity, and ultimately the muscles of respiration. Death took place five weeks from the commencement of the paralysis. In the lumbar region the white as well as the grey substance was implicated, and there was ascending sclerosis of both the columns of Goll and of the direct cerebellar tracts throughout the entire length of the cord; the remaining portions of the white substance were healthy in the dorsal and cervical regions. The central grey column was diseased throughout the whole length of the cord, the cells of the postero-lateral group and medio-lateral area having entirely disappeared in the dorsal region, and again in the upper cervical regions; while the anterior groups of cells appeared

FIG. 146.

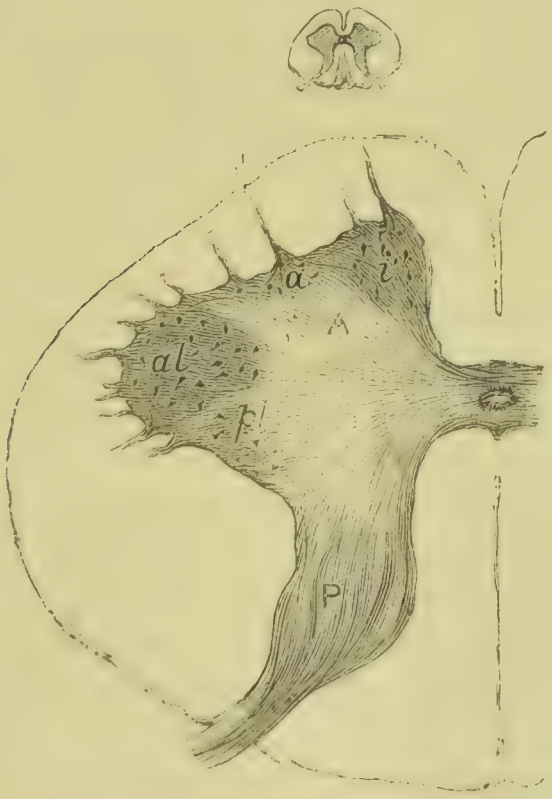


FIG. 146 (Young). *Section of the Middle of the Cervical Enlargement of the Spinal Cord from a case of central myelitis.*—*i*, The internal group; the remaining letters indicate the same as the corresponding ones in *Fig.* 145. The median area was completely destitute of cells, and a large number of the marginal cells of the different groups of the anterior horn were destroyed or diseased.

to be quite normal. In the cervical enlargement (*Fig.* 146) the cells of the median area had entirely disappeared, and the marginal cells of the central and postero-lateral groups were notably altered, while the fundamental cells of the groups presented beautiful long processes, and appeared in every respect normal.

§ 389. In the white substance the last developed fibres will also, other things being equal, offer less resistance to the inroads of disease than the earlier-developed fibres. In proceeding to verify this statement, we must compare the later with the earlier-formed fibres of the same segment, or, in other words, the same functional system of the white substance, otherwise the whole result will be vitiated. The posterior and anterior root-zones, for instance, are developed about the same time, yet the former is more liable to become diseased than the latter. The posterior is probably more exposed to the exciting causes of disease, such as peripheral injuries and ascending neuritis, than the anterior root-zone, and the small fibres of the former are more apt to be injured in inflammatory affections of the cord than the larger fibres of the latter. But if the accessory be compared with the fundamental fibres of the pyramidal tract, it will be seen that the former are much more exposed to injurious influences than the latter. The small diameter of the greater number of the accessory fibres permits a relatively larger amount of nourishment to gain access to their interior than can take place in fibres of larger diameter; hence both reparative and destructive changes are more rapidly effected in the former than in the latter.

The accessory fibres are, as we have seen, more closely related to the connective tissue septa of the cord than the fundamental fibres, hence the former are more liable to be injured in the course of the diseases which begin in the connective tissue and neuroglia than the latter. An appearance which is presented by the spinal cord in various diseases, and which for a long time puzzled me very much, is that which has been described as miliary sclerosis (Rutherford, Kesteven). This condition appears to consist of a swelling or thickening of the septa in which the blood-vessels run. In the lozenge-shaped spaces (*Fig.* 133) of

the pyramidal tract a considerable number of the small fibres which lie close to the vessels are destroyed, while the larger central fibres remain more or less healthy. When a transverse section of the cord is examined under these circumstances the part presents a spotted appearance, but instead of the miliary spots being in a state of sclerosis, they really are the most healthy portions of the section. The proximity of the fibres of the accessory system to the blood-vessels renders them also more liable than the fundamental fibres to be injured by inflammatory and other effusions.

§ 390. *Secondary Degenerations.*

The medullated fibres of the spinal cord undergo degeneration whenever their continuity is interrupted. The short looped fibres of the anterior and posterior root-zones, however, only degenerate in the neighbourhood of the lesion, probably because they soon terminate in grey matter. But the fibres which pass from one end of the cord to another are sometimes found degenerated throughout their whole length. As a rule, however, a focal lesion interrupts the continuity of the long fibres in some part of their course, and the fibres either above or below the seat of disease undergo degeneration. Some pathologists think that an irritative change spreads from the primary lesion as a centre along these fibres, but the most reasonable supposition is that the degeneration is analogous to what occurs in the fibres of peripheral nerves after they have been severed from their trophic centres. The trophic centres of the fibres of the columns of Goll and of the direct cerebellar tract are situated at their inferior extremities—the posterior horn containing the trophic centres of the former, and the vesicular column of Clarke possibly that of the latter. When, therefore, the continuity of these fibres is interrupted at any point, the portions above the seat of the lesion undergo degeneration, consequently degeneration of these fibres is called *ascending sclerosis*. But the trophic centres of the fibres of the pyramidal tract are situated at their superior extremities, these centres being probably formed by the large ganglion cells of the fourth layer of the cortex of the brain. When the continuity of these fibres is interrupted at any

point of their course, the portions below the seat of the lesion undergo degeneration, consequently this form is called *descending sclerosis.* The time occupied by the degeneration appears to be from four to eight weeks. Schiefferdecker found in experiments on dogs that it began at the end of fourteen days, was well marked at the end of four to five weeks, but changes in the connective tissue were not observed until the eighth week. Degeneration of the fibres of the spinal cord appears always to take place in the line of their conduction. When a transverse section of the spinal cord is examined by the naked eye the degenerated portion usually presents a grey or greyish discolouration, but in recent cases the cord presents no abnormal appearances until it is hardened in chromic acid or bichromate of ammonia. In cases of long standing the degenerated columns may be atrophied to such an extent that the symmetry of the cord becomes altered.

Microscopic examination shows that in the earlier stages the nerve fibres are exclusively affected. The medullary sheaths undergo fatty degeneration and ultimately disappear, while there is a considerable development of granule cells; the axis-cylinders, however, persist for some time afterwards.

In the later stages of degeneration the nerve fibres disappear entirely, the neuroglia is increased in quantity, and changes into a dense finely fibrillated tissue, which contains numerous nuclei and spindle cells.

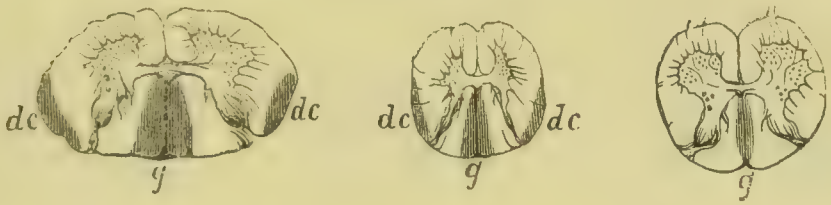
1. *History.*—Secondary atrophy, extending to the pons and pyramids of the medulla, was observed in disease of the brain by Cruveilhier and Rokitansky, but they did not follow it to the spinal cord. Türck made a thorough examination of the secondary degenerations of the spinal cord in 1851 and 1853, and their histological characters were investigated in 1863 by Leyden. Various French authors, as Charcot, Cornil, and others, published cases in which these degenerations were observed, but the most exhaustive work on the pathology of the affection was published by Bouchard in 1866. Soon afterwards Westphal showed that secondary degenerations could be produced experimentally in dogs, and this was afterwards confirmed by Vulpian.

2. *Distribution of the Degeneration.*—The observations of Charcot and Pierret, and subsequently of Flechsig, tend to show that these secondary degenerations of the spinal cord are determined by the order of its development. The development of the functional systems of the white sub-

istance of the cord affords a good illustration of the law of evolution, while the secondary degenerations afford an almost equally good illustration of the law of dissolution. The distribution of these degenerations, therefore, may be readily understood by reference to *Figs.* 134 to 140, which illustrate the development of the cord.

(*a*) *Ascending degeneration* takes place above the seat of the lesion in the columns of Goll, and terminates in the upper end of the medulla oblongata, where the fibres end in the cuneate nucleus. The direct cerebellar fibres also undergo ascending degeneration. It may begin as a thin lamella of degenerated tissue on the external surface of the lateral column in the lower dorsal region, the area of the degeneration gradually increasing in size upwards along the cord and the external surface of the restiform bodies. In lesions of the cauda equina, and sometimes after severe traumatic injuries of the sciatic nerve, the posterior root-zones, as well as the columns of Goll, undergo ascending degeneration in the lumbar and greater portion of the dorsal regions, but the degeneration becomes limited to the columns of Goll in the upper dorsal and cervical regions.

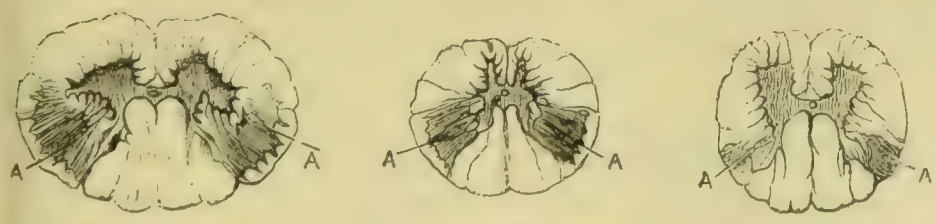
Fig. 147. Fig. 148. Fig. 149.



Figs. 147, 148, and 149. *Transverse Sections of the Spinal Cord, from the middle of the cervical enlargement, middle of the dorsal region, and middle of the lumbar region respectively*, showing ascending degeneration of the column of Goll (*g*), and of the direct cerebellar tract (*dc*).

(*b*) *Descending degeneration* occurs in all destructive lesions of the brain or spinal cord which injure the fibres of the pyramidal tract in their passage through the corona radiata, internal capsule, crus cerebri, pons,

Fig. 150. Fig. 151. Fig. 152.



Figs. 150, 151, and 152 (After Charcot). *Transverse Sections of the Spinal cord, from the middle of the cervical enlargement, middle of the dorsal region, and middle of the lumbar region respectively*, showing primary lateral sclerosis of the cord, or secondary to a lesion high up in the cord or medulla oblongata.—A, A, A, Degenerated pyramidal tracts.

medulla, or cord. In the diseases of the cord, the degeneration is generally bilateral and symmetrical, and the position occupied by the diseased portions of the cord in the lateral columns is represented in *Figs.* 150, 151, and 152; the degeneration of the columns of Türck is, however, not shown. The position occupied by the diseased portion in the medulla oblongata is represented in the annexed woodcut (*Fig.* 153, A). In cerebral lesions the degenerative tract is generally limited to one side—the side of the cord opposite the lesion in the brain—as represented in *Figs.* 154 to 156. The columns of Türck on the same side as the lesion of the brain are also usually simultaneously degenerated, but this is not represented in the figure.

Fig. 153.

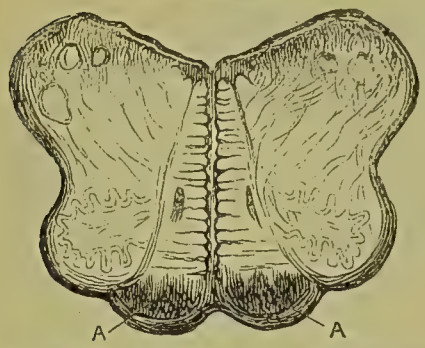


Fig. 153 (After Charcot). *Transverse section of the medulla oblongata, on a level with the middle of the olivary body.*—A, A, Sclerosis of the anterior pyramids.

Fig. 154.

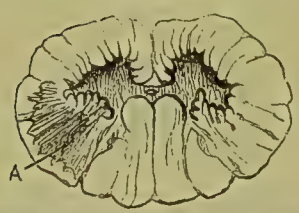


Fig. 155.

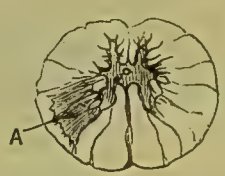
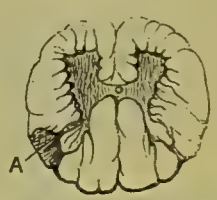


Fig. 156.



Figs. 154, 155, and 156 (After Charcot). *Transverse Sections of the Spinal Cord, from the middle of the cervical enlargement, middle of the dorsal region, and middle of the lumbar region respectively*, showing descending sclerosis of the pyramidal tract in the lateral column secondary to a cerebral lesion.—A, A, A, Degenerated pyramidal tract.

3. *Degeneration of the Spinal Cord Secondary to Amputation.*—The changes which occur in the spinal cord after amputation have been studied by Bèrard, Cruveilhier, Türck, Dickinson, Lockhart Clarke, Vulpian, and others; and, in a recent number of the *Journal of Anatomy and Physiology*, Dr. Dreschfeld has given a good resumé of the previous observations of others, while adding new observations of his own. The general result appears to be that the peripheric nerves and the white substance of the cord are unaffected, the posterior roots are often slightly diminished in size, and changes in the ganglion cells of the anterior horns are of constant occurrence. Some of the ganglion cells of the anterior horns have completely disappeared, whilst those that remain are atrophied and shorn of their processes. Judging from the various drawings, the ganglion cells of the margins of the various groups disappear first, and those of their centres remain to the last. The cells of the postero-lateral group are particularly liable to be affected. No mention is made of the disappearance of any of the ganglion cells from the anterior horn on the side opposite to that of

the amputated limb; but judging from the diagrams which illustrate Dr. Dreschfeld's paper, I should think that the number of cells in the internal group of the opposite side is much diminished. The fibres of the external fasciculus of the posterior root pass through the anterior commissure to join the cells of the internal group, and in future cases it would be worth while to observe whether a streak of degeneration might not be detected along the course of these fibres to reach the internal group of the opposite side. Hayem tore out the sciatic nerve of one side in rabbits, and found in the lumbar region of the cord on the same side sclerosis of the posterior root and posterior grey matter, along with degenerative atrophy of the ganglion cells of the intermedio-lateral tract.

§ 391. *Deformities and Malformations of the Spinal Cord.*

The deformities and malformations of the spinal cord may be subdivided into—(1) the congenital deformities which are incompatible with the maintenance of extra-uterine life; (2) the congenital deformities which are compatible with life, and do not betray themselves by any symptom during life; (3) the congenital deformities which may be recognised during life; (4) acquired deformities resulting from pathological processes (Syringomyelia, Hydromyelus acquisitus) (Leyden).

The following are the more frequent conditions observed (Leyden):—

1. *Congenital Deformities of Still-born Children.*

(*a*) *Amyelia*, or absence of the spinal cord. It is only met with when the brain is also absent.

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

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

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

2. *Congenital Deformities which cannot be recognised during Life.*

(*a*) *Anomalies in the Length and Thickness of the Cord.*—The cord is found at times thick and voluminous, and at other times thin and small. It descends at times to the third lumbar vertebra, and ends at other times opposite the eleventh or twelfth dorsal.

(*b*) Abnormal smallness of the entire spinal cord and medulla oblongata, with corresponding smallness of the nerve fibres and axis cylinders,

has recently been described by F. Schultze, in one of Friedreich's cases of "hereditary ataxy."

(*c*) *Asymmetry of the grey substance*, showing unequal width and depth of the anterior grey horns on a transverse section.

(*d*) *Abnormalities of the Pyramidal Tracts.*—Flechsig has recently shown that the fibres of the pyramidal tracts are very variable in their distribution. Each pyramid may send its mass of fibres into the spinal cord, either entirely crossed or only partly crossed, or down the anterior columns almost entirely uncrossed. These tracts are absent in anencephalous monsters (Flechsig).

In cases of congenital absence or intra-uterine arrest of development of certain extremities atrophy of definite portions of the spinal cord may be observed producing asymmetry, which is limited to the cervical or lumbar enlargement according to the extremity affected.

In a case of congenital talipes equino-varus of both legs, I found the conus medullaris remarkably thin and tapering. On transverse section the anterior grey horns were seen to be deformed, the internal border which in health runs parallel with the anterior fissure being drawn outwards and backwards, so as to be almost in a line with the anterior border of

FIG. 157.

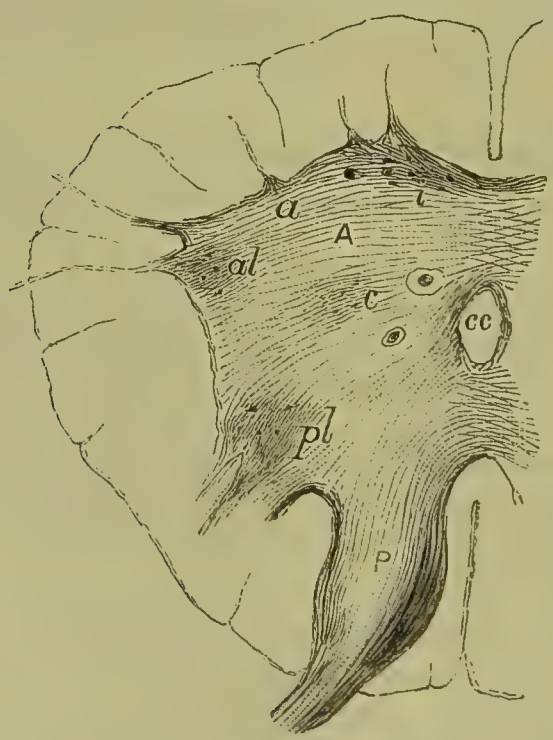


FIG. 157. *Transverse Section of the upper end of the Conus Medullaris of the Spinal Cord, from a case of congenital talipes equino-varus.*—A, P, Anterior and posterior horns respectively; *i*, internal group showing healthy cells; *a*, anterior, *al*, antero-lateral, *pl*, postero-lateral, and *c*, central groups of cells, each being represented only by a few small round cells without processes.

the anterior commissure. The ganglion cells of the internal group were well developed, although it was displaced from its usual position (*Fig.* 157, *i*). A few cells were observed in the postero-lateral area; but the cells of the anterior, central, and antero-lateral groups were entirely absent in many sections, while in others a few imperfectly-developed cells were observed in these areas (*Fig.* 157, *a*, *c*, *al*). The fine fibrillated texture of Gerlach's network and the small glistening nuclei of the neuroglia appeared to have been replaced by a loose connective tissue, thickly studded with connective tissue corpuscles. Mr. Hardie long ago maintained that congenital talipes is due to an arrest of development, and that the feet occupy postures similar to those of the embryo.

Unusual outgrowths or absence of portions of the grey matter, such as of the tractus intermedio-lateralis, are occasionally met with. Duplications of one of the grey horns for a longer or shorter distance have also been observed.

3. *Congenital Deformities which may be recognised during Life.*

(*a*) *Congenital Enlargement of the Central Canal*, a condition which has been variously called *hydrorrhachis interna*, *hydromyelus*, or *hydromyelus congenitus*. In the lighter grades of the congenital affections the central canal in the fœtus is converted into a cavity varying in width from that of an ordinary knitting needle to that of a crow's quill. The canal may extend the entire length of the cord, but is at other times restricted to certain portions, generally the cervical or lumbar enlargement, while the dilatation may occasionally be moniliform, or the anterior and posterior walls may have grown together across the middle giving rise to the appearance of a double canal. The cord does not appear to undergo any abnormal changes apart from the displacement of its various segments occasioned by the great dilatation of the canal.

In the *higher grade* of congenital hydromyelus either the spinal cord disappears entirely, or becomes split into two halves for a greater or lesser distance, while the cavity of the central canal freely communicates with the cavity of the spinal arachnoid; the hydrorrachis interna is then merged into hydrorrachis externa, as not unfrequently happens in spina bifida.

(*b*) *Spina bifida* consists of an abnormal accumulation of fluid within the cavity of the spinal arachnoid, associated with a greater or lesser deformity of the vertebral column. As it gives rise to serious symptoms during life it will be subsequently described in detail along with the diseases of the membranes of the spinal cord.

4. *Acquired Deformities resulting from Pathological Processes.*

(*a*) *Syringomyelia*, or the pathological formation of cavities, may be caused in various ways.

(i.) Cavities are formed by the softening of the central portions of new formations, such as gliomata, gliomyxomata, and gliosarcomata. The tumour is sometimes so completely disintegrated that only a capsule of connective tissue or mere traces of the tumour remain. This softening

is sometimes initiated by hæmorrhage into the interior of the tumour. This accident is particularly apt to occur in the teleangiectatic varieties.

(ii.) Breaking down and softening of apoplectic foci.

(iii.) Central softening in areas of grey degeneration and chronic myelitis.

(iv.) Obstruction of lymph channels produced by the pressure of a tumour and other causes (Westphal). Cavities have been formed in the spinal cords of animals subsequent to various injuries, and these have been supposed to have been caused by obstruction of lymph channels (Naunyn and Eichhorst).

(*b*) *Hydromyelus acquisitus*, or acquired dilatation of the central canal, may result from the following causes :—

(i.) Peri-ependymal myelitis, which consists of a proliferation of the connective tissue surrounding the central canal, may cause secondary dilatation by the shrinking of the newly-formed tissue (Hallopeau).

(ii.) Chronic meningitis, by producing adhesions of the pia mater to the dura mater at definite points, may also cause dilatation of the central canal, probably by shrinking of the newly-formed tissue (Simon).

(iii.) Obliteration of the canal at one point may lead to dilatation of the neighbouring portions.

The cavities vary greatly in size. They may indeed be only a few millimetres in length, or extend the entire length of the cord. Their number also varies ; in many cases one only is found, but at other times a large number of them may be present. They are almost always situated near the centre of the cord, and their relations to the central canal can only be determined by careful examination. The transverse diameter of these cavities may vary from that of a needle to the tip of a man's little finger. On transverse section their form is roundish, oval, or angular, and their contents consist of light and clear or turbid and yellowish fluid.

The walls of the cavities may be smooth and firm, and are often lined with a layer of cylindrical epithelium, or they may be rough, ragged, and uneven. Their walls may also be dense, and formed of cirrhotic tissue or of tissue which has undergone grey degeneration, or of the various new formations which have already been described.

The symptoms caused by the formation of cavities in the cord depend entirely upon their situation, and no definite disease which can be recognised during life can be ascribed to the presence of these cavities.

(II.)—CLASSIFICATION OF THE DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

§ 392. The rule which has hitherto been followed in this work is to describe first the simplest and most elementary diseases and to reserve consideration of the most complicated affections

o the last. In no diseases is it more advisable to follow this ule than in those affecting the spinal cord and medulla oblongata, with their membranes. The annexed table, in which hese diseases are classified, carries with it in the main its own explanation, but it may not be out of place to make a few emarks with regard to the principle adopted in arranging the tructural diseases of the nervous organs themselves as distinguished from those of their membranes and vessels, their unctional affections, injuries, malformations, and neoplasms.

It has already been remarked that the spinal cord may be livided into longitudinal segments, each of which possesses functional unity, and may be separately diseased. Diseases of one of the functional segments of the cord are called *ystem-diseases* or *fasciculated* diseases, while those involving everal of these segments may be called *mixed diseases*. In he *simple* system-diseases one functional segment of the cord nd medulla oblongata alone is affected; but it sometimes appens that two or more of them become simultaneously or onsecutively attacked, and these affections may be called *compound* system-diseases.

The system-diseases may be divided into those affecting the grey matter or the *poliomyelopathies*, and those affecting the white matter or the *leucomyelopathies*. The poliomyelopathies nay be subdivided into the diseases affecting the anterior grey horns, the central grey column, and the posterior grey horns; but the latter is never a true system-disease, being always complicated by lesions of other structures, such as the posterior roots and posterior columns. Disease of the central column is also probably never observed as an isolated affection, the prominent symptoms being caused by extension of the lesion into the anterior horns; but we shall nevertheless classify some at least of he diseases of the central column amongst the system-diseases.

The *leucomyelopathies* consist theoretically of diseases of the posterior root-zone (locomotor ataxy); of the anterior root-zone, disease of which is probably not capable of being separated rom disease of the anterior roots and anterior grey horns; of he column of Goll and the direct cerebellar tract, to both of which, however, no definite symptoms have been observed to ttach, and of the pyramidal tract (primary lateral sclerosis).

The compound system-diseases are probably numerous, but only one of them—*amyotrophic lateral sclerosis*—is recognised as a distinct type of disease. The annexed diagram (*Fig.* 158), copied from Charcot, represents the localisation of the lesion on transverse section of the cord in the various system-diseases.

In the mixed diseases of the spinal cord and medulla oblongata Landry's paralysis is first mentioned, not because it has been proved to be connected with anatomical changes in the cord, but because it is closely allied clinically with the acute forms of central myelitis. The classification adopted of the different forms of acute and chronic myelitis does not require any explanation.

Fig. 158.

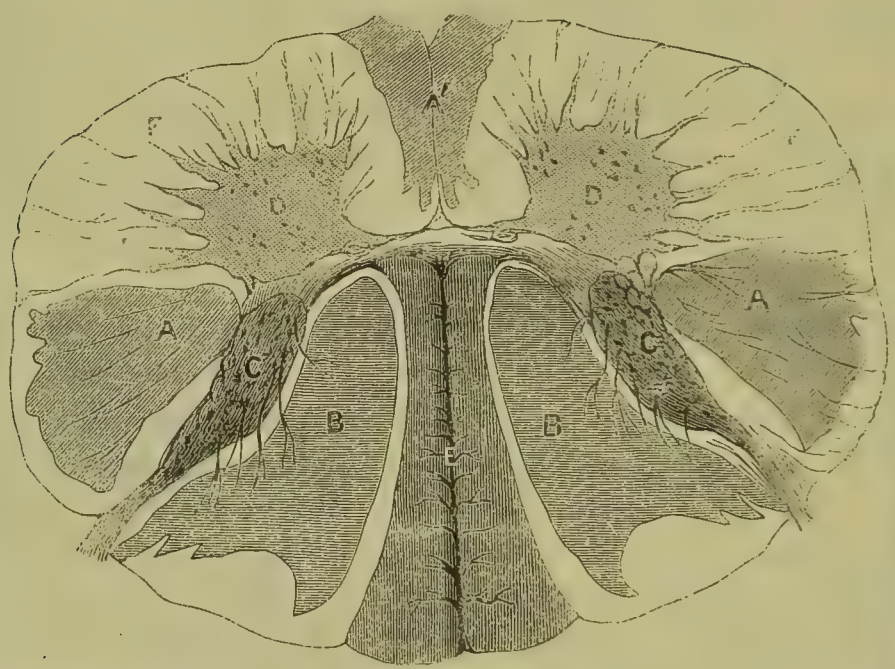


Fig. 158 (After Charcot). *Diagram of the Morbid Anatomy of the System-Diseases of the Spinal Cord.*—A, A, Pyramidal tract of the lateral column; A', Columns of Türck; B, B, Posterior root-zones; C, C, Posterior grey horns; D, D, Anterior horn; F, F, Anterior root-zone; E, Columns of Goll.

A. *Diseases of the Spinal Cord and Medulla Oblongata.*

I. System diseases.

(i.) Poliomyelopathies.

1. Poliomyelitis anterior acuta.
   - (*a*) Infantile spinal paralysis.
   - (*b*) Atrophic spinal paralysis of adults.

2. Poliomyelitis anterior chronica.
3. Progressive muscular atrophy.
4. Labio-glosso-laryngeal paralysis.
5. Pseudo-hypertrophic paralysis.

(II.) Leucomyelopathies.

1. Posterior sclerosis (Locomotor ataxy).
2. Sclerosis of the columns of Goll.
   - (*a*) Primary.
   - (*b*) Secondary and Ascending.
3. Sclerosis of the direct cerebellar tract. Secondary and Ascending sclerosis.
4. Lateral sclerosis.
   - (*a*) Primary—Tabes dorsalis spasmodica.
   - (*b*) Compound—(i.) Amyotrophic lateral sclerosis.
     (ii.) Combined posterior and lateral sclerosis.
   - (*c*) Secondary and Descending.

II. Mixed diseases of the spinal cord and medulla oblongata.

(I.) Paralysis ascendens acuta (Landry's paralysis).

(II.) Acute myelitis.

1. Acute central myelitis.
2. Hyperacute central myelitis (hæmatomyelitis).
3. Acute bulbar myelitis.
4. Acute transverse myelitis.
5. Acute hemilateral myelitis.
6. Acute myelomeningitis.
7. Acute disseminated myelitis.

(III.) Chronic myelitis.

1. Chronic central myelitis.
2. Chronic transverse myelitis.
3. Universal progressive myelitis.
4. Chronic bulbar myelitis.
5. Chronic myelomeningitis.
6. Chronic disseminated myelitis or multiple sclerosis.

(IV.) Myelomalacia.

III. Vascular diseases of the spinal cord and medulla oblongata.

(I.) Anæmia, Thrombosis, and Embolism.

(II.) Hyperæmia and Hæmorrhage.

IV. Functional and secondary diseases of the spinal cord and medulla oblongata.

(I.) Spinal irritation.
(II.) Neurasthenia spinalis.
(III.) Reflex and secondary paraplegiæ.
(IV.) Saltatory spasms.
(V.) Reflex muscular spasms.
(VI.) Intermittent spinal paralysis.
(VII.) Toxic spinal paralysis.
(VIII.) Hysterical paraplegia.

V. Traumatic diseases, tumours, and abnormalities of the spinal cord and medulla oblongata.

(I.) Wounds of the cord and of the medulla oblongata.
(II.) Slow compression of the cord and of the medulla oblongata.
(III.) Hemiparaplegia spinalis.
(IV.) Concussion.
(V.) Tumours, and abnormalities of the spinal cord and medulla oblongata.

B. *Diseases of the Membranes of the Spinal Cord and Medulla Oblongata.*

I. Vascular diseases.

(I.) Hyperæmia of the membranes.
(II.) Meningeal hæmorrhage.

II. Inflammation of the spinal dura mater.

(I.) External pachymeningitis.
(II.) Internal pachymeningitis.

III. Inflammation of the spinal pia mater and arachnoid.

(I.) Acute spinal leptomeningitis.
(II.) Chronic spinal leptomeningitis.

IV. Tumours and abnormalities of the membranes.

CHAPTER III.

I.—SYSTEM DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

(I.) POLIOMYELOPATHIES.

1. *Poliomyelitis Anterior Acuta* (Kussmaul).

Acute Inflammation of the Grey Anterior Horns.—Acute Atrophic Spinal Paralysis.

§ 393. *Definition.*—Acute atrophic spinal paralysis begins suddenly with fever, general convulsions, or other cerebral symptoms, and paralysis which reaches its maximum intensity at once. The paralysis is variable in its distribution, the affected muscles are flaccid, reflex action is diminished or abolished, some of the muscles implicated undergo rapid atrophy, and there is entire absence of sensory disturbances and of disorders of the functions of the bladder and rectum.

§ 394. *History.*—This disease was first described by Underwood in 1784, but he did not separate it distinctly from other forms of paralysis to which children are liable. The affection, indeed, does not appear to have attracted much notice until Heine, in 1840, directed particular attention to it. A good description of it was given by Barthez and Rilliet, in 1851; but it was much more thoroughly investigated about the same time by Duchenne, who named it *paralysie atrophique graisseuse de l'enfance.* In 1864, two monographs appeared—the thesis of Duchenne the younger, and that of Laborde—both of which are very important on account of the wealth of clinical facts contained in them. Dr. Müller has recently collected and analysed all the published cases of atrophic spinal paralysis in the adult from the time of Duchenne to the present day.

§ 395. *Etiology.*—The most remarkable feature with respect to the etiology of this paralysis is the strong predisposition to the affection manifested by the age of childhood. In thirty-two out of forty-four cases observed by Dr. West, the disease came

on between the age of six months and three years; while, if we analyse the cases collected by Heine, Duchenne the younger, and those observed by Barlow, more than three-fourths (154 out of 205) of all the cases occurred between the age of six months and two years. But Duchenne reports a case in a child twelve days old, and another in a child a month old, while essentially the same disease occurs in the adult.

Sex does not appear to exercise any influence in its production, nor has any direct or indirect hereditary tendency to the affection been traced. Heine, indeed, asserts that the disease attacks by preference the healthiest and most robust children.

The disease appears to be most common during the summer months; thus, out of fifty-three cases in which the date of attack could be fixed with accuracy by Dr. Barlow, twenty-seven occurred in the months of July and August.

The exciting causes of the affection are equally obscure, and it occurs frequently in the midst of robust health. Of all the alleged causes, difficult or even normal dentition is the one most frequently assigned; and it is probable that too much rather than too little importance has been attributed to this process in the production of the affection. Injuries of various kinds are often assigned as causes of the disease; while nurses are frequently blamed unjustly by parents, who, unable to believe that such a striking phenomenon as paralysis can occur suddenly without appreciable cause, imagine that the child has been lamed by a fall through the carelessness of its attendant.

Exposure to cold, more especially when the body is overheated, appears to have immediately preceded the paralysis in a considerable number of cases; and the affection often occurs in children during the progress or soon after an attack of measles, scarlatina, smallpox, typhus, and other acute affections.

§ 396. *Symptoms.*—Although this disease is essentially the same in children and in adults, yet the symptoms in each differ so much as to demand separate description. The disease as it occurs in children will be first described.

(*a*) *Infantile Spinal Paralysis.*

It will conduce to clearness of description if, like Laborde, we

divide the clinical history of this affection into four periods: (1) Invasion; (2) Remission; (3) Regression of paralytic phenomena; (4) Atrophy and deformities. It must, however, be remembered that these periods overlap, instead of being distinctly separated from each other, and that this subdivision is merely adopted for the sake of convenience.

(1) *Period of Invasion.*—The disease is commonly ushered in by a more or less intense fever, which is often preceded by general malaise, pain in the head, mental irritability, fretfulness, and startings. The fever is as a rule of short duration, lasting only from one to two days. In some cases it passes off in a few hours, while in other cases it may continue from six to fourteen days, or even longer. As the fever becomes established the cerebral symptoms become more pronounced, confusion of ideas and slight somnolency are observed, the child may become unconscious, or delirium of varying degrees of intensity may supervene.

The disease is not unfrequently ushered in by convulsions. Sometimes the paralysis occurs after a single convulsion of short duration, while at other times they are repeated many times at variable intervals before the paralysis is definitely declared (Laborde). The convulsions, according to Laborde, often assume the *tonic* form, the spasms, as a rule, being restricted to the extremities, and only extending on rare occasions to the face; and he believes that even in these latter cases the attacks are unaccompanied by any other cerebral symptoms. But in one of the cases quoted by Laborde in support of this opinion the convulsion was accompanied by unconsciousness, so that there are not sufficient grounds for believing that these attacks differ in any way from ordinary eclamptic attacks so common in children. In the cases ushered in by convulsions fever is often not mentioned as having been present, but, as Laborde suggests, it is probable that the convulsions assume such paramount proportions in the minds of the attendants that minor symptoms are not observed. In some few cases all general symptoms are absent, the child is put to bed apparently in good health and is found in the morning paralysed. In most of these cases the paralysis is limited to a portion of a limb, indicating that the primary lesion is circumscribed. It is, however, probable that in many of these cases transitory

fever and other general symptoms may have been present, and overlooked owing to the defective observation of parents.

(2) *Period of Remission.*—The initial symptoms subside in a few days and the general health improves, but when the child is taken out of bed to be bathed, or for some other purpose, it is observed for the first time that one or all the limbs hang down relaxed and powerless. The paralysis is as a rule developed with great rapidity, probably never with the instantaneousness of that caused by cerebral hæmorrhage; but it creeps on somewhat gradually during several hours, half a day or a night before attaining its acme. In some few cases the paralysis may spread more slowly, and not reach its maximum for several days. In other cases two or more attacks of paralysis succeed one another; at the first one limb is affected, and this is followed by improvement, but the child relapses in a few days into a feverish state, when another limb is found paralysed (Althaus). Still more remarkable cases are recorded by Laborde, in which the paralysis did not become permanently established until the third attack.

But notwithstanding slight variations, one of the most characteristic features of this affection is that the paralysis reaches its maximum of extent and intensity within a comparatively brief space of time from the onset. The paralysis possesses no progressive character; it recedes but does not advance further.

The distribution of the paralysis is exceedingly variable. It is frequently general, involving the muscles of the four extremities, as well as a great part of the muscles of the trunk, especially those of the vertebral column, and sometimes those of the neck. It also frequently assumes the paraplegic form; but the upper extremities are probably never exclusively affected. The disease occasionally presents itself in the form of a hemiplegia (Duchenne fils, Barlow), and in these cases the side of the neck, of the face, and of the tongue may be implicated at first, but do not remain permanently paralysed.

The sensibility is almost entirely unaffected throughout the whole progress of the disease. At the outset of the affection patients may complain of pains and paræsthesiæ, but these symptoms are of short duration. A certain degree of cutaneous

hyperæsthesia, or rather hyperalgesia, has been described as being present during the febrile stage, but the tenderness to touch described may have been due to affections of deeper structures, such as rheumatic inflammation of joints.

Reflex action, both cutaneous and tendinous, is completely abolished in all the muscles which are severely attacked, and it is much lowered or temporarily extinguished in those muscles that are only slightly affected.

The functions of the bladder and rectum are rarely affected. During the first days, however, there may be retention of urine, but more frequently there is incontinence, and the stools may be passed involuntarily. In young children a slight weakness of the bladder with occasional incontinence may remain for some time, but as a rule all disturbances of the bladder and rectum have disappeared in from three to eight days from the onset of the disease.

(3) *Period of Regression.*—After a certain time, which varies from a few days to a few weeks, a gradual improvement of the paralysis takes place. This improvement may affect a greater or smaller number of the muscles involved, and some authors think that all the paralysed muscles may completely recover. The cases in which complete recovery takes place have been called *temporary spinal paralysis* (Kennedy). Dr. Edge, of Manchester, reports an interesting case which appears to have belonged to this category. It was the case of a boy, aged ten years, in whom the muscles of both extremities as well as those of the back were paralysed. Some of the paralysed muscles were slightly atrophied, the faradic contractility was diminished, there were no bed-sores, and no disturbances in the functions of the bladder or rectum ; but there was transitory impairment of cutaneous sensibility in the lower extremities. Recovery was complete in four weeks from the commencement of the attack.

As a rule, however, there is only complete restitution of some of the muscles, while the rest remain permanently paralysed. The mode in which the paralysis recedes is peculiar. In six cases of generalised paralysis, which Laborde had the opportunity of observing accurately from the commencement of the attack, the paralysis in the upper half of the body began to

improve between the third to the fifteenth day from the commencement; and it disappeared rapidly from the neck, upper extremities, and trunk, and became restricted to the lower extremities. This improvement Laborde calls the period of *first regression*, inasmuch as it is followed after a variable interval of time by a second period of amendment which he calls the *second regression*. During the second regression there is a gradual improvement of the paralysis in both lower extremities, and the muscles of one of them may be completely restored to full power; but the paralysis becomes permanently established in one or more groups of the muscles of the other lower extremity, the anterior and external group of muscles being those most frequently left paralysed. But, although the improvement usually takes place from above downwards, it sometimes occurs in the reverse order, and then the paralysis becomes permanently localised in a superior extremity; and in rare cases it becomes localised in the muscles of the trunk or neck. In the case of a child two years of age, under the care of Dr. Simon at the Southern Hospital, the muscles of the neck alone remained paralysed, and all of these were completely paralysed and atrophied.

The chiefs facts which concern us in this affection are that the paralysis reaches its maximum of extent and intensity at once; that in all cases, without exception, improvement occurs in some of the paralysed muscles; that the improvement proceeds most actively during the first four to eight weeks, and subsequently at a much slower rate; and that this improvement may continue for from six to nine months, and under appropriate treatment may go on for one or two years from the commencement of the attack.

(4) *Period of Atrophy and Deformities.*—All muscles, in which motor power is not soon restored, become the subjects of a *rapidly progressive atrophy;* and even the muscles which are but slightly affected emaciate to some extent, but soon recover on the restoration of voluntary power.

The atrophy usually begins in the first week of the disease, and it is generally well marked in the course of a few weeks in the muscles which are severely affected. The muscles become more and more flaccid and attenuated, and after a time may

disappear so completely that the skin seems to lie immediately upon the bone. But the extent of the atrophy of the muscular substance is not always exactly measured by the loss of bulk of the muscle, inasmuch as the amount of atrophy is frequently masked by the accumulation of fat in the connective tissue. At times, indeed, the volume of the muscle appears to be increased, owing to the fatty accumulation, giving rise to the condition which Duchenne has called pseudo-hypertrophy; but in these cases advanced atrophy can be readily recognised by the extreme flaccidity and doughy feeling of the affected muscles when compared with the healthy.

The condition of the electrical irritability of the motor nerves and muscles deserves special attention. Duchenne was the first to use the faradic current as a test of the degree of alteration in the muscles; and he found that the faradic irritability of both nerves and muscles begins to sink quickly in those which are severely affected. He found, indeed, that it was materially diminished at the end of three to five days, and entirely abolished by the seventh day or during the course of the second week. He also laid down a rule which has been confirmed by all subsequent observers, and the practical importance of which it is difficult to exaggerate—viz., that all the paralysed muscles in which the faradic irritability is only more or less diminished, but not completely lost, during the course of the second week, do not remain permanently paralysed, the restoration being the more prompt and complete the less their faradic irritability has been diminished.

The galvanic irritability was first investigated by Solomon, who showed that the course of the alteration resembled that of severe traumatic paralysis. There is rapid loss of galvanic irritability in the nerves during the first two weeks of the paralysis, and the irritability of the muscles manifests the qualitative changes which characterise the reaction of degeneration. During the first weeks of the paralysis there is an increase of the galvanic irritability, the contraction on anodal is stronger than on cathodal closure, and the contraction is sluggish and protracted, instead of being instantaneous as in health. In the course of two or three months the galvanic irritability sinks again much below the normal standard, but

retains the characteristic qualitative alterations, and in the course of one or two years from the beginning of the disease there is only a trace of it generally left. In the muscles that do not atrophy, and in those which have regained their motor power, a greater or lesser diminution in the faradic and galvanic irritability is generally found, but the reaction of degeneration is absent.

Arrest of development of the osseous system is an important symptom to which Duchenne and Volkmann have directed special attention. The atrophy of the bones has no necessary relation with that of the muscles. The greater part of the muscles may indeed be lost in a limb, while the bones are almost entirely unaffected; and, conversely, a limb may be considerably shortened, while only one or two muscles are atrophied. Volkmann has indeed described cases in which the motor paralysis was temporary, and followed after a few days by a complete return of motor functions, but where the trophic lesions of the bones persisted during life.

The paralysed leg may after some years be found from two to six inches shorter than the sound limb; and the upper extremity may be similarly shortened, although not generally to the same degree. The long bones are thinner than normal, they are porous, friable, and yielding. Their epiphyses and processes grow smaller and less distinct; the paralysed hand or foot is shorter, narrower, and thinner than the sound one; and even the pelvis may be arrested in its development.

The joints become deformed and unusually movable. The increased mobility of the joints is caused partly by the disappearance of the articular extremities of the bones, and partly by relaxation and stretching of their ligaments. The relaxation of the latter is, indeed, sometimes so great that the patient can dislocate a joint without experiencing any discomfort.

The skin of the affected extremity becomes flabby; its loss of elasticity being manifested by long retention of slight pressure marks, such as that produced by the stocking. The surface of the limb assumes a mottled or bluish colour, and it is remarkably cold to the touch; the reduction of temperature in old-established cases being, according to Heine, as much as 10° to 12° F., as compared with the corresponding

ealthy limb. The skin is liable to be affected with chilblains, lcers which are slow in healing form on slight provocation, hilst the defective nutrition and great coldness of the skin ften cause a certain diminution of cutaneous sensibility. The utritive and vascular changes in the affected extremity are ccompanied by diminution in the calibre of the arteries Charcot).

Deformities.—The various deformities which occur in the ffected limbs in infantile spinal paralysis furnish very haracteristic features of the disease. Various opinions have een held by different authors with respect to the mechanism y which these deformities are produced. Some pathologists elieve that the healthy muscles are always maintained in a ondition of moderate contraction, constituting the normal *onus* of the muscles, and when this *tonus* is destroyed by aralysis in a certain group of muscles, the predominant action f their healthy antagonists produces a deformity. Volkmann, owever, denies the existence of muscular *tonus*, and thinks that ne deformity is mainly produced by the weight of the limb self. He points out that in paralytic deformities of the lower xtremities, the position generally assumed by the foot is only high degree of that which it takes when unsupported and left ee from the action of the muscles. The weight of the limb nd the direction in which mechanical forces act upon it during comotion are undoubtedly important causes of deformities; ut two other co-operating factors may be mentioned. The aralysed muscles permit the limb frequently to assume a osition in which the ends of their healthy antagonists are more r less permanently approximated, and the latter consequently ndergo adapted atrophy (Adams), and become permanently hortened. The second factor consists of an affection of the aralysed muscles themselves. The atrophied muscles are robably at times, like the bones, the subjects of arrested evelopment; their growth does not keep pace with that of ne healthy muscles and of the body generally, hence they ecome permanently shortened. Some have supposed that roliferation of the interstitial connective tissue and its subse-uent retraction may occur in the course of the atrophy of the uscles, and thus lead to permanent shortening in them; but

this is doubtful. When the deformity is caused by shortening of the paralysed muscles, the latter are found in the concavity of the distorted extremity. But whatever may be the mechanism by which these deformities are produced, it would seem that, disregarding a certain degree of inequality and disfigurement caused by the arrest of development of the long bones, paralysis of certain muscles and groups of muscles, along with relaxation of the ligaments, is the main cause of the various distortions observed.

Some of the muscles of one lower extremity suffer more frequently than others from permanent paralysis; and of these the antero-external group of the leg—the long extensor of the toes, tibialis anticus, special extensor of the great toe, and the long and short peronei—are those most commonly affected. The most frequent forms of paralytic talipes are, therefore, as might have been expected, talipes equinus and equino-varus (Plate II., 5). When the anterior group and the adductors of the foot are affected at the same time talipes equino-varus results; and when the muscles of the calf are alone affected talipes calcaneus is produced, but this form is exceedingly rare; and simple paralytic talipes varus is of still rarer occurrence. Another common deformity is the "pes cavus"—"talus pied creux" of the French—in which the sole is hollowed and the instep rendered prominent. Duchenne thinks it is caused by a more or less complete paralysis of the muscles of the calf, along with simultaneous contraction of the flexors of the foot, either the long flexor of the toes or the long peroneus. The great laxity of the ligaments of the foot allows the latter to become bent upon itself from the transverse tarsal joint, where the foot is unsupported; but when it is placed upon the ground it assumes the form of "flat foot."

Various deformities occur in the inferior extremity, according to the extent and localisation of the paralysis. The anterior and internal muscles of the thigh are most usually affected above the knee, and in that case the predominant action of the flexors of the leg on the thigh maintains the former in a permanent condition of partial flexion (Genu recurvatum), the leg being also abducted. The condition is always associated with talipes equino-varus. All the muscles of both legs are some

imes paralysed so that the patient is compelled to walk on his nees, dragging his small thin legs after him. In still more ggravated cases the muscles of both legs and thighs are permanently paralysed so that the small flexible limbs dangle bout like the limbs of a doll (membre de polichinelle).

Paralysis of the muscles of the trunk does not give rise in his disease to a true active curvature of the vertebral column, ut the attitudes imposed by other deformities may produce ompensatory curvatures. Of the curvatures directly attributable o the paralysis, *lordosis* is the most frequent and most imortant. Lordosis is caused by partial paralysis of the sacrospinal muscles, and in order to prevent the permanent bending orwards of the body by the predominant action of the flexors, he patient voluntarily throws the trunk backwards, thus relieving he weakened extensors and throwing additional weight on the exors, so that the balance between the action of the two sets f muscles is re-established. The spinal curvature which results om this action differs from other forms of lordosis, inasmuch s the pelvis is pushed forwards instead of backwards, and the uttocks become less instead of more prominent.

The deformities of the upper extremities are much less equent and serious than those in the lower extremities. The uscles of the shoulder, and particularly the deltoid, are the ost usual subjects of paralysis and atrophy in the upper xtremity. In these cases the shoulder is flattened, and the rominence of the deltoid is replaced by a more or less deep epression according to the degree of atrophy; the humerus ecomes separated from the glenoid cavity, so that dislocation ay occur spontaneously, or is readily produced; the arm hangs owerless by the side; and, to use the apt comparison of eine, dangles about like the loose end of a flail. In exceponal cases the forearm and hand may undergo distortion; but ese deformities are not of sufficient importance or frequency require description. All the organic functions are well perrmed, and the patient may live to extreme old age, as in the se of a patient observed by Charcot, who died at the age of venty, carrying with him indelible traces of the disease from hich he had suffered sixty-five years before.

The muscles are paralysed in infantile spinal paralysis in

groups, in accordance with their association in action. Particular attention has recently been directed to this point by E. Remak. In what he calls "the upper-arm type" of atrophic paralysis, the supinator longus is involved along with the deltoid, coracobrachialis, and biceps muscles. In what Remak calls "the forearm type" of infantile paralysis, as well as in lead paralysis, the extensor muscles of the hand are paralysed, while the supinator longus is spared.

Analogous facts have been observed in the various atrophic paralyses of the lower extremities. Cases of infantile paralysis are recorded by E. Remak in which the tibialis anticus and all the muscles supplied by the crural nerve, with the exception of the sartorius, were paralysed, and he therefore conjectures that the spinal nuclei of the former and those of the latter, with the exception of that of the branch to the sartorius, lie near each other in the spinal cord, and are liable to be diseased at the same time. Duchenne has proved that the sartorius is associated in its functions, not so much with the quadriceps and adductors, as with the flexors. The sartorius flexes the leg on the thigh, and the thigh on the pelvis. Bernhardt has compared the sartorius to the supinator longus, and it appears also to correspond with the latter in having its spinal nucleus near that of the flexors, and not of the extensors with which it is in anatomical relation. Cases, however, have not yet been recorded showing that the sartorius is paralysed along with the flexors of the leg, the extensors being spared, corresponding to what occurs with the supinator longus in the upper-arm type of atrophic paralysis. The tibialis anticus is also frequently spared in infantile paralysis when the other anterior muscles of the leg are implicated. Remak states that when lead paralysis affects the lower extremities the peroneal group are affected, but the tibialis anticus is spared, and he conjectures that the spinal nucleus of the tibialis anticus is on a higher level than those of the other muscles of the peroneal region.

(b) Acute Spinal Paralysis of Adults.

Acute spinal paralysis of adults is essentially the same disease as infantile spinal paralysis. The differences between the two affections result from the facts that the brain of the adul

offers greater resistance than that of the infant to the initial disturbances; that the organism of the former is not so disposed to fever; that the growth of the bones is already completed; and that the ligaments and joints are firm and resisting.

The disease begins in the adult by pain in the back and the extremities, paræsthesiæ, such as formication or numbness, and fever, which at times is very intense. There may be severe headache, vomiting, somnolency, or even slight delirium, but convulsions have never been observed.

The paralysis is developed more or less rapidly, generally in the course of a few hours, and, as in the case of children, it is more or less widely spread, complete, and associated with entire flaccidity of the paralysed muscles. Reflex action is either much lowered or abolished in the paralysed muscles, but may be retained in those which are only slightly affected. Temporary weakness of the bladder may be present at first.

The initial general symptoms pass off in a few days, soon afterwards the paralytic symptoms begin to improve, and after some weeks or months restitution of motor power may be complete. Frey has called this variety *temporary paralysis*, corresponding to the form of the same name in children. Generally, however, there is only partial restoration of motor power, some of the muscles remaining permanently paralysed. The latter muscles undergo rapidly progressive atrophy, as in the case of children, and afford the usual evidences of the reaction of degeneration. The skin becomes lax and withered and the extremities cold and cyanotic.

The sensory disturbances which may have existed at the beginning soon subside, and the sensibility becomes normal, the sexual functions are throughout unaffected, there are no bed-sores, and the general health is good.

Paralytic contractions supervene with their resulting deformities, but the latter never attain the same degree as in children, because the joints and ligaments are firmer in the adult, and the long bones of the extremities have attained their full development.

§ 397. *Course, Duration, and Terminations.*—The ordinary course of the disease is generally the same as that already

described. Cases of this disease divide themselves into two classes: (1) Those in which complete recovery takes place, and (2) those in which the recovery remains incomplete. In the first variety complete restoration of all the muscles takes place in the course of a few weeks or months. In the second variety some of the muscles remain permanently paralysed, and atrophy, with secondary deformities, results. The paralysis does not greatly interfere with the general well-being of the patient, and does not appear to have any influence in accelerating death, at least directly, although the resulting deformities may do so indirectly. Persons who have had an attack of spinal paralysis do not indeed appear to be more liable in later life to other affections of the spinal cord than healthy persons generally.

§ 398. *Morbid Anatomy.*—The main pathological changes which have been found in infantile paralysis may be subdivided into those which have been met with (1) in the muscles, and (2) in the nervous system. Changes have been found in the tendons and bones, skin and joints, but these are of subordinate importance.

(1) *Morbid Changes in the Paralysed Muscles.*—It is quite

FIG. 159.

FIG. 159 (Young). *Muscular Fibres from a case of advanced Infantile Paralysis, withdrawn by Leech's trochar.*—*a*, Muscular fibres presenting a more or less healthy appearance; *b*, muscular fibres, somewhat atrophied and with granular contents; *c*, muscular fibres greatly atrophied, but presenting faint traces of transverse striation, and having their surfaces thickly studded with nuclei.

unnecessary to describe at length the changes which occur in the paralysed muscles, inasmuch as they have already been described in the first part of this work (§ 108), along with the other trophoneuroses. By the kindness of Dr. Leech, I am, however, enabled to show in *Fig.* 159 the condition of the muscular fibres in advanced atrophy when a portion of the muscle is withdrawn by means of Leech's trochar. In almost every instance, Dr. Leech assures me that some of the fibres appear more or less healthy (*a*), while others have lost their normal striation, their contents are granular, but they are not much diminished in size (*b*). A large number are, however, reduced to slender and transparent fibres; their surfaces are covered by nuclei; the transverse striation is still distinctly visible, although it is very faint (*c*). At times two, or even three, nuclei may be seen close together, suggesting that they have been derived by proliferation from one nucleus originally. The nuclei may also be observed to project distinctly from the surface of the atrophied fibre, and it is therefore probable that they have been derived either from the nuclei of the sarcolemma or of the endomysium.

(2) *Nervous System.*—The lesions which have been found in the spinal cord are undoubtedly the most interesting and important of all those which have been observed in atrophic paralysis. For a long time theoretical arguments were adduced on the one hand to show that this disease was a nervous affection either of spinal or peripheral origin; while on the other hand it was maintained that the seat of the lesion was primarily in the muscles, and hence it was called "essential paralysis." Heine declared in favour of the spinal theory of the disease in 1860, in the second edition of his work. This view was also adopted by Duchenne, but it was not confirmed by post-mortem examination until 1864, when Cornil, a pupil of Charcot, first recognised distinct alterations in the spinal cord, and drew special attention to the atrophy of the anterior grey horns. Prévost and Vulpian, however, in 1866, were the first to make the positive observation that the essential anatomical lesion was situated in the grey anterior horn. This observation was subsequently confirmed by the observations of Lockhart Clarke, Charcot and Joffroy, and of many others.

When the anatomical basis of the disease was once established, it soon appeared that the affection was not exclusively confined to childhood. Moritz Meyer was the first to point out that essentially the same disease was met with in adults, and this opinion was afterwards confirmed by Duchenne. Reports of cases have recently accumulated, establishing the occurrence of acute atrophic spinal paralysis in adults (Hallopeau, Gombault, Bernhardt, Frey, Charcot, Seguin, Erb, Weiss, F. Schultze, Sturge, and others).

It is unnecessary to enter into a minute description of all the published reports of post-mortem examinations in cases of infantile spinal paralysis. The essential anatomical change consists in the destruction of a large number of the ganglion cells of the anterior horns, and this lesion is the cause of the paralysis and subsequent atrophy. The lesion is generally more or less diffused through the anterior grey horns, but it generally reaches its greatest intensity at the cervical and lumbar enlargements, and as a rule leaves no permanent alteration except at these points. It may extend at certain points somewhat backwards towards the posterior horns, and also forwards and outwards to the antero-lateral columns, and the anterior roots of the nerves are usually atrophied, but these are secondary changes and do not appear to be necessary to the production of the symptoms. The observations upon which this conclusion is based may be divided into those which have been observed within two years from the beginning of the disease, and those which have been observed after long intervals of time.

Unfortunately no observations have yet been made with respect to the disease during the first few days or weeks, owing to the fact that the disease of itself is not fatal. Dr. Clifford Allbutt reports the case of an infant seven months old who was suddenly paralysed in all the extremities. Death resulted in a short time from implication of the respiratory nerves. On post-mortem examination two hæmorrhagic clots were discovered in the cervical region, one of small size being situated in the left posterior horn, the other being larger and situated in the right posterior horn and lateral column. Dr. Allbutt thinks that if these lesions had been found in the lower dorsal region the infant would probably have survived, and the case

night have been regarded as one of infantile spinal paralysis. It is, however, much more probable that this was a case of hæmatomyelia. An instructive case is reported by Dr. Charlewood Turner in the Pathological Transactions for 1879. A child two and a half years of age fell on her back, but played about as usual for a fortnight afterwards, and then became suddenly paralysed in her lower extremities, and in a few days afterwards in her upper extremities likewise. On admission to the London Hospital, a fortnight after the beginning of the attack, all the extremities were completely paralysed, reflex action in them was also abolished, there was absence of sensation in the lower extremities, and the stools were passed involuntarily. The child had an attack of measles, and died about six weeks after the commencement of the paralysis. On post-mortem examination, which was made by Mr. R. W. Parker, changes were observed in the anterior horns and antero-lateral columns throughout the whole length of the cord, these being more pronounced on the left than the right side. A patch of reddened gelatinous-looking matter, about the size of a swan shot, was observed in the left anterior grey horn about the centre of the lumbar enlargement. The margin of the patch was of a darker colour than the centre, "as from the decolorisation of an hæmorrhagic extravasation." In the neighbourhood of this hæmorrhagic focus the nervous tissues were completely disintegrated, so that no nerve structure could be distinguished in the anterior grey horn, the outer part of the base of the posterior cornua. The whole grey substance was abundantly infiltrated with leucocytes, a considerable number of them being also observed in the white substance; while they were massed in great numbers in the sheaths of the larger arterioles. The vesicular column of Clarke did not appear to have been anywhere affected. In the portions of the cord which were remote from the seat of hæmorrhage, the nervous structure was not completely destroyed, although many other evidences of disease were observed. This case tends to confirm Dr. Allbutt's theory of the origin of the disease. It is, indeed, quite probable that a small hæmorrhage into the substance of the anterior horn may sometimes be the starting point of the affection.

In the Pathological Transactions for 1879 the case of a child

three and a half years of age, who had suffered from an attack of infantile paralysis at the age of seventeen months, is reported by Dr. Henry Humphreys. On admission to the Pendlebury Hospital the child presented well-marked talipes calcaneus of the left heel. Soon after admission the patient developed a severe attack of scarlet fever, from which she died. The changes observed by Dr. Humphreys in the spinal cord were limited to the lumbar region, and consisted mainly of a remarkable diminution in the number of the ganglion cells belonging to the anterior and lateral parts of the left anterior grey horn. The annexed diagram (*Fig.* 160) shows the condition of the anterior cornua at the middle of the lumbar region. Dr. Humphreys examined eighty-seven sections of the lumbar region of the cord, and averaged the number of cells they contained.

The other most notable cases which have been reported at an early period of the disease are those of Roger and Damaschino, Roth, Leyden's second case, Parrot and Joffroy, and a case briefly reported by Rinecker, which was examined by Von Recklinghausen. No marked changes were discovered in the

FIG. 160.

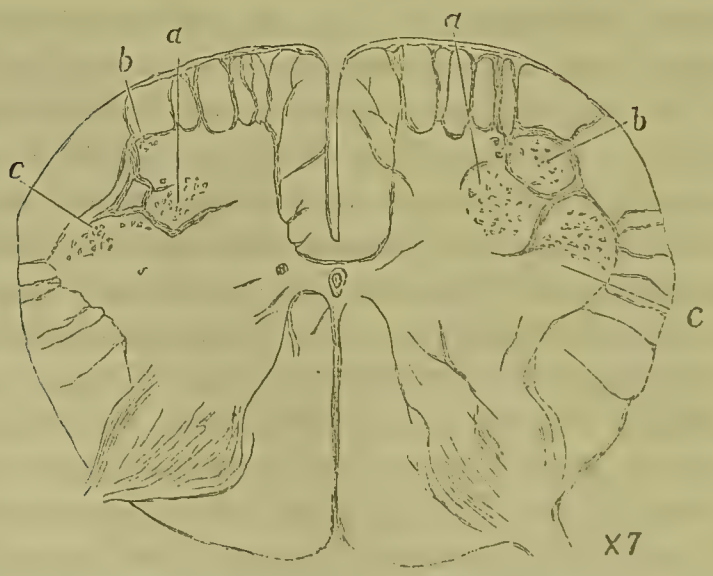


FIG. 160 (After Humphreys). The letters *a*, *b*, *c* indicate respectively the central, antero-lateral, and postero-lateral groups of ganglion cells. On the left side the group *b* has almost entirely disappeared, causing a marked falling in of the circumference of the grey matter. The groups *a* and *c* are fairly well represented on the left side, but the cells composing them are not so numerous as on the right. The internal group has disappeared from both sides.

ord with the naked eye. In some cases the substance of the ord seemed tougher at the level of the cervical or lumbar nlargements, and the antero-lateral column on the side affected ppeared atrophied and distorted. On transverse section the nterior grey horns were observed to be more or less discoloured, hitish or reddish, sometimes soft, diffluent, and diminished volume. The anterior roots at the level of the parts mainly ffected were found grey, translucent, and atrophied.

On microscopical examination, the main lesions hitherto bserved have been diffused through the grey substance of the nterior horns with areas of greater intensity in the cervical and umbar enlargements, especially in the latter. In the lumbar egion, sometimes on one side only, but usually on both sides, an rea of softening has been found in the anterior grey horn, some- imes extending the whole length of the lumbar enlargement, nd sometimes occupying only a portion of it in longitudinal xtent. The area of softening was sometimes situated towards ne centre, sometimes towards the anterior part of the horn, eing separated from the surrounding parts by a more or less harp line of demarcation. Similar areas were often also found ccasionally in the cervical enlargement of the cord, and occa- ionally in the dorsal and upper cervical portions. The substance f these areas was friable, soft, and disseminated with numerous ranulation cells. The blood-vessels were dilated, there was a arge increase of connective tissue, and the nuclei were also ncreased in number. Many of the large multipolar ganglion ells had disappeared, and of those which still remained a large roportion were observed in all stages of degeneration and trophy. The nerve fibres and axis cylinders within the area f softening were also found to have entirely disappeared.

Slighter and more diffused changes occur much beyond the mits of the softened areas. These changes consist of single ranular cells scattered through the grey substance, multiplica- ion of nuclei, dilatation of blood-vessels, and disappearance or egeneration of individual ganglion cells. Similar changes are ften observed throughout a greater or lesser portion of the grey ubstance of the dorsal region. The antero-lateral columns have ccasionally been found diminished in size, and the seat of a light sclerosis. The trabeculæ are then thickened, and indi-

vidual nerve fibres are atrophied (Joffroy and Damaschino). The anterior roots are diminished in size, and show signs of degenerative atrophy when examined microscopically.

Observations have been made from seventeen to sixty-one years after the origin of the disease, by Cornil, Prévost and David, Vulpian, Lockhart Clarke, Charcot and Joffroy, Petit-fils and Pierret, Leyden, Gombault, Déjerine, F. Schultze, and others.

The morbid changes which have been observed in these cases are generally the same as in those which have been examined within two years of the onset of the disease. The anterior horns are shrunk, and the antero-lateral columns appear to the naked eye grey, translucent, and atrophied. The posterior columns, posterior grey horns, and vesicular column of Clarke are almost, if not quite, normal.

On microscopic examination circumscribed lesions are found in the anterior horns at the lumbar and cervical enlargements, and in addition to the main lesions more or less diffused changes are met with in the grey substance and white columns. The anterior horns are atrophied and shrunk, and within the diseased foci which they contain there is a more or less firm, fibrillated connective tissue, rich in nuclei. The blood-vessels are enlarged, probably also increased in number, and their walls are thickened. Granule cells are generally absent, but a large number of corpora amylacea as well as pigment granules have been found. The multipolar ganglion cells and nerve fibres are more or less completely destroyed in the diseased foci, and some of the ganglion cells which remain are found in all stages of degenerative atrophy, pigmentary degeneration, and shrivelling. Well preserved ganglion cells may be found outside the diseased foci.

In the portions of the grey cornua which are comparatively healthy, such as the dorsal region, the ganglion cells are less numerous than normal, the connective tissue is increased, and the nuclei are abundant.

A greater or lesser degree of sclerosis of the antero-lateral columns may be discovered, the neuroglia is thickened, and generally there is some degree of atrophy of the nerve fibres. The sclerosis may vary greatly in extent. It is sometimes confined to the immediate vicinity of the anterior horns, and at

ther times it is diffused over the entire antero-lateral columns, ıe pyramidal tract being specially liable to suffer.

Fig. 161.

IG. 161 (From Charcot). *Transverse Section of the Spinal Cord* taken from the cervical region of a woman, aged fifty years, who died in the Salpétrière, of general paralysis of the insane, and who was the subject of infantile spinal paralysis of the right superior extremity. There was fibroid atrophy of the right anterior cornu, and atrophy of all the white columns of the corresponding side.

The annexed diagram (*Fig.* 161) well illustrates the morbid ılterations which are usually observed. The anterior roots are hin, grey, translucent, and the greater part of their nerve ibres are atrophied, the connective tissue is often infiltrated vith fat cells, its nuclei are abundant, and the walls of the 'essels are thickened. Almost all pathologists now regard he primary lesion as an inflammation of the anterior grey ıorns, although the cases reported by Drs. Clifford Allbutt and). Turner appear to show that a slight hemorrhage into the grey ubstance may occasionally be the starting point of the morbid ›rocess. The inflammatory process spreads along more or less liffusely over the greater part of the anterior horns, but attains ts greatest intensity in the lumbar and cervical enlargements, n which localities distinct areas of softening, and destruction ›f the multipolar ganglion cells are produced.

When the inflammation subsides, a gradual improvement takes place in those places where the destruction of the grey substance has been incomplete; but where the nerve structure has been thoroughly disintegrated there is a gradual development of cicatricial connective tissue in its place. The antero-lateral columns become secondarily affected, and when the lesion takes place during childhood they become retarded in their development, appear narrow and atrophied, and cause a considerable change in the form of the spinal cord.

Whether this affection is to be regarded as a parenchymatous or an interstitial affection is not yet settled. Charcot and others support the former view; while Roger and Damaschino, Roth and others, are in favour of the latter. Dujardin-Beaumetz, however, suggests that both tissues become inflamed at the same time, and that the myelitis is both parenchymatous and interstitial.

The peripheral nerves undergo degenerative atrophy. F. Schultze found increase of the interstitial connective tissue, with atrophy of the nerve fibres.

The tendons, atrophied and stretched, appear as thin, narrow bands.

The bones are always retarded in growth when the disease occurs in childhood, the normal protuberances and processes being less developed, and their epiphyses stunted. The medullary portion is relatively increased, its fatty contents are more abundant, and the external hard lamella of the bone is thin and friable.

The ligaments of *the joints* are thin and loose, while the articular extremities of the bones are stunted, ground off, eroded, and their cartilages attenuated. The alterations in the joints, ligaments, and articular cartilages greatly aid the muscular paralysis in the production of the different forms of club-foot, and the various other deformities already described.

The *arteries* are slightly diminished in calibre, *the skin and internal organs* are either normal or only show changes which have no necessary connection with the spinal disease. The brain is normal. In one case Sander found the two ascending convolutions and the paracentral lobule—the motor area of the cortex in relation with the paralysed parts—relatively

iminished in size, but as the spinal disease was associated vith idiocy, the connection between the atrophy of the cortex nd the spinal lesion may have been merely accidental. Par- icular attention should be paid to the condition of the cortex n future post-mortem examinations in cases of the disease.

§ 399. *Localisation of the Lesion in the Anterior Horns.*

A very interesting case in this reference is described by Prévost and)avid.* It was that of a man, aged sixty years, who suffered from ebrile and typhoid symptoms, which caused his death. The man had omplete atrophy of the muscles of the thenar eminence of the right hand, vhich according to his own account came on in childhood. The anterior oot of the eighth cervical nerve of the right side was notably diminished n size, as compared with that of the left side, and the anterior root of the eventh nerve was also slightly diminished in volume on the right side.)pposite the atrophied root of the eighth nerve the anterior horn on the ame side was observed to be sensibly diminished as compared with that of he left. The diseased portion had a longitudinal extent of about two entimetres, and the centre of the lesion was on a level with the atrophied oot. Its greatest transverse extent was also opposite the diseased root of ne nerve and it gradually diminished in size, both upwards and downwards. he author says that in the diseased portions the external or lateral (postero- ateral) group were represented by a few healthy cells, while the anterior antero-lateral) and the middle or internal (internal) groups were normal. udging from the drawing, however, the median and central groups vere entirely destitute of cells, while the antero-lateral group was only epresented by one cell.

† A case has recently been described by Kahler and Pick which appears o determine the localisation, in the anterior horns, of the spinal centres or the muscles of the calf of the leg. The case was that of a woman, wenty-four years of age, who died from an attack of typhoid fever. The nuscles of the calf of the right leg were found almost completely atrophied.)n examination of the spinal cord the right anterior grey horn was found trophied through the greater portion of the lumbar enlargement, but the nost marked changes were observed on a level with the fourth and fifth acral nerves. The roots of these nerves were also atrophied, there was a light increase of the interstitial connective tissue, which was especially vell marked in some bundles. The central group of cells was mainly ffected.

‡ In a case of atrophic spinal paralysis of adults, observed by Schultze n a man aged forty-two years, the muscles in the regions of distri-

* Archiv. de Physiologie, Serie II., Tome i., 1874, p. 595.
† Archiv. fur Psychiatrie, Bd. x., 2 Heft, 1880, s. 358.
‡ Virchow's Archiv., Bd. lxxii., 1878, s. 443.

bution of the sciatic nerve in both legs were completely paralysed, while those supplied by the obturator and crural nerves were spared. Schultze found sclerosis of both anterior horns over the whole of the lower half of the lumbar region, and he consequently concluded that the crural and obturator nerve nuclei do not lie in the lower half of the lumbar region of the cord. Remak, however, thinks that the tibiales antici were also spared in this case, and placed the nuclei of these muscles in the upper half of the lumbar region of the cord.

The condition of the upper extremity in Schultze's case was no less interesting. The muscles of the back from the sixth dorsal vertebra downwards were yellow and atrophied. The shoulder muscles and the rhomboids of the left side were atrophied, and the left trapezius was degenerated to a less degree, the left deltoid was completely degenerated, the supinator longus of the left arm was considerably altered, while the biceps and triceps were said to have been normal, and the coraco-brachialis was not mentioned. On examination of the spinal cord the left anterior horn, in the upper segment of the cervical enlargement, was smaller than the right, and appeared to be reduced to one-third of its volume. A red circumscribed spot was observed occupying the *lateral* part of the anterior horn (postero-lateral group). The condition of the dorsal region of the cord is not mentioned, but so far as the case goes it supports the idea that the muscles of the scapula and shoulder are mainly innervated from the postero-lateral group.

A case of extreme muscular atrophy is reported by Dr. Zach. Johnson, which was evidently an example of infantile paralysis, and in which "the muscles of the shoulder and arm had altogether disappeared. The muscles of the forearm remained apparently unaffected. The muscles of the ball of each thumb were almost altogether gone, while the muscles of the fingers continued to be well developed. The muscles on the dorsum of each scapula were nearly gone also."* The lower third of the cervical enlargement was alone sent to Dr. Lockhart Clarke for examination, which is a source of quite as much regret to others as it was to Dr. Clarke himself. An examination of the careful drawings made by Dr. Clarke, as well as of his description, shows that the weight of the disease falls upon the central portion of the grey substance along the distribution of the median branch of the central artery, and that the postero-lateral group of cells was the one which was principally injured. This case, then, so far as it goes, supports the opinion already advanced, that the muscles about the scapula and shoulder-joint are innervated from the postero-lateral group of cells in the cervical region. But in this case "the muscles of the ball of each thumb were almost gone." Are we to believe that they also are innervated from the postero-lateral group? The case alone certainly does not prove it. Indeed one of the illustrations shows distinctly that on one side the disease had spread forwards into the area of distribution of the anterior branch of the central artery,

* Medico-Chirurgical Transactions, vol. li., 1868, p. 249.

nd consequently amongst the median group of small cells, while Dr. larke makes special mention of a streak of disintegrated tissue which xtended backward along the middle of the posterior horns and hence in ie area of distribution of the posterior branch of the central artery. here is nothing in this case, therefore, to contradict the opinion that the uscles of the ball of the thumb are innervated from the median area.

§ 400. *Morbid Physiology.*—Infantile spinal paralysis is one f the diseases the morbid anatomy of which largely contri- uted to clear up our knowledge of the functions of the grey nterior horns. The multipolar cells probably constitute gan- lionic centres, both for reflex action and for the transmission of npulses received through the pyramidal tracts, and when they e destroyed both reflex and voluntary actions are impaired or olished according as the destruction of the cells is complete incomplete. Destruction of these cells is also followed, as e have seen, by various trophic changes in the muscles, bones, ndons, and joints.

As already mentioned, I believe that the ganglion cells of the nterior grey horns which constitute the spinal centre for the gulation of the movements of a muscle also constitute for it a ophic centre. Most muscles are, however, connected with ndamental and accessory ganglion cells, and it is only when e connection between the former and the muscle is severed at profound effects both upon its motor power and nutrition are oduced. The acute nature of the lesion in infantile paralysis, well as its localisation, is well adapted to sever the muscles om their connection with the fundamental cells, even if the tter were to remain themselves entirely unaffected. In a case served by Charcot, for instance (*Fig.* 162), the lesion occupied a sition in which only a few of the fundamental cells would be jured, yet a large number of fibres, as they converge to pass it to the anterior roots, must have been destroyed, and the fect would consequently be very similar to that which would llow a peripheral lesion of the nerve. The similarities be- veen the clinical phenomena of infantile spinal paralysis and eripheral disease of the nerves are too obvious to require inting out. The case is wholly different when we have to do ith a chronic and gradually progressive affection like pro- essive muscular atrophy, in which the accessory cells are first

attacked, and the disease by slow and successive steps gradually invades the more fundamental cells. In such a disease we may expect that the clinical symptoms of paralysis and atrophy will pursue a totally different course from that which obtains in infantile spinal paralysis.

Fig. 162.

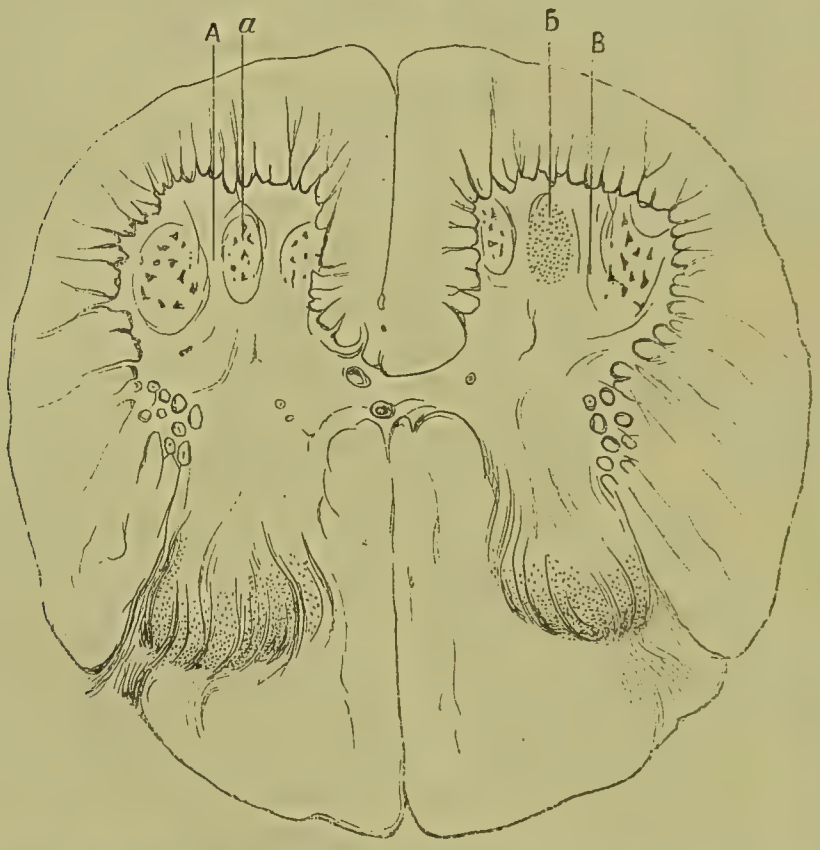


Fig. 162 (From Charcot). *Section of the Spinal Cord in the Lumbar Region, from a case of infantile paralysis.*—A, Left anterior cornu, healthy; *a*, Healthy median group of ganglion cells. B, Right anterior cornu; *b*, Median group of ganglion cells. The cells are destroyed, and the group is represented by a patch of sclerosis.

The fact that the disease occurs in certain circumscribed areas explains the distribution and extent of the paralysis, and the immunity of certain muscles and groups of muscles. The acute inflammatory nature of the process explains the sudden appearance of the paralysis as well as the fever and other violent symptoms which occur at the onset of the disease. The resolution of the acute inflammation, in part or in whole explains the rapid disappearance of the first severe symptoms

nd the partial or complete restitution of the paralysed muscles hich afterwards takes place.

§ 401. *Diagnosis.*—The symptoms of acute anterior polio-ıyelitis are so definite and characteristic that it is not easy to ɔnfound it with any other disease. We must, however, be ıreful not to regard, as true instances of this affection, every ıse of spinal paralysis which arises in childhood, even if ac-ɔmpanied with atrophy.

Hæmatomyelia, or hæmorrhage into the grey substance, say f the lumbar region, may produce sudden paralysis, which is ɔllowed by muscular atrophy, loss of reflex excitability, and the eaction of degeneration in the related parts; but the initial ver is absent, the invasion is more sudden even than that of ıfantile spinal paralysis, the former being almost apoplectiform ı its onset, and disturbances of sensibility, paralysis of the ɔhincters, and bed-sores are present.

In *acute central* or *transverse myelitis,* cutaneous anæsthesia, ıralysis of the sphincters, and bed-sores are almost invariably esent; while reflex excitability is increased in transverse yelitis, and, as a rule, there is no atrophy.

In *myelitis from compression,* disturbances of sensibility, ıralysis of the bladder, tremors of the lower extremities, in-eased reflex excitability, severe pains, and affections of the ertebral column are present; the general health usually suffers eatly, but there is no muscular atrophy.

From cerebral hemiplegia this disease may be distinguished y the different commencement of the former disease, by the ıplication of the facial nerve, and by the circumstance that cerebral paralysis there is no muscular atrophy, that the elec-ical irritability is preserved, that the bones are not affected, ıd that there is increased reflex excitability of the tendons.

Progressive muscular atrophy develops slowly and gradually, ıd the wasting of the muscular tissue precedes the paralysis, hile the electrical irritability is retained.

Paralysis following obstetric operations is observed imme-ately after birth, the initial fever is absent, the seventh nerve the one most frequently affected, while this nerve is never ermanently affected in anterior poliomyelitis. When the arm

has been paralysed by pressure of the blades of the forceps on the brachial plexus, anæsthesia remains with the paralysis.

The spasmodic spinal paralysis of children may be readily distinguished from anterior poliomyelitis by the slow and gradual development of the paresis, which rarely goes on to complete paralysis, by the muscular tension and contraction, the increased irritability of the tendons, and by the absence of atrophy and the reaction of degeneration.

Amyotrophic lateral sclerosis begins in the upper extremities, which become more or less paralysed and wasted, while the antagonists of the paralysed muscles become rigid and contracted; the arm is held tightly to the body, the forearm is flexed and pronated, and the hands and fingers are strongly flexed. The initial fever is absent, and the subsequent progress of the disease totally differs from that of anterior poliomyelitis.

The *peripheral paralysis* of single groups of muscles from pressure on their nerves by tight bandaging or other causes will be distinguished from anterior poliomyelitis by the absence of the characteristic initial stage, the strict limitation of the paralysis to the area of distribution of a single nerve trunk, the occurrence of an injury to the nerve, the presence of disturbances of sensibility, and the rapid recovery which generally takes place.

§ 402. *Prognosis.*—Anterior poliomyelitis does not appear ever directly to threaten life; and consequently, so far as life is concerned, the prognosis is very favourable. It is, however, possible that some of the children who die from convulsions may be suffering from the initial stage of this affection, although this opinion has not yet been confirmed by post-mortem examination.

So far as complete recovery is concerned the prognosis is unfavourable. In recent cases, therefore, the only prognosis we are warranted in making is, that recovery will take place to a very considerable extent, but that a certain amount of permanent paralysis, with atrophy and deformity, is likely to be left behind. The electrical reactions of the paralysed muscles form a valuable aid in prognosis. If the faradic contractility of certain muscles and nerves is diminished at the end of five days and abolished during the course of the second week, these

ill, according to the law of Duchenne, remain permanently aralysed and atrophied; and, conversely, when it is not bolished by that time, the muscles will regain their mobility, nd the restoration will be the more prompt and complete the ess their faradic irritability is diminished. After the second eek the galvanic current may be usefully employed to test he probability of the degree of recovery which may be ex- ected in the paralysed muscles. So long as a muscle, or even portion of the muscle, responds in the slightest degree to ither current, a certain degree of recovery of motor power may e expected.

The muscles that do not recover a certain amount of motor ower during the first few months seldom recover at a later eriod; and after six months of complete paralysis all hope of ecovery may be abandoned, although even then slight and artial improvement may occur under appropriate treatment.

The usefulness of the paralysed limbs may, however, be reatly improved by means of orthopœdic operations, gymnas- cs, and electrical treatment. The prognosis in this respect ill depend upon the degree and extent of the paralysis and rophy, the amount of deformity already present, the age of ie patient, and the duration of the disease at the beginning f the treatment.

§ 403. *Treatment.*—The treatment may be subdivided into iat which is appropriate during the acute initial stage and that hich is to be adopted during the subsequent stages of paralysis, rophy, and deformity. During the initial stage, when fever present, rest in bed is absolutely necessary, and leeches may e applied over the lumbar and cervical enlargements. Rubbing of mercurial ointment and counter-irritation by means of ncture of iodine and blisters have been recommended, but iese measures should certainly not be adopted until the mperature has fallen to the normal standard. Ergotine has een employed subcutaneously in doses of one-fourth of a grain r a child from one to two years of age, one-third of a grain r one from three to five years, half a grain for children from ve to ten years of age, and a grain for patients upwards of ten ears of age, repeated either daily or twice a day, according to

the severity of the symptoms as tested by the degree of fever (Althaus). Belladonna has been employed with apparent benefit in this stage of the affection. Iodide of potassium has also been administered, but it is a more appropriate remedy when the thermometer indicates that the fever has subsided, and it is then that mercurial inunction and counter irritation can be employed with benefit. For my own part I should trust during the feverish stage to the application of ice along the spine, or the employment of Priessnitz's cold compresses about the trunk, and frequently repeated small doses of aconite, given internally; and when the fever had subsided I would then give iodide of potassium internally, and use mild counter irritation along the spine, followed by the application of mercurial inunction. By the first method I would hope to arrest the active inflammatory process and limit its extension, and by the second to aid the removal of the products effused into the grey substance of the anterior horns, and thus promote recovery.

Resolution of the changes within the cord may be advanced by everything which aids the nutrition and advances the tissue changes of the body generally. A very nutritious and abundant diet should be prescribed with prolonged sojourn in the open air; mountainous or sea air is especially useful. The thermal springs of Wildbad, Teplitz, or Gastein have been specially recommended.

The constant galvanic current should be used as soon as the fever has subsided, and it should be made to pass through the diseased area of the cord. If the leg alone be affected, the current should be directed to the lumbar enlargement; if an arm only be affected, the cervical enlargement must be acted upon; and if the muscles of the trunk suffer likewise, the whole dorsal region of the cord should be included in the circuit. In order to reach the cord, it is better to place one pole on the spine, and to apply the other to the anterior surface of the trunk. The electrodes should be large, the one placed over the back being large enough to cover the entire diseased area (Erb); the force of the current should be gentle, and the application continued for from three to ten minutes according to the extent of the lesion (Althaus). Erb has recommended that

ıe current be sent through the cord first in one direction and ıen in another, but Althaus prefers the action of the positive ɔle alone. The treatment must be continued for a long time, ıd afterwards repeated at intervals for years.

At the later period of the disease, when atrophy of the aralysed muscles has set in, a peripheral application of the ɔnstant current and faradisation of the paralysed nerves and ıuscles may be combined with the application of the current) the spine. So long as the nerves and muscles have not ıtirely lost their faradic irritability, local application of the ıradic current will be of service. The constant current, however, is on the whole superior to the induced, even for peripheral application, since in the majority of cases it is the only gent which will produce any muscular response. Appropriate ymnastic exercises of the muscles, shampooing and friction, ith or without stimulating liniments, may be employed as ljuncts to the electrical treatment. When the case comes nder treatment, six months or longer after the invasion of the isease, iodide of potassium is useless, and greater benefit may e expected from phosphorous and cod-liver oil. Arsenic has so been highly recommended at this stage of the disease. he use of strychnia has been advocated, especially in the form f subcutaneous injection, but I have never seen any good esults from its employment, although I have seen the remedy ushed to an almost dangerous degree. A strenuous endeavour ıould be made to prevent the occurrence of contractures and eformities. A great deal may be done in this respect by means f electrical treatment, gymnastics, and light frictions. In uarding against talipes equinus, Volkmann advises, during ıe earliest stages of the disease, that, when the patient is lying own, the foot be fastened to a light footboard by means of flannel bandage, and its extremity drawn up somewhat ɔwards the leg. Children should wear stout laced boots, with steel shank on the outer or inner side, or with the sole slightly hicker on one side, so that the tendency to the development of alipes varus or valgus may be counteracted. The formation of alipes calcaneus may be counteracted by supplementing the efective action of the posterior muscles of the leg with a trong indiarubber band or ring, which passes from the heel

to a trough-like fixture that is applied to the leg just below the knee, and which is held firmly in its place by a side-bar fastened to the shoe. In the severer forms tenotomy and forcible means of correction must be adopted; but it is no part of this work to enter into the details of orthopœdic surgery, and the reader must, therefore, be referred to special works for the further discussion of the subject.

2. *Poliomyelitis Anterior Chronica* (*Chronic Atrophic Spinal Paralysis*).

§ 404. *Definition.*—Chronic atrophic spinal paralysis of adults presents itself as a motor paralysis associated with muscular atrophy, which begins in the lower extremities, and gradually progresses upwards until the muscles of the trunk and upward extremities are involved. The affection may terminate in death from respiratory paralysis, or in gradual recovery, the motor power returning in the reverse order to that in which it was lost.

History.—Duchenne first described this affection in 1849, then in 1852, and he gave a detailed description of it in the third edition of his *Electrization Localisée* in 1872. He believed on theoretical grounds that the disease consisted in chronic degeneration of the grey anterior horns, and consequently he designated it "Paralysie générale spinale anterieure subaiguë." Single instances of the disease have since been described by various authors, such as Poché, Frey, Erb, Webber, Cornil and Lépine, Klose, Goltdammer, Bernhardt, Aufrecht, and others.

§ 405. *Etiology.*—The causes of this disease are exceedingly obscure. All the cases which have been observed occurred in adults between the ages of thirty and fifty years.

Amongst the exciting causes the most frequent are traumatic injuries, such as a fall on the back or hip, exposure to severe cold, damp dwellings, and alcoholic and sexual excesses. Chronic lead poisoning leads to a condition very similar to chronic atrophic spinal paralysis.

§ 406. *Symptoms.*—The first symptoms are usually lassitude and fatigue in walking, with pain and stiffness in the loins and

wer extremities, which may be accompanied by slight fever, gas-
ic disturbances, and headache. The patient may also complain
various paræsthesiæ. After a time there is distinct muscular
eakness, sometimes only in one, at other times in both legs.
he muscular weakness gradually increases, the movements of
ıe ankle-joint being usually more interfered with than those
the hip-joint. After a time the paresis increases to complete
ıralysis of single muscles and groups of muscles, or of the
ıtire extremity. The muscles are flaccid and soft, and no
sistance is offered to passive movements of the paralysed
xtremities. The rapidity with which the paralysis takes place
ıries greatly. Sometimes it occurs in a few days, sometimes
ɔt till after the lapse of many months, or even years.

Soon after the paralysis is established the affected muscles
egin to waste; the calves of the legs become converted into
ose, flabby sacks, the muscles of the thigh and gluteal region
ow thin and soft, and the limbs may ultimately be reduced to
condition in which the skin appears to rest immediately upon
ıe bones. Fibrillary twitchings of the muscles usually accom-
ıny the earlier stages of atrophy. Reflex action, both cuta-
eous and tendinous, is completely abolished in the paralysed
uscles.

The sensibility of the skin usually remains normal; but
casionally the patient may complain of a slight degree of
ısensibility and numbness.

The paralysis gradually spreads to the upper extremities,
ıeir movements become awkward and feeble, and complete
ıralysis of them ultimately supervenes. All the muscles of
ıe upper extremities are not simultaneously affected; at times
ıe extensor muscles of the forearm are earlier and more
verely paralysed than the rest; at other times the flexors and
ıtrinsic muscles of the hand are the first to be attacked; and,
s a rule, the fingers and hands are more severely paralysed
ıan the forearm and shoulder. The hands assume characteristic
ositions, and the arms lie flaccid and immovable as they are
laced. Rapidly progressive atrophy ensues, which leads to the
ighest degrees of emaciation, especially in the hands and fore-
rms. Reflex action is generally abolished in the paralysed
ıuscles, sensation is normal, but the patient may complain of

numbness in the fingers, and of paræsthesia in the region of distribution of the ulnar nerve.

The muscles of the back and abdomen are occasionally implicated; the patients can no longer sit up; expiration, coughing, sneezing, and defecation are rendered difficult. The bladder, rectum, and sexual organs remain entirely unaffected. There are no bed-sores, and the general health is satisfactory.

The electrical phenomena in the paralysed nerves and muscles are the same as in acute anterior poliomyelitis, only modified slightly in correspondence with the slower development of the malady.

Duchenne showed that faradic excitability was diminished at an early period of the paralysis, and was soon entirely lost. In a case observed by Erb the nerves did not respond either to the faradic or galvanic currents, and the muscles manifested the typical reaction of degeneration.

During recovery the electrical excitability returns to the normal standard only very slowly and gradually. The further course of the disease is somewhat variable. In the majority of cases the paralytic symptoms remain stationary for a time; although the muscular atrophy may continue to advance to some extent and moderate "paralytic contractions" to be developed. After some weeks or months gradual improvement sets in, which begins in the arms and hands, and as it gradually advances from muscle to muscle the galvanic excitability of the muscles sinks more and more, and slowly gives place to the normal reaction, while the contractures also gradually disappear.

Recovery is so slow that it is only after the lapse of months that the patients can feed themselves and perform other actions with their hands.

The improvement extends after a time to the lower extremities, the movements of the hip-joint first becoming more powerful than those of the knee-joint, and last of all those of the foot and toes, until ultimately recovery may be complete.

More frequently, however, the recovery is *incomplete*. Certain sets of muscles, especially those in the region of distribution of the peroneal nerve, remain paralysed and wasted, so that the patient is partially disabled for life.

In a certain small number of cases the disease progresses upards to the medulla oblongata, when articulation, mastication, eglutition, and ultimately respiration are interfered with, and ıe patient dies from asphyxia. At other times death supervenes from simple exhaustion. The progressive cases terminate from one to four years, and the favourable cases generally last onths or years.

I am indebted to Mr. E. L. Luckman, one of the House hysicians to the Royal Infirmary, for the notes of the following ıse :—

Eliza R——, aged 15 years, entered the Royal Infirmary on August rd, 1880, under the care of Dr. Ross.

History.—She has been weakly from infancy, and has worked in the ill in a hot room since she was eleven years of age. About six months o her work caused her an unwonted amount of fatigue, and she soon terwards noticed that there was distinct loss of power in the left leg and m, followed after a brief interval of time by weakness of the left leg. ıe weakness of the lower extremities gradually increased, so that in two onths from the commencement of the attack she was compelled to leave f work. She states that she has been unable to walk for the last three onths, but it was found that, with assistance, she could make a few steps, e limbs being, as it were, dragged forward. She has entire control over e sphincters, and the only sensory disturbances complained of have en "springing" pains in both legs.

Present Condition.—As she lies in bed she has a suffering, anxious pression, and the muscles of the trunk and extremities are seen to be uch wasted. The upper lips are dry and cracked, the teeth are covered th sordes, and the tongue has a beef-steak appearance.

Left arm lies by the side, the elbow being removed two inches from the dy. The left forearm is flexed at right angles to the upper arm ; it is rongly pronated, so that the ulnar side of the hand is directed upwards. ıe hand is slightly extended on the forearm, the first phalanges are semixed on the metacarpal bones, the second phalanges are semi-flexed on e first, and the third on the second. The muscles of the ball of the umb are decidedly wasted, and those of the hypothenar eminence are so atrophied. The patient cannot produce opposition of the thumb, ıd adduction is feeble. The metacarpal bone of the thumb lies on a vel with the metacarpal bone of the index finger. The first phalanx of e thumb is extended, and slightly abducted, the second phalanx being ightly flexed on the first. The general position of the right arm correonds to that of the left. Abduction of the thumb is, however, much ore powerfully performed on the right side than on the left side, the ıgers of both hands are in a semi-closed position, the index and middle ss closed than the ring and little fingers. The interossei are atrophied,

causing deep grooves to appear between the metacarpal bones. All the movements of the different segments of the right arm can be performed, but supination of the forearm is very feeble, and can only be effected to a position midway between pronation and supination. The left hand lies powerless, in the position already described, and can be moved only to a slight extent.

The lower extremities are almost completely paralysed, and when the patient is asked to move them only a slight movement occurs, which is effected by the muscles of the thigh. The anterior muscles of the calf are quite paralysed. Both feet occupy the position of talipes equinus; but the deformity can be readily made to disappear by producing passive dorsal flexion of the foot. The different segments of the lower extremities can be readily moved upon one another, the muscles are flaccid, and there is a complete absence of the quadriceps tendo-reflex and of ankle clonus. There are no tremors or fibrillary contractions of the muscles of the lower extremity, but a few fibrillary contractions are occasionally observed in the left hypothenar eminence. The patient cannot raise herself in bed, but on being asked to do so the recti muscles of the abdomen may be felt to contract slightly, but have not sufficient power to raise the body. With the exception of an occasional dribbling of urine, the functions of the bladder and rectum are normally performed, and the abdominal muscles contract slightly during the acts of defecation and urination. When she is raised in a sitting posture she cannot hold the body erect.

At the onset of the attack she had some "springing" pains in the lower extremities, but these abnormal sensations have now disappeared. She can distinguish two points touching the surface of the outer side of the leg when two inches apart.

The sense of temperature is very accurate and that of touch good. Every form of sensibility is, indeed, perfectly normal all over the body.

The reflex of the sole of the foot, the gluteal, abdominal, epigastric, and scapular reflexes are absent.

The faradic contractility of the affected nerves and muscles is entirely abolished.

The galvanic current, applied percutaneously, obtains no response from the anterior muscles of the legs, even when fifty Leclanché cells are used.

On the current being applied by electric acupuncture, the muscles of the anterior part of the leg contract slightly with fifteen cells on cathodal closure, but do not contract on anodal.

When the galvanic current is now applied after the needles have been removed, the anterior muscles of the leg contract distinctly on cathodal closure with fifty cells.

The extensors of the right forearm contract slightly on cathodal closure with fifty Leclanché cells, but give no reaction on anodal closure.

The extensors of the left forearm give no response either on cathodal or anodal closure or opening when fifty cells are used.

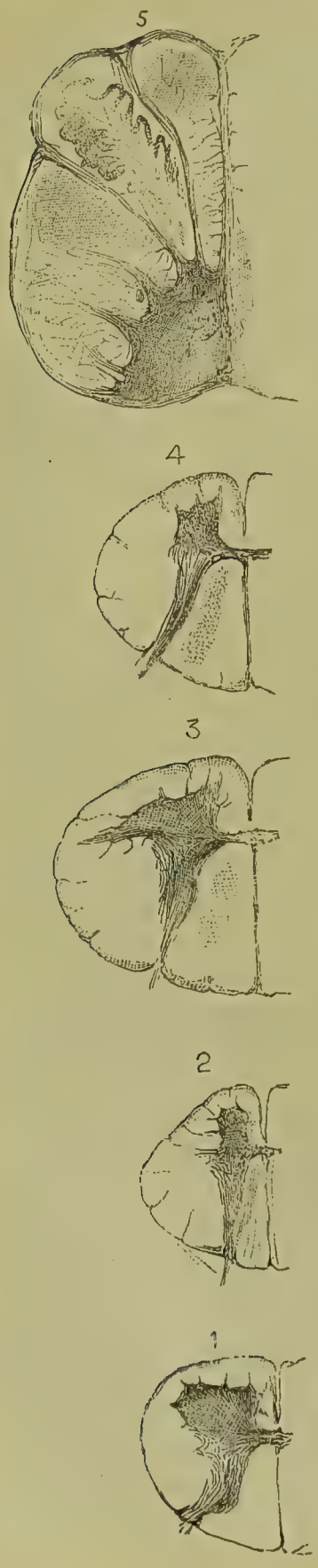
The treatment consisted of the stabile application of the constant
rrent to the spine for a few minutes daily, the current being also passed
· a longer period daily through the affected nerves and muscles. No
ısible alteration took place in her condition until the evening of Sep-
mber 16th, when the breathing was observed to be embarrassed. At
ree o'clock in the morning the hands and lips were livid; the eyes were
lf closed; her face and body were bathed in cold perspiration; the voice
ıs weak; the diaphragm had ceased to play, respiration consisting chiefly
an elevation movement, and could not be made to contract by a strong
:adic current passed through the phrenic nerves. She seemed to rally
.ittle for a short time, but the diaphragm remained paralysed.

At nine a.m., September 17th, the surface was bathed in profuse per-
iration; the skin was cold and clammy, the temperature being 97·6;
e lips and hands were livid; and the pulse was feeble and quick, beating
2 in the minute; the respirations were slow and ineffectual, but she
mained conscious to the last, and died at eleven a.m.

Autopsy.—Immediatly after death the body was placed face downwards,
e spine was covered with ice until the post-mortem, conducted by
:. Alfred Young, in the evening. No changes worth recording were
served by the naked eye in the brain, or even in the spinal cord. The
ins over the posterior surface of the lumbar region of the cord were
eatly distended. On making transverse sections of the spinal cord at
:ervals of a quarter of an inch from above downwards, it was observed
at definite areas of the white substance were of a grey colour and
latinous appearance. The grey substance of the central columns and
terior horns from the fifth or sixth cervical nerves downwards was
pressed below the white substance in each section, and appeared of
ft consistence, and was intersected in every direction by dilated and
gorged vessels.

Microscopic examination showed that the ganglion cells of the anterior
ey horns had almost completely disappeared throughout the entire length
the spinal cord (*Fig.* 163, 1 to 4). The central column and anterior horns
ere intersected with dilated blood-vessels, the walls of the vessels were
ickened, the nuclei of the neuroglia were greatly increased in number,
d the tissue was infiltrated with leucocytes. In some sections the cells
the vesicular column of Clarke appeared smaller and rounder than
rmal, but on the whole this column did not seem to be much affected
th disease. The posterior grey horns appeared normal in every respect.
ıe upward continuation of the central grey column in the medulla oblon-
ta (*Fig.* 163, 5) presented similar morbid appearances to those observed
the grey substance of the spinal cord, and the cells of the accessory
ıclei, as well as those of the nucleus of the eleventh nerve, had
sappeared; but the fundamental cells of the hypoglossal nucleus, instead
being destroyed, were hypertrophied. A few hypertrophied cells were
so observed in some sections in the centres of the internal and antero-
teral groups in the cord, especially in the cervical region, while others

Fig. 163.



were represented by small angular masses without processes; but all the accessory cells, and, indeed, the majority of the fundamental cells in the cord, had disappeared without a trace of them being left.

On holding a section from the middle of the dorsal region up to the light, a patch, which was more highly coloured by carmine than the surrounding tissue, could be distinctly observed in the posterior root-zone, where it adjoins the column of Goll. It began near the posterior commissure, and extended backwards towards, although it did not reach, the posterior surface of the cord. The deeply-stained portions were symmetrically placed on each side of the columns of Goll, and to the naked eye they presented all the characters of patches of sclerosis (*Fig.* 163, 2). Similar patches were observed in the cervical region, but they were more diffused than those in the dorsal region, their areas were larger, and they did not stain so deeply with carmine. In many sections the peripheral layer of the cord was deeply stained, this being especially marked in the anterior root-zones and columns of Türck. When the deeply-stained portions were examined microscopically, the connective-tissue septa were found swollen, a few of the nerve fibres had disappeared, but the majority of these were normal. The most remarkable morbid alteration, however, observed was the great increase in the number of Deiter's cells.

A large number of the nerve fibres of the anterior roots had undergone atrophy

Fig. 163 (Young). *Transverse Sections of the Spinal and Medulla Oblongata at different levels, from a case of chronic atrophic spinal paralysis, showing the disappearance of the ganglion cells.*—1, Middle of the lumbar enlargement; 2, Middle of the dorsal region; 3, Middle of the cervical enlargement; 4, Section on a level with the origin of the second cervical nerve; 5, Section of the medulla oblongata on a level with the middle third of the olivary body.

d some of the bundles were replaced by connective tissue. A consider-
e number of the fibres, however, appeared normal.

Portions of the anterior muscles of the leg, and of those of the hypo-
enar eminence, were subjected to microscopical examination by Dr.
ech, who kindly examined those muscles for me, and submitted the fol-
ving report:—

"On transverse section the muscular fibres are seen to be separated
an undue amount of fibrous tissue, while the nuclei of the
domysium are greatly increased in number. The fibres themselves
y in diameter, some of them being considerably smaller than others,
d the nuclei beneath the sarcolemma are increased in number.
amination of longitudinal sections shows that the muscle corpuscles are
reased in number, and that the structure of the muscular fibre is
atly altered in other respects. Many of the muscular fibres are
nular, their transverse striation is indistinct or wanting, and the
scle corpuscles are increased in number. The most remarkable changes
served, however, consisted in an alteration of the normal relation of the
tractile and interstitial discs. The contractile discs seemed to be
rtened, and swelled out laterally. In consequence of this change, the
erstitial discs appeared as transverse, more or less transparent, bands
ween the darker bars formed by the contractile discs, and the former
ng also narrower than the latter, the outline of the fibre has a rugose
serrated appearance.

"Several nuclei are sometimes observed in the transparent bands,
ile one or more muscle corpuscles are obscurely seen in the darker
s.

"It is doubtful how far the changes just described are the result of
ease, inasmuch as similar appearances may sometimes be seen, although
er to the same extent, in healthy muscle withdrawn during life by the
scle trocar; and the autopsy in this case being conducted a few hours
er death, the muscle would have been placed in preservative fluid before
t-mortem rigidity had taken place."

A case of chronic atrophic spinal paralysis has recently been
scribed by Aufrecht, in which a post-mortem examination
d been obtained, and the spinal cord, nerves, and muscles
bjected to careful microscopic examination. The appearances
served correspond on the whole pretty closely with those just
scribed. In Aufrecht's case, however, the ganglion cells of
e anterior horns were by no means changed to anything like the
ne extent they were in the case observed by me. From a
eful examination of Aufrecht's description of the morbid
erations in the anterior horns, it is evident to me that the
ndamental cells were hypertrophied, and that some of the

accessory cells were shrivelled, while probably a considerable number of them had disappeared.

§ 407. *Diagnosis.*—The *chronic* may be distinguished from the *acute* form of anterior poliomyelitis by the slow and gradual manner in which the former and the sudden way in which the latter begins. The subacute or chronic form has for some time a progressive course, and extends more or less gradually upwards, and the disease may terminate fatally or advance slowly towards recovery. The course of this disease, therefore, differs greatly from that of the acute form.

Progressive muscular atrophy may be distinguished from chronic atrophic spinal paralysis by the circumstance that in the former the paralysis and atrophy proceed side by side, while in the latter the paralysis precedes the atrophy; again, in the former the atrophy is partial, and in the latter the muscle wastes as a whole. In progressive muscular atrophy the middle form of the reaction of degeneration is met with, and reflex action is retained; while in chronic atrophic spinal paralysis the reaction of degeneration is well marked and reflex action is abolished; and, lastly, progressive muscular atrophy runs a slow and always unfavourable course, while chronic poliomyelitis runs a comparatively rapid course and frequently ends favourably. It is not improbable that some cases which are usually classed as partial progressive muscular atrophy, but which are not progressive, really belong to the category of chronic anterior poliomyelitis.

Amyotrophic lateral sclerosis resembles chronic poliomyelitis in the paralysis and atrophy of the muscles of the upper extremities, but in the lower extremities there is paralysis without atrophy along with tension of the muscles, contractures, and increase of the tendon reflexes, and only the middle form of the reaction of degeneration is met with. The diagnosis between paralysis ascendens acuta and chronic poliomyelitis will be subsequently described.

Chronic atrophic spinal paralysis may be distinguished from transverse myelitis, multiple sclerosis, tabes dorsalis, spastic spinal paralysis, and all other forms of chronic spinal disease, if due attention be paid to the state of the sensibility, the

functions of the bladder, the nutrition of the skin, reflex action, and the electrical excitability of the muscles.

§ 408. *Prognosis.*—The prognosis is comparatively favourable. Recovery takes place in the majority of cases, and improvement, as a rule, goes much further than in the acute form. At the same time it must be remembered that chronic atrophic spinal paralysis is not like infantile paralysis in being free from all danger to life. The more partial forms of the disease are never dangerous to life, although they may lead to permanent atrophy of the muscles affected.

§ 409. *Treatment.*—The same principles are applicable in the treatment of this disease as for subacute and chronic myelitis generally. Antiphlogistic treatment should first be employed, and afterwards the use of the galvanic current and a stimulating and supporting treatment.

3. *Progressive Muscular Atrophy.*

Progressive muscular atrophy is, as its name implies, a progressive wasting of the voluntary muscles, which pursues a chronic course, and attacks successively individual muscles and groups of muscles.

§ 410. *History.*—Hippocrates made a distinction between paralysis with and without wasting of the limbs, and observed that the former was incurable. Cases of muscular wasting, but without paralysis, were published in the first half of this century by Abercrombie, Darwal, Cooke, Bell, Romberg, Graves, Dubois, and Duchenne, but the affection was not recognised as a distinct disease. Duchenne, Aran, and Cruveilhier, in 1850, independently of each other, gave more accurate descriptions of the affection, and recognised its claims to be regarded as a distinct type of disease. Dr. William Roberts in 1858 collected all the information existing on the subject up to that time in an essay entitled "On Wasting Palsy;" and since that time the pathology of the disease has been investigated by Gull, Lockhart Clarke, Luys, Charcot, Hayem, Leyden, Friedreich, Erb, and many others.

§ 411. *Etiology.*—Hereditary predisposition is a powerful factor in the production of progressive muscular atrophy. Dr. Roberts collected the histories of ten families in which a

tendency to the disease prevailed; but the cases described by Dr. Meryon, which are included in this list, were probably instances of the advanced stage of pseudo-hypertrophic paralysis. After Dr. Meryon's cases are eliminated, it may be stated that out of the eight families referred to by Dr. Roberts twenty-three individuals were affected, and of these four only were females.

In a case described by Hemptenmacher, the disease could be traced amongst the branches of three families, who had repeatedly intermarried, and who had sprung from one parentage a hundred and fifty years ago. In this instance males only were attacked, but the disease was frequently transmitted through the female. Friedreich found that the disease was transmitted by a woman to her children, though they were the products of three separate marriages. Trousseau mentions a family in which the great-grandfather, grandfather, father, and son suffered from the disease, the course of which closely coincided in all the generations. Eulenburg mentions the case of a family where, out of seven children, two brothers and two sisters were attacked, while the remaining three brothers escaped.

The male sex shows a much greater tendency to the disease than the female sex. Out of 176 cases collected by Friedreich only 33 were females. The disproportion between the sexes probably depends on men being much more exposed to the exciting causes of the disease. Dr. Roberts asserts that women of the working classes, such as washerwomen, domestic servants, and sempstresses, are not much less liable to the disease than men employed in kindred occupations, while on the other hand females belonging to the middle and upper classes enjoy a remarkable immunity from the disease. It is difficult to explain the cases which arise in childhood and in which the male members of the family alone are attacked. Mr. Darwin, however, has shown that many variations which first appear in one sex are transmitted to that sex only. If this fact does not afford an explanation, it at least merges the special into a general difficulty. With regard to the influence of age, the disease is found amongst young adults and middle-aged individuals; and where there is a marked hereditary tendency to the affection, children are not unfrequently attacked. The development of the disease in advanced life is exceptional.

Progressive muscular atrophy is often developed during convalescence from acute diseases, such as typhoid fever, measles, acute rheumatism, and cholera with protracted typhoid stage; and Charcot and Joffroy have observed it to occur immediately after childbed. Venereal excess, especially onanism, has been supposed by many authors to be a fruitful source of the affection, although the evidence upon which the opinion is founded is doubtful. Chronic lead poisoning is not unfrequently attended by a diffused wasting of the muscles, closely resembling progressive muscular atrophy; and a similar wasting also occurs in constitutional syphilis.

Of the exciting causes of the disease unusual muscular exertion deserves the chief place. That excessive muscular efforts tend to develop the disease is shown by the fact that the atrophy attacks by preference the groups of muscles which must be maintained in long-continued contraction with persons following certain avocations, such as blacksmiths, tailors, masons, and shoemakers. Betz observed atrophy of the muscles of the right side in smiths and saddlers, who had to do heavy work with their right hands, and Gull observed the same in a tailor after excessive exertion. In the case of a stonemason which came under my notice the atrophy began in the muscles of the right hand. In persons who have to perform manual labour the disease generally begins in the muscles of the shoulders, arms, and hands; and the right side is generally the first to be affected. In children the atrophy not unfrequently begins in the lumbar muscles, and extends to those of the lower extremities, a mode of invasion which is probably due to the preponderant use of these muscles in standing and walking. I have observed a similar mode of invasion in a collier, who was compelled to work in a bent posture.

Exposure to cold and wet appears to be of itself sufficient to produce the disease. C. H. Richter saw a total atrophy of the hands in a man who suffered from severe sweating of the hands and who was accustomed to bathe them in ice-cold water and snow. Duménil observed atrophy of the lower extremities after long-continued standing in water while fishing; but in this case it is doubtful how much of the effect is to be

attributed to exposure to cold and how much to excess of muscular exertion. The disease is doubtless more likely to be developed when these causes are combined. Cases arising from exposure to cold are subject to neuralgic or rheumatic pains in the affected parts, hence these cases are frequently assumed to be due to rheumatism. In this class of cases the invasion is often sudden and accompanied by cramps and muscular twitching (Roberts), and the atrophy is more apt to extend to the muscles of the trunk than in cases due to overwork. According to Dr. Roberts, of twenty-five cases attributed to overwork eighteen were partial and only seven general; whereas of the sixteen cases charged to the agency of cold six were local and ten general.

Injuries of various kinds may be the exciting causes of this affection. In a youth, under the care of Dr. Roberts, who ultimately died from implication of the respiratory muscles, the first symptom of atrophy occurred in the ball of the right thumb six months after the fall of a bale of cotton on his neck. Cases similar in essential particulars are recorded by Clarke and other authors. Local injury to some of the muscles of the body is sometimes followed by progressive muscular atrophy. Friedreich relates a case in which the hand had been crushed, and subsequently the atrophy extended progressively upwards over the entire upper extremity, and finally led to the complication of bulbar paralysis. At other times the inflammatory irritation appears to be propagated from neighbouring parts, such as the shoulder and hip joints, and the disease appears at times to have been caused by cicatrices or suppurating wounds. These cases are grouped by Friedreich under the name of *myopathica propagata.*

§ 412. *Symptoms.*—The invasion of the disease is slow and insidious, and it is usually in existence some weeks or months before its presence is discovered. The patient first experiences some difficulty in performing certain movements; and on attention being directed to the affected limbs, some of the muscles are discovered to be more or less wasted. At other times, especially when the disease has been caused by exposure to cold, the mode of invasion is attended by more prominent

symptoms. Paroxysmal pains, like those of rheumatism or of neuralgia, are felt in the affected limb several weeks or months before the atrophy of the muscles is noticed, and when once the atrophy begins in these cases it proceeds more rapidly and becomes more generalised than in the painless variety.

The disease usually begins in one of the upper extremities, more commonly in the right, either in the interossei, the muscles of the thenar and hypothenar eminences, or in those of the shoulder. Eulenburg says that when the disease begins in the hand the interossei (and especially the first interosseus) are generally attacked before the muscles of the ball of the thumb; while the contrary opinion is held by Roberts and Friedreich. The opponens pollicis and the adductor pollicis are the first muscles to be affected in the ball of the thumb, while the extensors, abductor, and flexor of the thumb are spared for a long time, or may escape.

In some few cases the disease begins in the muscles of the shoulder, and in these the deltoid is almost always exclusively affected at first. When the atrophy begins in the lumbar muscles and lower extremities children are almost always the subjects of the disease, and it then frequently simulates pseudo-hypertrophic paralysis, which will be subsequently described. During the progress of the disease certain muscles or groups of muscles are attacked while their neighbours are spared, and the healthy or less atrophied muscles overcome the resistance of those more diseased, so that characteristic contractions and deformities are produced.

The disappearance of the interossei is shown by the deep furrows which appear between the metacarpal bones, the thenar and hypothenar eminences are flattened, and the disappearance of the muscles of the palm brings into view the diverging flexor tendons which are stretched between the wrists and the bulging bases of the fingers (Roberts). The deformity produced by paralysis of the interossei gives to the hand the appearance of the talons of a bird of prey; hence it has been called the claw-shaped hand or *main en griffe* (*Fig.* 77). This deformity, however, is not peculiar to progressive muscular atrophy, inasmuch as it may be caused by injury to the ulnar nerve. In consequence of the atrophy of the opponens and

adductor pollicis, the thumb is extended and abducted (Plate II., 1, 2, 3).

When the forearm is affected, the anterior, posterior, or exterior aspect of the limb is flattened according as the flexors, extensors, or supinators are affected.

When the muscles of the shoulders are affected, the arms may hang by the side or rather in front of the patient, as if they were merely attached to him by strings and did not belong to him; the natural rounded configuration of the shoulders is replaced by a hollow in which the palm of the hand may be lodged under the projecting acromial and coracoid processes of the scapula, which stand out in relief. The biceps and the other muscles of the arm may also waste, so that the limb loses its roundness and becomes flattened, and the humerus appears to be surrounded merely by the skin.

When the abdominal muscles are affected, the lumbar curve is greatly exaggerated by the unopposed action of the erector spinæ, the abdomen is loose and protruding, but the thorax is held well forwards, so that a plumb-line let drop from the most prominent of the spinous processes of the vertebræ will pass well within the sacrum, contrary to what occurs when the lumbar muscles are affected. When the atrophy is unequally distributed on both sides of the body, scoliotic or kyphotic bending of the vertebral column may be produced. When the erector spinæ and extensors of the thigh are implicated, the deformities produced, as well as the gait, are very similar to those seen in pseudo-hypertrophic paralysis, and it is unnecessary to describe them here.

When the lower extremities are invaded, deformities occur corresponding to those observed in the upper extremities; but the former are of much rarer occurrence than the latter. The various forms of club-foot may appear, especially the paralytic pes equino-varus.

The accessory respiratory muscles, as the pectoralis major, serratus magnus, trapezius, &c., are frequently implicated; and although the wasting and loss of power of these muscles do not directly endanger life, yet they may do so indirectly, inasmuch as a slight intercurrent attack of bronchitis may lead to asphyxia since the inability to make a strong expiratory effort

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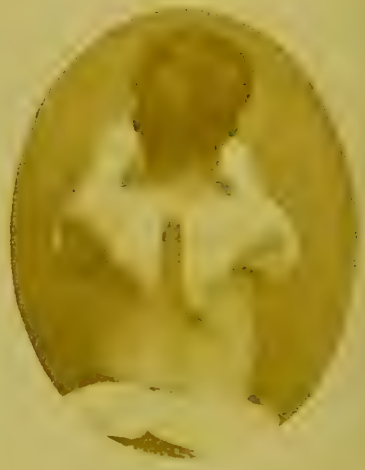

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prevents the tubes from being effectually cleared of mucus. In the later stages of the affection, the diaphragm and the intercostal muscles become affected, expectoration fails, mucus collects in the tubes, and the patient dies asphyxiated.

The facial, lingual, and laryngeal muscles, as well as the muscles of deglutition, are frequently affected towards the terminal period of the disease; but the symptoms caused by implication of these muscles will be described as labio-glosso-laryngeal paralysis.

As the following case, which is carefully reported by Mr. Cullingworth,* affords a good example of progressive muscular atrophy, where the muscles of the back and some of the muscular groups of the lower extremities are affected, I shall quote it at length :—

Charlotte A——, aged forty-one, admitted into St. Mary's Hospital, Manchester, February 3, 1878. She is married, and has had three living children, all of whom died in infancy. There is no family history of nervous disorder or of impairment of power of locomotion. Her father, an intemperate man, died of chest disease at the age of thirty-six; her mother died in her fiftieth year of heart disease. Of six brothers and sisters, two died in infancy, one from the consequences of her husband's ill-treatment, and three are living in good health.

She worked in a factory from the age of nineteen until five years ago, having had constantly two, and sometimes more, looms under her charge. The nature of her work necessitated the stooping posture, and for some years this had been a painful strain to her. She was a long time in straightening herself when the day's labour was over, and the process was not only difficult but painful. About six years ago, when she was pregnant of her last child, she was suddenly seized with an attack of unconsciousness while at her work; she fell down, and was carried unconscious home. She had other attacks of the same kind both before and after her confinement, and, indeed, had one shortly before her admission. Her husband says that there is absolutely no warning, that she frequently hurts herself in falling, that she foams at the mouth and rolls her eyes, but that there is no violent struggling. It was on account of these fits that her overlooker advised her, for her own safety, to cease work several years ago. She cannot tell exactly when the peculiarity in walking was first noticed, but is certain that she has had difficulty in rising from a chair ever since her last confinement. This difficulty has gradually increased.

She is a thin, sallow-complexioned woman, of average height, and of

* The Medical Times and Gazette, vol. ii., 1878, p. 121.

feeble intellectual power. Her lips become markedly livid on the least exertion or exposure; the whole body is sensitive to cold. There is nothing abnormal in the condition of the thoracic or abdominal viscera.

Examination while Standing.—The head is erect, and movable by the patient in all directions; the shoulders are somewhat higher than usual in a woman; the upper part of the spine is carried backwards more than usual. A plumb-line from the most prominent of the spines of the upper dorsal vertebræ falls an inch behind and away from the sacrum. The knees are very slightly bent; the feet are placed firmly on the ground, with the heels touching, the toes turned outwards and extended naturally. The upper extremities present no appearance of muscular deficiency, and the deltoids are prominent and well developed. The lower extremities present this anomaly: that while the thighs are thinner, softer, and more flaccid than natural, the calves, on the contrary, are of a size quite out of proportion with the muscular development of the rest of the body. The following measurements were taken:—Circumference of upper arm below pectorals, 9½in.; forearm at thickest part, 9⅛in.; middle of thigh, 16in.; thickest part of calf, 14½in. It will thus be seen that the circumference of the calf is nearly equal to that of the middle of the thigh. This

FIG. 164.

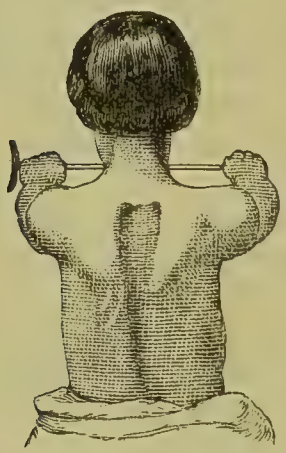


FIG. 165.

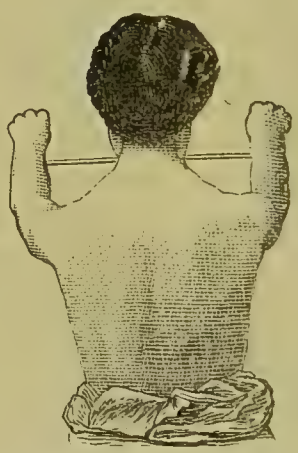


FIG. 164 shows the position assumed by the scapulæ when the arms are extended forwards. There is a deep sulcus between the two bones, the posterior borders of which project two inches from the costal wall. The posterior border of the left scapula is parallel with the median line, having been adjusted by the action of the serratus magnus, whose fibres (passing downwards, outwards, and forwards from the lower angle) are seen contracting beneath a fold of skin. This movement of adjustment has not yet taken place on the right side, where the lower angle of the scapula is nearer the vertebral column, a little higher, and altogether more prominent than on the opposite side, owing to the unopposed action of the deltoid; the serratus magnus, being in a condition of relaxation, is much less noticeable on this (right) side. The transverse fibres of the trapezius, passing from the outer half of the spine of the scapula to the last cervical and first dorsal vertebræ, are well seen; with these fibres the upper and unaffected half of the muscle abruptly terminates.

FIG. 165 shows the appearance of the back when a healthy subject is placed in the same attitude. The scapulæ are applied so closely against the costal wall that, although the person is by no means stout, the outline of the bone is scarcely traceable. The lower angle is in the axillary line.

circumstance gave rise to a suspicion of pseudo-hypertrophy, and a minute portion of muscle was withdrawn from the calf by the muscle-trocar, and kindly examined for me under the microscope by my friend Dr. Dreschfeld. The muscular tissue was not found to have undergone any change.

As the patient stands at ease, with her back to the observer, attention is at once attracted to the unusual projection of the posterior borders of the scapulæ. They stand back an inch from the posterior chest-wall, retaining their parallelism with the median line, and leaving a fossa between them four inches in breadth and an inch in depth, bounded on each side by a wall of skin, which passes perpendicularly from the scapular borders to the back of the thorax. The condition of the inter-scapular muscles can be best studied when the arms are held horizontally forwards (*Fig.* 164, and Plate II., 4 and 6). The spinal borders of the scapulæ then project backwards to a distance of two inches from the chest-wall, and approach within an inch and a half of each other, still preserving their parallelism. Between the anterior surface of the scapulæ and the chest-wall there is a deep groove posteriorly, easily admitting the tips of the fingers when they are bent over the posterior edge of the scapula. The trapezius, perfectly developed in its clavicular portion and in the upper half of its middle third, terminates abruptly by a strong bundle of fibres stretching across from the spinous processes of the last cervical and first dorsal vertebræ to the outer half of the spine of the scapula. There is not a trace of the muscle to be felt below this point. Between the posterior border of the scapulæ and the spinal column there are no muscular fibres to be felt, except one little thin band passing to the middle of the edge of the scapula. This is all that remains of the rhomboids. The latissimus dorsi has also disappeared, and there is no response to the strongest faradic current in the course either of this muscle or of the lower half of the trapezius, or of the rhomboids, except in the slender fasciculus of fibres just named. On the other hand, the levator anguli scapulæ and serratus magnus can be felt to contract forcibly, and they respond readily to a moderate current. The lower segment of the latter muscle, passing downwards and forwards from the lower angle of the scapula to the lower ribs, stands out prominently when the arms are raised; and, being uncovered by the latissimus dorsi, can be grasped underneath the skin, which is raised into a fold by the contraction of the muscle. The deep scapular muscles (supra-spinatus, infra-spinatus, sub-scapularis, teres minor, and teres major) are well developed and easily defined. The pectoral muscles are also unaffected.

The patient cannot elevate her arms vertically; the nearest approach to this which she can make is to raise her elbows until they are on a level with the ears, at a distance of about nine inches from the head. When this position is assumed, instead of the scapulæ being closely applied to the chest-wall, and rotated so as to bring the lower angle outwards and forwards as far as the axillary line, the posterior borders of the scapulæ are brought into actual contact with each other at their upper extremity, slightly diverging from above downwards, so that the lower angles are two

inches apart. The anterior face of the scapula is in the meantime one inch and three-quarters behind and away from the thorax. When the arms are stretched out horizontally right and left, the posterior borders of the scapulæ touch each other both at their upper and lower angles, on a plane one inch and three-quarters behind the chest-wall. If the patient places one hand upon the opposite shoulder, the scapula assumes the following position :—The external angle is raised along with the whole shoulder, the internal angle is depressed, the inferior angle is drawn outwards, the posterior border projects an inch from the chest-wall and is distant from the vertebral column three inches at its upper end and four inches at its lower. When, again, the shoulders are drawn back, the elbows placed by the side and flexed, so that the fingers point upwards, with their palmar aspect directed forwards, the posterior borders of the scapulæ are pressed forcibly together, closing over the vertebral column on a plane two inches behind it (see *Fig.* 166).

Fig. 166.

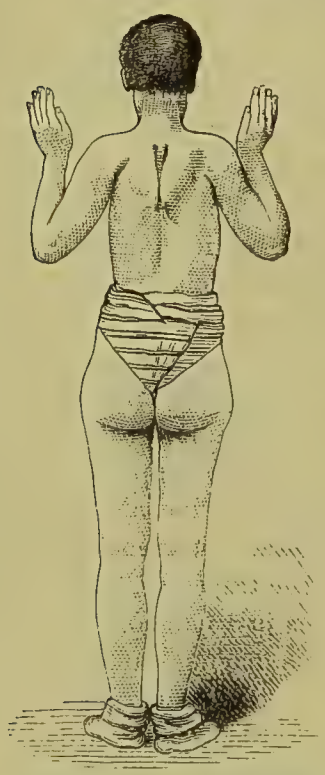


Fig. 166 shows the patient, Charlotte A——, when the shoulders are thrown back and the scapulæ brought together. The posterior borders are partially in contact on a plane two inches behind the costal wall. The sharp edge of the unaffected half of the trapezius is again clearly seen, as well as the inferior segment of the serratus magnus on each side. The levator anguli scapulæ is acting more powerfully on the left than the right. This drawing also shows the loss of rotundity in the gluteal region, and the remarkable contrast between the hypertrophied calf and the attenuated muscles of the thigh.

The patient, in attempting to carry the arm behind the trunk, cannot rotate it so as to direct the posterior aspect of the upper arm outwards, or carry the upper arm from the plane of the axilla towards the middle line of the back.

Walking.—In walking there is a general unsteadiness of gait, but no waddling. The knees are slightly flexed, and the head and shoulders are held well back, so that the feet are always kept in front of the patient. The mode of progression remains unaltered when the patient closes her eyes. Ascending stairs is a matter of extreme difficulty, and it is exceedingly painful to witness her condition of exhaustion and lividity after making the effort.

Sitting.—When about to sit down she steadies herself for a moment, grasps her thighs firmly with her hands, and falls suddenly on to the chair as though the trunk were a dead weight. At the moment, and in the act of sitting down, the trunk is in-

voluntarily thrown forwards upon the thighs, and it remains in that position until by moving the feet forward and planting them firmly, and by then grasping the knees, she pushes up the trunk into the erect posture "by force of arms."

Rising from the Sitting Posture.—She rises from the sitting posture with great difficulty, and in the following manner:—Compressing her lips, she separates the feet widely, and grasps both her knees; she then flexes the trunk upon the thighs until the trunk is horizontal, and raises the buttocks from the chair by a movement of extension at the knee. In this stooping attitude she shuffles away from the chair until she reaches something to lay hold of—mantelpiece, table, or bystander—when, having rested for a moment, she raises the hands, one by one, from the knees, and clutches firmly the object near which she has halted. Then very slowly and with great effort, keeping the back stiff and straight, she raises herself by means of the arms into the upright position. The mode in which she rises from the floor is even more painful to witness. Placing her hands on the ground in front of her, she first of all scrambles on to her hands and knees. In this posture she makes her way to the nearest available article of furniture, seizing which, she regains her feet. The trunk, however, is still horizontal, and it is the tremendous, and for a long time futile, efforts that she now makes to straighten herself that constitute the most distressing part of the performance. Striving to obtain a firm purchase with her feet, the knees being fully extended, the feet slip backwards time after time. Eventually, after violent exertions, which leave her exhausted and breathless, she accomplishes her task.

Flexion of Thigh upon Pelvis, &c.—When lying in bed horizontally upon the back, she is able to flex the leg upon the thigh, and the thigh upon the trunk, so long as the heel remains upon the bed; but when the leg is fully extended she is unable to lift the heel a single inch from the plane of the bed. As she sits, with the knees flexed, she cannot raise the foot from the ground; but the movements of adduction and abduction are accomplished fairly well. She crosses one thigh over the other by the following manœuvre:—Supposing the right thigh is to be crossed over the left, the left knee, bent to an acute angle, is adducted and pushed under the right knee, which latter is thus lifted up and carried over to the left side, resting upon its fellow.

Ankle.—The movements at the ankle-joint are performed without difficulty.

Owing to the kindness of Mr. Cullingworth I have had repeated opportunities of examining this woman, and the only addition I should like to make to his valuable report of the case is to draw attention to the gait of the patient, especially noting the points in which it differs from the gait characteristic of pseudo-hypertrophic paralysis.

In the case of this woman the feet are held close to each other, and, as Mr. Cullingworth observes, the gait is not *waddling*. In walking the head does not deviate laterally from the middle line during the transference of the centre of gravity from the active to the passive leg, but it may be observed to advance by a series of vertical curves. In his remarks on the case Mr. Cullingworth observes that the patient is unable, when lying horizontally on her side with the legs extended, to separate her thighs, thus showing that the gluteus medius and minimus are, at least to some extent, affected by paralysis. On placing one's own hands over the pelvis of the patient, one being held on each side immediately above the trochanter of the femur, it is felt that the gluteus medius on the side of the active leg does not contract during locomotion. The consequence is that, instead of the pelvis on the side of the passive leg being slightly elevated as in health, by the contraction of the gluteus medius of the opposite side, so as to allow the leg to swing forwards, it is distinctly felt to drop on that side to a lower level. The pelvis, therefore, forms a more or less acute angle with the active leg instead of, as in health, forming an obtuse angle with it. It is, however, maintained in a nearly horizontal position by the fact that the active leg itself slants downwards and inwards from the hip-joint. The line of gravity passes through the pelvis about its middle ; and in order that it may pass through the arch of the foot of the active leg, the latter must occupy a position directly below the middle of the pelvis, and consequently the hip of that side projects outwards. The passive leg is prevented from swinging forwards with the normal pendulum motion, inasmuch as the hip-joint on that side becomes lower when the leg is raised off the ground, instead of being elevated by contraction of the gluteus medius of the opposite side as in health. The necessary elevation of the passive foot is obtained by strong flexion of the thigh upon the body, so that the legs, as described by Mr. Cullingworth, appear to be in advance of the body. The alternate projection of the hip on the side of the active leg, and the alternate falling down of the hip on the side of the passive leg during successive steps, render the gait of this patient totally unlike that which is so characteristic of pseudo-hypertrophic paralysis.

The loss of muscular power keeps pace with the atrophy, and is, as a rule, directly in proportion to the degree of the latter, and so long as any muscular fibres are left, they can be made to contract by voluntary effort. For a very long time, indeed, the various movements are capable of being performed, although with much diminished power, and it is only in the last stage of the affection that complete immobility of the limb is produced.

At times the loss of motor power apparently much exceeds

the loss of muscular substance; but in these cases the bulk of the muscles is maintained or even increased by an interstitial fatty hyperplasia, while the individual muscular fibres are atrophied, so that the disproportion between the loss of muscular power and the loss of muscular substance is only apparent and not real. This condition will be more fully described when the closely-allied disease called pseudo-hypertrophic paralysis comes under consideration.

The *reflex movements* are occasionally exaggerated, but this modification is not constant (Jaccoud).

The *electrical reaction* of the atrophied muscles, as a rule, corresponds closely with the diminished volume of the muscles and the loss of voluntary power. The normal faradic contractility is maintained until the muscle has undergone a high degree of atrophy, and it is only in the last stage of muscular atrophy that the excitability is diminished or abolished. It need scarcely be added that, although the faradic excitability is not diminished, yet the energy of contraction becomes weaker and weaker in proportion as the contractile elements of the muscle disappear. The faradic excitability of the nerve-trunks is retained longer than that of the muscle, and both disappear some time before complete loss of voluntary power occurs. Galvanic muscular contractility usually remains normal for a long time, although the energy of the contraction diminishes in proportion to the degree of atrophy, so that stronger currents are required to produce a minimum contraction (Eulenburg). The galvanic excitability of the nerve-trunks also remains unimpaired for a long time. Rosenthal has directed attention to the fact that the nerve-trunks behave differently at different points in their course, so that while electric stimulation applied to a portion situated near the centre may produce normal effects, its results may be less than normal, or entirely wanting when a more peripheral tract is stimulated. Slight qualitative changes in the muscular reaction may attend the ultimate stage of atrophy.

Fibrillary contractions of the affected muscles are frequently observed during the entire active stage of the disease. These consist of vibratory tremors or quivering of the muscular fibres. They occur spontaneously, but may be provoked by

gently tapping the surface, by exposing to the air parts which are usually covered, by electrical excitation, and by active or passive movements of the affected muscles. These fibrillary contractions are sometimes the earliest symptoms of a fresh advance of the disease into parts previously unaffected, and they disappear altogether when the atrophy has reached an extreme degree, or when its progress is arrested (Roberts).

Occasionally clonic or tonic contractions of entire muscles, or groups of muscles, may occur, accompanied by intense pain, analogous to the well-known cramp of the calf.

The sensibility, as a rule, is entirely unaffected. In some cases, however, the atrophy of the muscles is preceded by paroxysms of pain in the affected parts. At times the pains follow the course and distribution of single nerve trunks, as that of the median and ulnar nerves; but at other times the pains appear to have their origin in the sensory nerves of the muscles. In the latter case compression of the affected muscles, as well as active and passive movements of them, provokes or aggravates the pain, and in some cases the electro-muscular sensibility seems to be increased. In the later stages of the affection a moderate degree of anæsthesia may be present, especially in the hands and tips of the fingers, in the form of blunting of common sensation. The farado-cutaneous sensibility may also be diminished, and complete analgesia of circumscribed areas is not uncommon. Sensations of cold and numbness in the finger tips, formication, and other sensations are frequently observed.

Vaso-motor disturbances of various degrees and extent may occur in the affected regions. In the beginning the temperature of the affected extremities is increased. Baerwinkel found an elevation of 1° in one case, and Frommann found in the side first attacked a rise of 0·2° or 0·3° C., as compared with the opposite side. In more advanced stages the temperature is not raised, and at a still later period a distinct lowering occurs, which, according to Rosenthal, may amount to 4° C., and according to Jaccoud to 3 or 4° C. below the normal.

The affected parts are cold and pale, and this is especially likely to be the case in the hands. This local ischæmia is followed by relaxation of the vessels, and consequent warmth

and redness of the affected part. An excessive sweating (hyperidrosis) of a generalised character occurs in the later stages of the affection, but whether this is due to vaso-motor disturbance is unknown (Frommann, Friedreich).

Trophic disturbances occur at times in other tissues in addition to the muscular affection. The skin is not unfrequently implicated; and in these cases both the epidermis, cutis, and subcutaneous tissues are affected. The affection of the skin may be entirely wanting, and scarcely ever reaches a high degree, even where the muscular disease is far advanced. Painful swellings of the joints have been observed in the early stages of the disease (Remak). These swellings (arthritis nodosa) generally occur in the phalangeal joints, and are in all probability closely related to the arthropathies of tabes dorsalis, except that the latter are more frequent in the large than in the small joints.

Oculo-pupillary symptoms are on rare occasions observed in this disease. They consist of flattening of the cornea (Voisin), and contraction and sluggish reaction to light of one or both pupils (Voisin, Baerwinkel, Schneevogt, Rosenthal). These symptoms are in all probability due to paralysis of the sympathetic fibres of the iris.

In the early stage of progressive muscular atrophy the patient may complain of chills, and there may be a continuous, though slight, increase of temperature, which lasts for days or months. This febrile condition may sometimes be associated with arthritis nodosa, and may probably be due to the affection of the joints (Remak). In the later stages of the disease transitory or permanent elevations of temperature may occur, which are perhaps due to such complications as diseases of the lungs or acute bed-sore. No constant changes have been found in the urine.

§ 413. *Course and Duration.*—The course of progressive muscular atrophy is essentially chronic. It may at times be permanently arrested after a certain group of muscles is destroyed, but it may progress steadily until nearly all the voluntary muscles are implicated and the unfortunate patient is reduced to such utter helplessness that he cannot raise a hand to feed

himself or turn himself in bed. The advance of the disease is seldom continuous even when it is progressive, but is interrupted by repeated remissions. These may extend over a few weeks, months, or years. Dr. Roberts thinks that the cases caused by over exercise of the muscles nearly always terminated in permanent arrest of the affection after the destruction of one or more groups of muscles; while cases which were caused by exposure to cold, or in which a decided hereditary predisposition could be traced, showed a greater tendency to a progressive course and a fatal termination.* In some few cases the atrophied muscles may by treatment be restored to their former bulk, but the affected muscles usually remain disabled to a more or less extent for the remainder of life.

The duration of the disease is very variable and uncertain. In twenty-eight cases analysed by Dr. Roberts the mean duration was thirty-eight months; of these four cases ended in recovery, their mean duration being fourteen months; in thirteen cases the disease was arrested with a mean duration of twenty-seven months, and the remaining eleven cases died with a mean duration of the disease of upwards of five years.

§ 414. *Morbid Anatomy.*—The essential anatomical changes found on post-mortem examination of those who have died from progressive muscular atrophy are confined to the muscles the spinal cord, and the nerves.

The *muscles* of the affected regions are wasted in various degrees, and even different parts of the same muscle may present differences in the degree to which the atrophy has extended. A small portion of an affected muscle may, indeed, retain its normal bulk and appearance, while the rest is reduced to a fibrous band. The altered muscles are generally of a pale red or rose colour, while at other times they may be buff or ochre, and streaks of adipose tissue may be seen to run in lines between the fibres.

The early investigators (Meryon, Duchenne, Cruveilhier, Wachsmuth, and Valentiner) regarded the muscular changes as being the result of fatty degeneration of the fibres, with secondary disappearance of the sarcolemma; but the labours of

* Reynolds' System of Medicine, Art. Wasting Palsy, vol. ii., p. 172.

ecent investigators (Robin, Friedberg, Foerster, Schueppel, Hayem, and Friedreich) have shown that the fatty metamorphosis of the primitive fibres is a secondary result of a previous inflammatory change. The first changes begin in the perimysium internum, as a hyperplastic growth of the interstitial connective tissue in its finest ramifications among the single primitive bundles. Swelling and multiplication of the muscular corpuscles, along with proliferation of their nuclei, may be observed, and at times parenchymatous granular cloudiness of the transverse striped fibrillary substance. Friedreich says that he has observed hypertrophied muscular fibres along with a trichotomous or trichotomous division of their fibres. Wasting of the muscular substance goes on side by side with increase of the interstitial tissue, a process which ultimately leads to a fibrous degeneration or true cirrhosis of the muscle. A development of fat may take place within the hyperplastic connective tissue, leading to a pseudo-hypertrophy of the muscle.

The condition of the spinal cord and of the anterior spinal nerve roots has been examined, according to Eulenburg, in forty-nine cases, and out of these positive changes have been found in thirty-four, while in fifteen the results were negative. If, however, the special methods and special skill which are required for conducting the examination of the spinal cord be taken into consideration, too much weight need not be attached to the negative statements. In the hands of experts in the present day changes are almost always found in the cord, hence the negative results of the older observers may be fairly attributed to defective methods. Cruveilhier was the first to draw attention to the condition of the anterior roots of the nerves in this disease. In the body of the showman, Le Compte, who died from progressive muscular atrophy of five years' duration, he found that the anterior roots, especially in the cervical region, were remarkably small as compared with the posterior roots; and in a second case, observed by him, a similar condition was found. In these cases the brain, cord, and posterior roots were stated to be normal. Atrophy of the anterior spinal roots, either with or without other morbid changes, has since that time been found by various competent observers, amongst whom may be mentioned Clarke, Vulpian, Luys, Rosenthal,

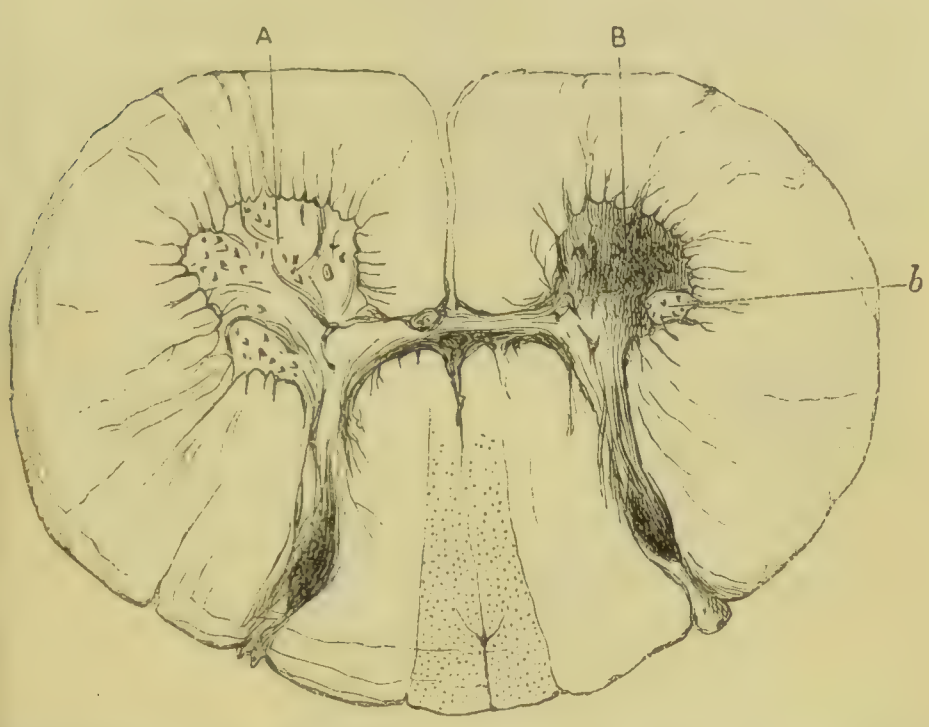
Hayem, Charcot, Joffroy, Friedreich, and many others. The anterior nerve roots have in some cases been found normal by Clarke, Frommann, Gull, Friedreich, Türck, Von Recklinghausen, Joffroy, and Frerichs. It may, therefore, be concluded that the atrophy of the anterior roots is not the essential morbid change. Valentiner, in 1855, found a central softening of the grey matter in the neighbourhood of the lowest cervical and the uppermost dorsal nerves; and Schneevogt also found a softening of the cord from the fifth cervical to the second dorsal nerves. Frommann observed changes in the anterior column and anterior commissure extending from the medulla oblongata downwards.

Luys, however, was the first to direct attention to the morbid changes in the grey substance. A man, the subject of advanced atrophy of the muscles of the left hand and forearm, along with slight atrophy of the muscles of the right hand, having died of pneumonia, the spinal cord at the autopsy appeared healthy to the naked eye, but microscopical examination showed increase of the capillary vessels in the grey substance of the cervical enlargement. The walls of the vessels were thickened and surrounded with granular exudation, which extended into the adjacent tissues. Many corpora amylacea were scattered through the grey substance. A considerable number of the ganglion cells of the anterior horns had disappeared in the part affected, and were replaced by granular masses, and of the remaining cells some were in a condition of degeneration, of a brownish colour, full of dark granules, and destitute of processes. The degeneration affected principally the left anterior cornu, corresponding with the seat of the muscular atrophy. The anterior nerve roots on the left side, corresponding to the disease in the anterior horn, were atrophied. Six cases have since been described by Lockhart Clarke, confirming, in all essential respects, the observations of Luys; and similar observations have been made by Duménil, Schueppel, Hayem, Charcot, and Joffroy.

In a case described by Charcot the ganglion cells of the left anterior grey horn (*Fig.* 167, A) could still be distinguished, but were observed to be in an advanced stage of atrophy. In the right anterior grey horn (*Fig.* 167, B), however, the cells

ould only be distinguished in one group—the postero-lateral *Fig.* 167, *b*)—while the cells of the remaining groups were ompletely destroyed. It has appeared to me, however, that oo little attention has hitherto been paid to the condition of

FIG. 167.



IG. 167 (Charcot). *Transverse Section of the Cervical Region of the Spinal Cord, from a case of progressive muscular atrophy.*—A, Left anterior grey horn; the ganglion cells have persisted, but are much altered in appearance. B, Right anterior grey horn, almost complete atrophy of the cells, one group only (*b*) having persisted.

ie central column. In the annexed diagram, from Charcot *Fig.* 167), the central column, especially the left one, is seen to e intersected by enlarged vessels, and that of itself affords me evidence that this column was not free from disease in the ction from which the drawing was taken. It seems to me, deed, that the morbid process begins on each side of the ntral canal, probably in the tissues immediately adjoining e central artery, and that it extends outwards and forwards well as upwards and downwards from this point as a centre. a transverse section of the middle of the cervical enlargement my possession, from an advanced case of progressive muscular rophy, the material of which I owe to the kindness of r. Dreschfeld, it was unmistakable that the central grey

column was more severely diseased than any other portion of the section. The central column was traversed by enlarged vessels, and almost all structure was obliterated, while the various groups of ganglion cells in the anterior horns were distinctly recognisable. The cells of the median area were, indeed, completely destroyed, so that not a trace of them could be seen, and a large number of the marginal cells of the other groups were also destroyed, so that the groups themselves were separated by unusually large spaces which were destitute of cells (*Fig.* 168). The cells of the centres of the groups were, however, distinctly recognisable, although all of them were observed to be in a

FIG. 168.

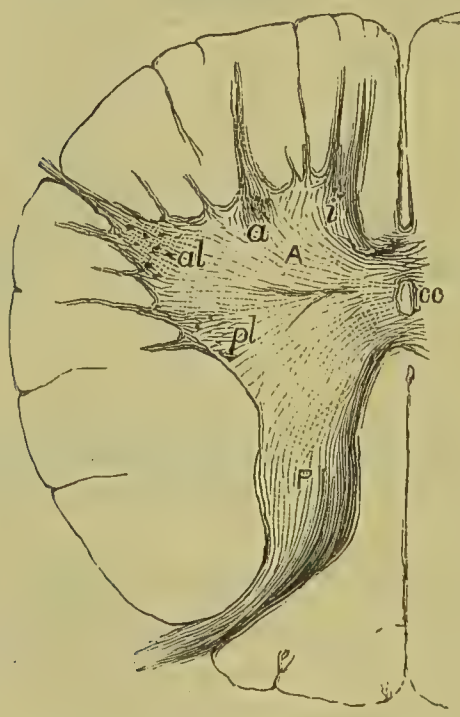


FIG. 168 (Young). *Transverse Section from the Middle of the Cervical Enlargement of the Spinal Cord, from an advanced case of progressive muscular atrophy.*—*cc*, Central canal; *i*, Internal, *al*, Antero-lateral, and *pl*, Postero-lateral groups of ganglion cells.

state of pigmentary atrophy (*Fig.* 144, 8). I have also observed in one of my sections a streak of degeneration to pass along the posterior branch of the central artery (*Fig.* 111, 1″) into the substance of the posterior grey horns, and this may explain why analgesia of patches of the skin is frequently associated with

progressive muscular atrophy. In the accompanying woodcut (*Fig.* 169), borrowed from Leyden's great work on the diseases of the spinal cord, it may also be distinctly recognised that the diseased portions occupy mainly the central columns of the cord, and that there are lateral extensions of the disease towards the anterior grey horns and between the groups of ganglion cells.

FIG. 169.

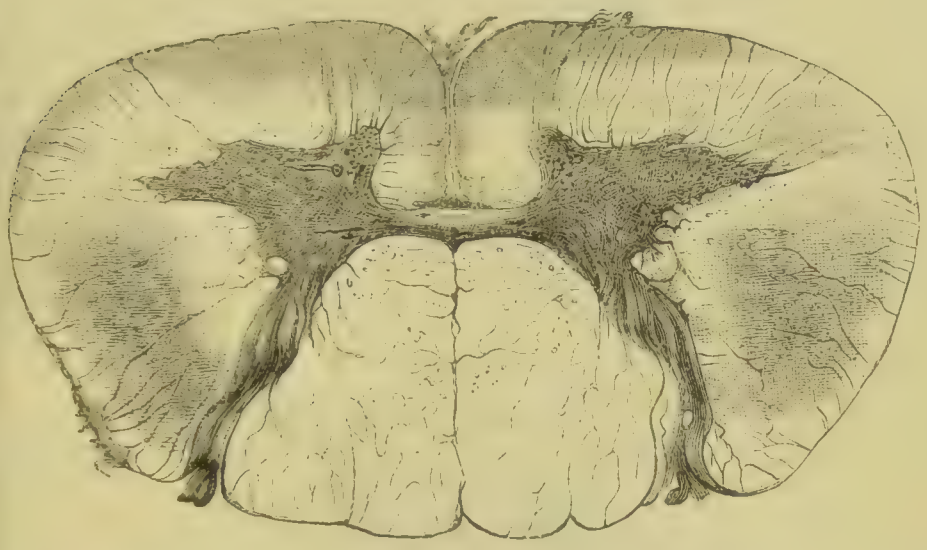


FIG. 169 (From Leyden). *Transverse Section of the Spinal Cord from the Middle of the Cervical Enlargement, showing that the central column and a large portion of the anterior grey horns are diseased.*

A case of progressive muscular paralysis has been recently described by Erb and Schultze, in which the erector spinæ throughout their entire extent, the trapezius on both sides, the muscles connected with the shoulder blades, those of the upper arm, the pectorals, the gluteal muscles, and the flexors of the legs on the thighs were atrophied. The case, indeed, appears to have been, so far as the distribution of the paralysis is concerned, very like that of Charlotte A——, already described. The patient died from an attack of syncope, but without any trace of bulbar symptoms, and a microscopical examination of the spinal cord showed that the most pronounced changes were found in the "central region of the grey substance." It is also mentioned that in the lower half of the lumbar and cervical enlargements the ganglion cells had disappeared from the median (central) group. The cells of the other groups were

degenerated. The whole microscopical report of the case, indeed, bears out the idea that the disease began in the central column, and extended forwards into the anterior horns.

The nature of the disease in the cases just described appears to have been a chronic inflammation of the grey matter, but in another series of cases the affection of the grey matter is caused in a different way.

In the annexed woodcut (*Fig.* 170), borrowed from Leyden's work, a section of the cervical enlargement of the spinal cord, from a case of syringomyelia, is represented. It will be recognised that the destruction of the ganglion cells of the anterior horns takes place in this case much in the same way as in cases of chronic inflammation already described, but in which distinct cavities are not observed. It is indeed doubtful whether any essential difference exists between the two classes of cases,

FIG. 170.

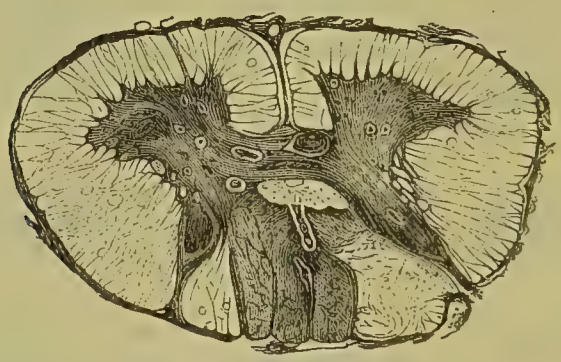


FIG. 170 (From Leyden). *Transverse Section of the Spinal Cord from the Middle of the Cervical Enlargement, from a case of Syringomyelia, showing a cavity behind the posterior commissure, and destruction of a large portion of the ganglion cells of the anterior grey horns.*

inasmuch as the cavities probably result either from inflammation of the tissue of the central grey column and of the adjoining white substance, or of the walls of the central canal.

In a case observed by Sir William Gull, a considerable dilatation of the spinal canal was found in the cervical region, between the fifth cervical vertebra and the origin of the third and fourth dorsal nerves. The cavity was full of serous fluid, and, with the exception of a thin layer which surrounded it, and could be stripped off like a membrane, the grey substance had disappeared, while the white substance and the anterior nerve

oots seemed normal. In a case observed by Schueppel, the ydromyelia extended down to the tenth dorsal vertebra. Grimm also found considerable enlargement of the central avity at the expense of the grey substance, the latter being lattened by pressure to a ring-shaped plate, but in this case he cord in the lower cervical and upper dorsal regions pre-ented a fusiform swelling, caused by the growth of a medul-ary sarcoma. In the latter series of cases, the destruction of he ganglion cells appears to have been caused indirectly by he pressure of the fluid which had distended the central canal.

The result, however, in both series of cases, is the same, the anglion cells being destroyed. Progressive destruction of the ells seems then to be the essential feature of the morbid natomy of the disease. It is right to add that some competent bservers (Lichtheim) found the anterior grey horns normal, ut it is probable that, in the earlier observations at least, the hanges in the ganglion cells had been overlooked. The posterior olumns, the posterior horns, the posterior roots, and the inter-ertebral ganglia have been found in a state of degeneration, n addition to the affection of the anterior horns.

Schneevogt found the sympathetic in the neck diseased in a ase examined by him. The cervical sympathetic was converted lmost into a fatty cord, in which the nerve fibres were pressed side by fat cells, which contained beautiful crystals. The cer-ical ganglia were almost entirely changed into fat cells, and he thoracic part of the sympathetic also abounded in fat. Two imilar observations were made by Jaccoud, and he inferred rom the appearances present that the disease began in the ympathetic and spread along the rami communicantes to the ord and along the peripheral nerves to the muscles. Changes n the sympathetic have been found by Swarzenski and by)uménil; while other observers as Landry, Frommann, Hayem, Charcot and Joffroy, and Friedreich have found the sympa-hetic healthy.

The peripheral nerves distributed to the affected muscles ave also been found affected by Schneevogt, Trousseau, Virchow, Freidberg, Hayem, Charcot and Joffroy, Bamberger, Rosenthal, Friedreich, and others. The degenerations were ometimes limited to the finer intra-muscular nerve branches,

and at other times they existed in the large nerve trunks and in the plexuses. The changes observed were hyperplasia of the neurilemma, multiplication of nuclei, and fibrillary thickening of the sheath of Schwann. Friedreich also found varicose dilatation of the medullary sheath with secondary atrophy of the nerve fibres. In some few cases examination of the peripheral nerves gave negative results.

§ 415. *Morbid Physiology.*—Various theories have from time to time been advanced to account for the symptoms of progressive muscular atrophy. The theory of Cruvielhier, who regarded atrophy of the anterior roots as the essential morbid alteration, as well as the views of those authors who believe that the symptoms are caused by disease of the peripheral nerves, antero-lateral columns, or posterior columns of the cord, is definitely abandoned by all pathologists in the present day. The theory first advanced by Schneevogt, and afterwards elaborated by Jaccoud, which attributes the disease to a morbid change of the sympathetic, now reckons only few adherents. In the first place, the sympathetic is by no means regularly affected in progressive muscular atrophy; and when it is implicated the morbid change is declared by the superadded symptoms, which may be briefly summed up as oculo-pupillary phenomena. In short, the disease of the sympathetic is an occasional concomitant affection; and in these cases the morbid changes appear to be propagated from the anterior horns along the rami communicantes to the cervical and dorsal portion of the sympathetic trunk and ganglia. It is not impossible that a reverse process may take place, but even in that case the disease of the anterior horns, so far as the nervous system is concerned, would be the fundamental lesion of the affection.

These theories being disposed of, two only remain—the myopathic theory, of which Friedreich is the declared champion, and the neuropathic theory, of which Charcot may be mentioned as the most prominent advocate. Aran first advanced the myopathic theory, but in his day the constant changes which have since been found in the nervous apparatus had not been discovered. No one denies the reality of these changes at present, the only question which arises is whether the muscular alteration pre-

edes the morbid changes in the cord, or whether the former are econdary to and caused by the latter. Progressive muscular atro-hy, according to Friedreich, begins as a primary chronic myositis. The intra-muscular nerves are secondarily implicated, and a hronic neuritis ascends along the course of the nerve trunks o their roots. The neuritis may then extend in the cord itself, roducing a chronic myelitis, which may spread in various irections. This change may extend to the anterior cornua, ut the nutrition of the peripheral nerve fibres and of the anglion cells of the grey anterior cornua of the cord is also ffected by the disturbed motor functions, caused by the disease n the muscles. Various objections may be urged against this heory, not the least important of these being the fact that the eripheral nerves and anterior nerve roots have been found uite normal in a considerable number of cases.

Friedreich would supplement the theory of neuritis ascendens y the subordinate theory that simple suspension of muscular ction would of itself cause atrophy of the ganglion cells. But ne changes observed in the anterior horns of the cord in the ase of amputated limbs do not equal in severity those found n progressive muscular atrophy. This theory also utterly fails o account for those cases in which the destruction of the grey natter of the anterior horns is produced by slow compression rom gradual distension of the central canal by fluid. Various ther objections might be urged against the myopathic theory, ut enough has been said to show that it at least presents ide gaps which must be filled up before it can be considered stablished.

The neuropathic theory has at least the merit of being mple, and of presenting fewer difficulties. According to it, ne atrophy is due to the progressive changes, primarily of an ritative character, of the ganglion cells of the anterior horns. rogressive bulbar paralysis, which is so frequently associated ith progressive muscular atrophy, is an analogous affection aused by morbid changes in the groups of motor cells lying in ne floor of the fourth ventricle, the reason that the two diseases re so frequently associated being merely that the morbid rocess extends from the anterior horns by continuity to the notor centres in the floor of the fourth ventricle. The dis-

tinction between the two diseases is, indeed, dependent upon the locality of the lesion in each case. In both these diseases the nature of the lesion which destroys the ganglion cells is of little importance. Much the same results (except probably in respect to the rapidity with which the atrophy is developed) follow ordinary grey degeneration, chronic induration, myelitis, red softening, Clarke's granular degeneration, or isolated pigmentary degeneration of the cell elements.

On the supposition that the disease begins in the central grey column and extends outwards and forwards into the anterior grey horns, it may be readily explained why the groups of muscles engaged in special actions are usually the first to be affected. We have already seen that the central column is the embryonic area of the grey substance, and that the median area of the anterior horns in the lumbar and cervical enlargements, and the medio-lateral areas in the dorsal and upper cervical regions of the cord, may be regarded as outgrowths of the central column. The median and medio-lateral areas will consequently be the first portions of the anterior horns to be affected, and the portions which contain the fundamental cells will be the last to become diseased. When, therefore, the lumbar and dorsal regions of the cord are affected, the muscles which maintain the erect posture in man will be those most liable to be affected, as will be more fully pointed out with regard to pseudo-hypertrophic paralysis. Again, when the cervical enlargement is the first to be affected, the morbid process will extend more readily forwards to the median area than in any other direction, and the small muscles of the hand will be first affected. It will hereafter be pointed out that when the medulla oblongata is first affected the disease begins in the upward continuation of the central column, and that the accessory nuclei will be liable to become first diseased; and hence it is that the complicated movements of articulation are generally the first to be affected.

On the supposition that the morbid process begins in the central column, it may also be readily explained why muscles innervated from different levels of the cord may be affected, while muscles innervated from the intervening portion are spared, without our being obliged to assume that the morbid

process in the cord has started from two or more centres of origin. The morbid process may, for instance, extend forwards into the median area in the cervical enlargement, while it may pass upwards through the upper cervical region and keep limited to the immediate neighbourhood of the central canal, where it would produce no symptoms, and then on reaching the medulla oblongata extend to the accessory nuclei, and thus produce the symptoms of bulbar paralysis.

§ 416. *Diagnosis.*—The partial form of the disease is liable to be confounded with muscular atrophy caused by direct mechanical injury to the muscle, or with the various diseases of the peripheral nerves. If the disease remain confined to the muscles originally affected, or to the region of a single nerve trunk, progressive muscular atrophy can be excluded; and muscular atrophy resulting from disease of a mixed nerve is usually accompanied by loss of sensation.

The disease may also be confounded with lead palsy; but in the latter the invasion is comparatively sudden, the paralysis being at its height in a week or a fortnight at most; while the electric contractility is diminished or lost at an early period. In the former the paralysis precedes the atrophy; while in the latter the loss of muscular power is almost always directly in proportion to the wasting of the muscular masses, and the electric contractility is maintained so long as any muscle is preserved. The general symptoms which characterise lead poisoning will also assist the diagnosis.

Ordinary general paralysis of central origin may be distinguished from progressive muscular atrophy by the fact that in the former the paralysis occurs as an early symptom, and it is rare that the muscular emaciation bears any proportion to the loss of power. Progressive muscular atrophy attacks the muscles in separate groups, dissecting out either individual muscles or groups of muscles from amongst others which remain healthy, and does not attack at the same time extensive regions or the entire body.

The diagnosis between progressive muscular atrophy and infantile paralysis has been mentioned already.

§ 417. *Prognosis.*—Progressive muscular atrophy is always very intractable, and when the muscles of the trunk are invaded it always progresses slowly towards a fatal termination. In the partial forms, when the disease is limited to one or two extremities, there is no danger to life, but the limbs are, as a rule, permanently damaged. In many cases the advance of the disease may be checked, and, so long as voluntary motion and the electrical reactions are not completely lost, some hope may be entertained that partial restoration of the affected muscles may take place. The most unfavourable cases are those which begin in a multiple form and spread rapidly. The cases in which the disease begins in the thorax or shoulder are unfavourable, because the affection is very liable to implicate the respiratory muscles. When bulbar symptoms supervene the prognosis is specially unfavourable, and when the muscles of respiration are invaded a fatal termination may be expected within a short time. When the disease can be traced back to a hereditary predisposition it manifests a greater tendency to become generalised, and consequently the prognosis is more unfavourable. The prognosis, on the other hand, is more favourable when the affection is caused by overwork and when it is confined to the hands and forearms.

§ 418. *Treatment.*—An attempt must first be made to remove the cause. When the disease, for instance, is caused by a syphilitic taint the usual antisyphilitic treatment must be adopted. When overwork of the affected muscles appears to have been the exciting cause of the disease, they must be allowed to rest. When a decided hereditary predisposition to the affection is manifested in a family, prophylactic measures may be employed, such as a regulated course of gymnastics. The members of such families should also be shielded from deleterious influences, especially those which are known to excite the disease.

The direct treatment of the established disease embraces the employment of hygienic measures, such as baths, methodical exercise, change of air and good diet, and the employment of galvanism and friction to the affected muscles. No medicine has hitherto been found of any use in the treatment of this disease. Tonics, as iron and quinine, may be useful adjuncts in

he treatment, and the nitrate of silver, arsenic, phosphorus, nd iodide of potassium have been employed, but with doubtful uccess.

Thermal and sulphur baths have been recommended, and the vaters of Aix-la-Chapelle have been much praised, but appa- ently on insufficient evidence. The cold water cure, conducted n a good hydropathic establishment, may occasionally be found iseful.

Galvanism is undoubtedly the most efficient remedy for the isease. The local use of the faradic current was applied by)uchenne, who obtained favourable results from it, but the alvanic is probably more efficient than the faradic current. The local use of both currents alternately has given good results. When the muscular excitability is very low, strong currents are equired, and their effects should be intensified by inter- uptions and reversals, but as the excitability returns, weaker urrents should be employed. I have observed favourable esults from the use of electric acupuncture. Suitable gym- astics, to call forth the activity of the affected muscles, passive notion, shampooing, and friction, are all useful in the treat- ient of the disease. When the muscular atrophy is associated vith neuralgia the subcutaneous injection of morphia may be mployed. Dr. Roberts recommends an injection to be given a the morning, and he states that it often enables the patient o pursue his employment with comfort during the day.

4. *Primary Labio-Glosso-Laryngeal Paralysis.*

(*Chronic Progressive Bulbar Paralysis.*—Wachsmuth.)

§ 419. *Definition.*—Labio-glosso-laryngeal paralysis consists f a progressive paralysis and atrophy of the muscles of the ongue, lips, soft palate, pharynx, and larynx.

§ 420. *History.*—A brief report of a case of this affection was sent to ir Charles Bell in 1825 by Dr. F. W. Robinson, and Trousseau wrote an ccurate account of the symptoms of the affection in 1841, but did not ublish his observations. The individuality of the disease was, however, ot distinctly recognised until 1861, when Duchenne described the affection vith his usual exhaustiveness and thoroughness.

§ 421. *Etiology.*—It does not appear that heredity exercises any influence in the production of labio-glosso-laryngeal paralysis. It occurs most frequently between the fortieth and seventieth years of age, and only exceptionally before the fortieth year. The disease attacks men more frequently than women, the proportion being two of the former to one of the latter (Dowse). All ranks of society from the highest to the lowest, and every profession, appear to be liable to the affection.

Of the exciting causes, the most frequently mentioned are exposure to cold, traumatic influences, as a blow on the back of the neck, violent and continuous mental excitement, excessive mental activity, straining of the muscles affected, as in singing and speaking, and bad and insufficient food. Syphilis is not an unfrequent cause of the disease; but the authors who regard this affection as being almost always of syphilitic origin are undoubtedly in error.

§ 422. *Symptoms.*—Slight premonitory symptoms usually precede the full development of the disease, but they are often entirely wanting and are not in any way characteristic of the affection. These consist of pain in the head and back of the neck, slight dizziness, and great diminution or complete loss of the reflex irritability of the larynx, œsophagus, and pharynx. The reflex insensibility of the mucous membrane of the fauces, epiglottis, and pharynx is sometimes so great as to lead to a certain amount of dysphagia, and the passage of food into the larynx for a considerable time before any actual paralysis is observed (Krishaber).

The disease may begin suddenly with difficulty in the movements of the tongue and lips, and of deglutition; but in these cases it is probable that a slight hæmorrhage has occurred in the medulla, and consequently they cannot be regarded as true instances of the primary disease.

The symptoms of the true progressive disease creep on gradually and stealthily. A slight affection of speech is usually the first symptom to attract attention. Utterance is less distinct, the pronunciation of certain letters presents special difficulty, and the tongue and lips are soon fatigued, so that prolonged reading aloud or speaking is impossible.

This is followed by a gradual weakness in the lips and palate. The expression of the face is altered, the voice becomes nasal, and fatigue of the muscles of mastication and deglutition is readily induced, so that the patient is soon compelled to eat only pulpy food, and is unable to swallow much at one meal.

The paralytic symptoms may at times begin in the lips and palate instead of the tongue, and then the order of succession of the symptoms will differ to some extent from that just described.

The initial period of debility and fatigue of the affected muscles may extend over a period of years before the stage of distinct paralysis is reached. When once distinct paralysis is established, the disease assumes a more progressive character, and advances steadily and surely to a fatal termination.

When the affection begins in the tongue, the patient experiences an ever-increasing difficulty in pronouncing the dental and guttural sounds which are respectively produced by approximation of the tongue to the teeth or hard palate, and of the root of the organ to the soft palate. Since the vowel i requires the greatest raising of the tongue for its production, its pronunciation is the first to suffer; and then the pronunciation of the consonants r, s, l, k, g, t, and lastly d and n, becomes difficult, imperfect, and finally impossible.

After a time the patient is unable to effect the coarser and fast complicated lingual movements. He may at first be able to protrude the tongue, but not to raise the tip towards the hard palate or towards the nose after protrusion; while inability to move the tip laterally indicates a still greater degree of paralysis. As the paralysis increases the tongue cannot be lengthened into a point, or made hollow in the centre; and, finally, protrusion is impossible, and the organ lies behind the lower row of teeth completely helpless and motionless, or maintained in constant vibration with fibrillary twitchings.

The tongue may maintain its normal aspect, or become large and flabby; but much more frequently it is sodden, grooved longitudinally, wrinkled, and shrunken, while simultaneous atrophy of the papillæ gives to the surface a glazed appearance.

At an early stage of the affection deglutition is rendered

difficult, simply by the increasing weakness of the tongue. Great difficulty is experienced in collecting the food in the mouth so as to form it into a bolus, and in pressing it back against the soft palate and into the pharynx; and the patient adopts various devices in order to supplement the deficiencies of the first stage of deglutition. He takes care, as Trousseau remarks, to chew well what he eats, and to facilitate its gliding down by drinking and throwing his head backwards, while at other times he assists the imperfect movements of the tongue with his fingers, using them to extract the food which has lodged between the teeth and cheeks, and to push the bolus to the back of the tongue till it is caught by the reflex movements of deglutition.

The muscles which pass from the inferior maxilla to the hyoid bone, and which elevate the larynx as well as the base of the tongue during deglutition, are implicated in the paralysis along with the intrinsic muscles of the tongue; hence it may be observed that the larynx does not rise so readily as in health during the second stage of deglutition. The root of the tongue cannot, therefore, be brought during deglutition over the depressed epiglottis, the glottis is not completely closed, particles of food and fluids easily find their way into the trachæa, and cause distressing paroxysms of cough and dyspnœa.

The saliva cannot be swallowed and accumulates in the mouth, and owing to the advancing paralysis of the orbicularis oris flows from it in an almost continuous stream. Of the muscles innervated by the facial nerve the orbicularis oris is the first to suffer. With the increasing weakness of this muscle the patient becomes unable to whistle, blow, compress his lips, or kiss. The patient experiences difficulty in pronouncing the vowels o and u; and with the advance of the paralysis the labial consonants p, f, b, and m, become increasingly difficult to articulate.

Paralysis of the palate renders the formation of the explosive labial consonants still more difficult; since the current of air necessary to force the lips suddenly asunder escapes through the nose, and the consonants p and b are consequently turned into *me* and *ve*. Duchenne has shown that if the patient's nose be closed these letters are much better pronounced. Paralysis

of the palate also gives a nasal resonance to the voice, and permits food and fluids to escape readily through the nose during efforts at deglutition. When the muscles of the tongue, lips, and palate are simultaneously paralysed, speech becomes more and more indistinct, and the patient can only give utterance to inarticulate and grunting sounds. The vowel *a*, however, can still be pronounced, inasmuch as it is a purely laryngeal sound, and quite independent of the articulatory movements of the tongue and lips.

But although the orbicularis oris suffers more profoundly than the other facial muscles, the quadratus and levator menti are more or less implicated in the paralysis. The muscles of the palpebral and nasal regions are never affected, and even the elevators of the superior lip, as well as the levator menti and buccinators, are only on rare occasions involved in the paralysis. The paralysed muscles are almost always distinctly atrophied, so that the lips look thin, sharp edged, and furrowed, and fibrillary contractions are not unfrequently observed in them. The patient now presents a very striking and characteristic appearance. The lower lip hangs loose and pendulous, the mouth cannot be closed, it is somewhat increased in breadth, and the nasso-labial folds become marked, and give to the patient a lachrymose expression. During states of emotional excitement the lower part of the face remains comparatively motionless, and contrasts strongly with the vivacious movements of the upper half of the face, and with the brightness and activity of the eyes.

The saliva now flows from the mouth in a continuous stream, and causes much annoyance to the patient, inasmuch as it soaks through the pillow at night and requires to be constantly wiped from the lips with a handkerchief during the day. The saliva appears to be secreted as a rule in normal quantity, but in some cases the amount of secretion is very largely increased. Schulz estimated in one case that the secretion was six or eight times the normal amount, and Kayser found that he could increase the flow by reflex irritation, and arrest it temporarily by means of atropine.

Mastication, as already mentioned, is impaired at an early stage of the affection from the difficulty of moving the tongue,

and the condition becomes aggravated when the lips and buccinators are simultaneously paralysed. But the difficulty of mastication is greatly augmented when the motor division of the trigeminus is involved in the disease. The pterygoid muscles are usually the first of the masticatory muscles to be affected, and paralysis of them abolishes the power of effecting the lateral movements of the lower jaw. With the advancing paralysis of the remaining muscles of mastication, the power of chewing the food becomes increasingly difficult, feeble, and finally impossible.

The difficulty of deglutition, caused by paralysis of the tongue, lips, and soft palate, is greatly augmented when the pharyngeal muscles and those which close the larynx are involved in the disease. When the *pharyngeal* muscles are paralysed particles of food are apt to lodge in the pharynx, and this increases the risk of foreign particles entering the larynx. At other times the whole bolus gets fast on a level with the glottis, causing danger of instant suffocation.

But when the muscles which close the glottis are paralysed, the danger of swallowing either solids or fluids becomes greatly intensified. Particles of food passing into the larynx produce distressing paroxysms of coughing and dyspnœa, and by passing into the bronchi often cause pneumonia. When the paralysis extends to the œsophagus deglutition becomes impossible, and to survive the patient must be fed by the stomach pump. When the nucleus of the spinal accessory nerve is involved in the disease the laryngoscope reveals paresis or paralysis of the vocal cords, the voice becomes hoarse and feeble, until finally there is complete aphonia. The power, not of articulation only, but of phonation also, is now abolished, the loss of this function being manifested by inability to pronounce the vowel *a*. The loss of phonation does not necessarily interfere with the respiratory functions, but as the disease advances disorders of respiration and circulation supervene, which soon prove fatal.

Not much is known with respect to disorders of the circulation in the early stages of the affection. There is no trustworthy record of retardation of the pulse which could with probability be referred to irritation of the vagus, but a pulse rising before death from 130 to 150, or even higher per minute, has been

frequently recorded, and is probably caused by paralysis of the vagus. In the terminal period of the disease patients often suffer from fainting fits, accompanied by great anxiety and a sensation of impending death, and, indeed, death may result from an attack of syncope. These phenomena are probably caused by the cardiac centres of innervation having become involved in the disease.

When the *respiratory mechanism* is affected a fatal termination is near. The respiratory movements become feeble, and, owing to the implication of the spinal accessory nerves, the auxiliary muscles of respiration are paralysed, and superior thoracic breathing is impossible. The inefficiency of the respiratory movements renders the breathing shallow, and all attempts at coughing or blowing the nose are weak and powerless. Patients complain of a feeling of constriction, accompanied by an oppressive feeling of want of breath. After a time the pneumogastric nerve appears to become implicated in the disease. Paroxysms of dyspnœa, with a tendency to syncope, supervene, but these must not be confounded with the suffocative attacks which occur at an early period from the accidental introduction of foreign bodies into the larynx. The attacks of dyspnœa become more and more frequent as the disease progresses, and the breathing power feebler and feebler, until ultimately the patient dies from asphyxia. Death may, indeed, be caused at an early period of the affection by a slight disease of the respiratory organs, such as a bronchial catarrh or pneumonia.

Atrophy of the paralysed muscles is one of the most constant and striking symptoms of this affection. It is usually most marked in the tongue, and the lips also become emaciated and thin, and both are often kept in constant movement by fibrillary contractions. Atrophy of the soft palate has not yet been recorded, and cannot probably be recognised with certainty.

Atrophy of the paralysed muscles is not an early symptom of the disease, and does not run a parallel course with the paralysis. The tongue may, however, retain a normal appearance and volume, and yet exhibit, on microscopical examination, extensive degeneration of its muscular fibres (Charcot).

Local atrophy and fibrillary contractions of the small muscles

of the hand are sometimes observed indicating a complication with progressive muscular atrophy.

The electric excitability is generally said not to undergo any noteworthy changes, but Erb states that he found the most marked "reaction of degeneration" on direct irritation of the muscles of the chin, lips, and tongue. The electric irritability of the nerves was, however, normal, or but slightly diminished.

The sensibility remains, as a rule, unaffected throughout the whole course of the disease, and even taste is only altered on rare occasions. Affections of the auditory nerve, consisting of buzzing of the ears and deafness, have occasionally been observed. The trigeminus is sometimes implicated, the symptoms observed being a furry feeling and anæsthesia on both sides of the face, and want of common sensation in the tongue, and in some cases pain has been felt in the occipital and upper part of the cervical region (Trousseau).

The intelligence remains quite clear to the last, the temper is somewhat excitable, and patients often manifest an inclination to laugh on slight provocation.

Reflex irritability is, as already mentioned, sometimes greatly diminished or abolished in the tongue, soft palate, pharynx, and even in the larynx before the appearance of any other symptoms, but on the other hand it is often retained in these parts until a late period of the disease. Even when the reflex irritability is lost the patient can feel and localise each touch quite distinctly.

Vaso-motor disturbances have not been recorded, and there is no fever during the whole course of the disease.

General nutritive disorders occur sooner or later in the course of the affection. These are in large part due to the insufficient quantity of food taken, and which ultimately produces a state of inanition. The helplessness of the patient is greatly aggravated by his inability to close the glottis, and thus all forcible expiratory actions are rendered impossible. Duchenne thought, probably on insufficient grounds, that the constant loss of the saliva exercised a deleterious influence on digestion, and consequently contributed to produce the general debility. But whatever may be the causes which co-operate to produce the state of emaciation and marasmus into which the

patient falls, the debility at last becomes so great that he is unable to get up. He sits in bed, with the upper part of the body propped up, and with the head resting on pillows and inclined to one side, in order to let the saliva flow out of the mouth, and death soon supervenes, either during a paroxysm of dyspnœa or suddenly and quietly from arrest of the heart's action.

§ 423. *Course, Duration, and Terminations.*—The course of bulbar paralysis is always slow and chronic, but surely progressive. There is seldom a remission of long duration; any degree of improvement is still rarer, and recovery has never been observed when the diagnosis of a primary affection was beyond question. Death usually results in from one to five years.

§ 424. *Complications.—Progressive muscular atrophy* is the most important and frequent complication of bulbar paralysis. Labio-glosso-laryngeal paralysis may either be the primary affection, or it may merely be a terminal phenomenon supervening in the course of progressive muscular atrophy, and caused by extension of the morbid process in the anterior grey horns of the cord to the motor nuclei of the medulla. The two affections are indeed essentially the same disease, both as regards the clinical symptoms and the anatomical changes found after death.

Amyotrophic lateral sclerosis is another important complication of progressive bulbar paralysis, and the latter may either be the primary or secondary affection. Amyotrophic lateral sclerosis occurs not unfrequently in the later stages of bulbar paralysis. The disease is then characterised by the symptoms of progressive muscular atrophy in the superior and those of spastic spinal paralysis in the inferior extremities. A number of cases described as bulbar paralysis or progressive muscular atrophy belong to this class. The disease is no doubt due to extension of the morbid process in the medulla to the anterior grey horns of the cervical enlargement and to the antero-lateral columns of the cord.

§ 425. *Morbid Anatomy.*—The first observations with respect to the morbid alterations of the nervous system were conducted

without careful microscopical examination of the medulla and pons. In a complex case of paralysis of the tongue, lips, and veil of the palate, combined with general muscular atrophy, recorded by Dr. Duménil, the roots of the hypoglossal, facial, and spinal accessory nerves, as well as the anterior spinal roots, were found wasted, the atrophy also extending to the trunks of the nerves themselves. Trousseau found increased consistency of the medulla oblongata and thickening of the dura mater of the medulla; but he regarded the atrophy of the roots of the bulbar nerves as the essential morbid alteration in this disease.

The close anatomical connection which the researches of Lockhart Clarke and others have proved to exist between the nuclei of origin of the nerves implicated in this disease, as well as the discovery which had already been made that the allied affections of progressive muscular atrophy and infantile spinal paralysis were due to disease of the ganglion cells of the anterior grey horns of the cord, had led pathologists to suspect that the essential anatomical changes in this disease would be found, not in the roots of the nerves, but in the ganglion cells of their nuclei of origin. It was in reference to this expectation that Dr. Wilks wrote: "The anatomist and the physiologist have in fact informed the clinical physician of the precise spot which is affected, and it only remains for the pathologist to prove it."

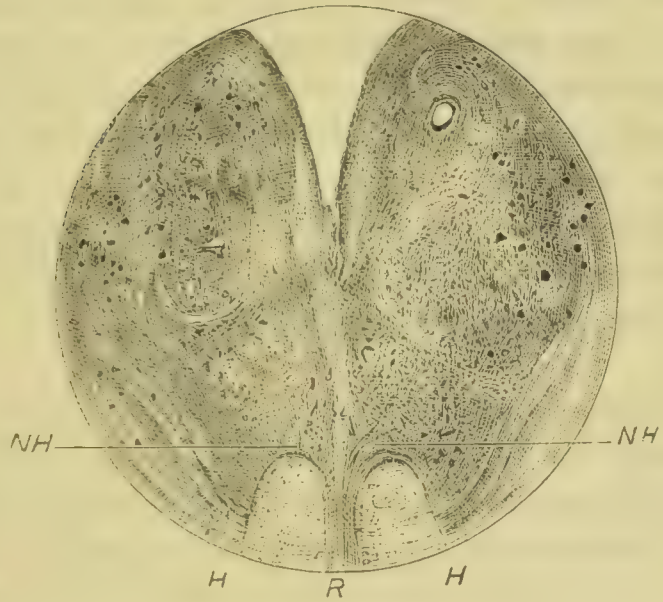
About the time that this sentence was being uttered by Dr. Wilks the opportunity for making the necessary verification of the hypothesis presented itself to the ever-vigilant eye of Charcot, and his observation was soon afterwards confirmed by Duchenne and Joffroy. The essential anatomical changes in the affection appear to consist of a degenerative atrophy of the ganglion cells in the grey nuclei on the floor of the fourth ventricle. The cells shrink and become filled with yellow or brown pigment, their nuclei disappear, and finally the cells themselves are only represented by angular, glistening pigmented masses.

In some cases the surrounding tissue was found to contain corpuscles of Gluge in varying quantity, increase of connective tissue and in the number of nuclei and of Deiter's cells, and

ıypertrophy and fatty degeneration of the vascular walls. The ıerve fibres themselves were found atrophied, the medullary heath had disappeared, and in chronic cases the axis cylinders ılso.

The nucleus of the hypoglossal nerve appears to be the tarting point of the disease, and the nuclei of the spinal ıccessory and vagus are next attacked, while the disease does ıot extend in all cases to the nucleus of the glosso-pharyngeal

FIG. 171.



'IG. 171 (From Leyden). *Portion of the Grey Substance on the floor of the fourth ventricle on a level with the middle of the Hypoglossal Nucleus, from a case of Progressive Muscular Atrophy with Bulbar Paralysis, showing the destruction of the Ganglion Cells of the nuclei of the Hypoglossal and Pneumogastric Nerves (NH). R*, Median raphé; *H, H*, Fibres of the hypoglossal nerves. The accessory nuclei have evidently disappeared.

ierve. The nuclei of the facial are attacked at a very early tage, especially those which are connected with the inferior ranches of the nerve, and which I have named the accessory uclei of the facial. The annexed diagram (*Fig.* 171), borrowed rom Leyden, represents the morbid changes observed in the nedulla oblongata in bulbar paralysis. Remnants of the fun- amental cells of the hypoglossal nuclei may still be observed, vhile every trace of the accessory nuclei has disappeared.

The motor nucleus of the trigeminus has been found affected, ıut the nucleus of the abducens and the acoustic and trigeminal

sensory nuclei appear never to suffer. Other changes have been described, but they appear to be quite secondary to the alterations in the ganglion cells. The pyramidal tracts have often been found diseased, and the degeneration could generally be traced into the pons and downwards into the antero-lateral columns of the cord; but such cases are not pure examples of the disease. The bulbar affection is often associated with diseases of the spinal cord, such as progressive muscular atrophy and amyotrophic lateral sclerosis. The roots of the bulbar nerves are almost always atrophied, and degenerative changes have been found in the nerves themselves. The muscles atrophy and undergo the same degenerative changes which are observed in progressive muscular atrophy. When the disease extends into the spinal cord, the ganglion cells of the anterior cornua are diseased in the same manner as in progressive muscular atrophy. When the cord is affected the spinal nerves issuing from the diseased portions and the muscles to which they are distributed are involved in morbid action.

§ 426. *Diagnosis.*—When bulbar paralysis is fully developed, the symptoms are so characteristic that it is hardly possible to mistake them, but the onset of the disease is often very insidious. The patient may complain of a feeling of pressure and traction in the back of the neck, and a slight nasal resonance of the voice, these being frequently the first symptoms to attract attention. On closer examination it may be observed that there is a slight alteration in the expression of the face, and a certain degree of stiffness of the lips, causing some imperfection of articulation, and that the tongue trembles on protrusion.

In the fully established disease the difficulty is to distinguish between the primary and secondary forms of the affection. The diagnosis must be made from a careful investigation into the history of the case, and the order of development of all the symptoms. The manner in which the disease begins is the best guide to distinguish it from embolism, thrombosis, and hæmorrhage in the medulla. These lesions always commence suddenly, and the resulting paralysis is frequently unilateral or more pronounced on one side than on the other, while pro-

ressive bulbar paralysis is always gradual in its onset, and the aralysis is uniformly bilateral.

§ 427. *Treatment.*—The treatment of true progressive bulbar aralysis has hitherto proved of little avail, but much may be lone to add to the comfort of the patient and probably to delay he progress of the disease. Nothing should be left undone vhich tends to improve the general health of the patient.

The special remedies which have been employed in the reatment of the disease are nitrate of silver, iodide of potas- ium, iodide of iron, chloride of gold and sodium, ergotine, and elladonna; but unless there is any special indication for the dministration of iodide of potassium I should, myself, follow he advice of Dr. Dowse, and trust to phosphorous, iron, and od-liver oil.

The galvanic current should be used. Erb advises the tabile application of the current transversely through the nastoid processes, and longitudinally through the skull; also alvanism of the cervical sympathetic, by placing the anode on he nucha while the cathode is run rapidly over the lateral urface of the larynx, twelve to twenty movements of degluti- ion being induced at each sitting. The galvanic or faradic urrent may be applied to the tongue, lips, and palate.

As the power of deglutition becomes more and more impaired, he food must be carefully selected and finely divided, and inally the patient must be fed through a tube. Care should be aken to wash the mouth after eating, and to remove particles f food which lodge about the pharynx and underneath the ongue.

5. *Pseudo-Hypertrophic Paralysis.*

(*Atrophia Musculorum Lipomatosa.*)

§ 428. *Definition.*—This disease is characterised by feebleness f the muscles of the lower limbs and of the erector muscles of he spine, gradually extending to those of the upper extremities. The paralysis is accompanied by atrophy of some of the muscles, and by an apparent increase in the volume of others.

§ 429. *History.*—Isolated cases of this disease were described many years ago by several authors. A case was reported by Sir Charles Bell in 1830,

one by Coste and Gioja in 1838, and a well marked case by Mr. Partridge in 1847. But Dr. Meryon, in 1852, was the first to draw attention to the clinical features of this affection, and Oppenheim, in 1855, described a well marked group of cases. The disease was thoroughly investigated by Duchenne. His friend, M. Bouvier, sent to his clinique in 1858 a child, who had been suffering from an unusual form of paralysis, and during the subsequent three years Duchenne collected other cases, which were similar to the one sent by Bouvier, and yet did not correspond to the description of any known disease. Duchenne described the principal clinical characters of the disease in the second edition of his work on Localised Electricity (Paris, 1861), but it was not until 1868 that he published in the "Archives Générales de Médecine" a full account of his investigations into the nature of the affection. He then gave detailed descriptions of thirteen cases, which had come under his own observation, and incorporated with these fifteen cases published up to that date by other observers. So thoroughly was the work done by this distinguished physician that nothing essential has since been added to our knowledge of the course and progress of the disease. Cases have been described in this country by Mr. William Adams, Dr. Langdon Down, Dr. Ord, Mr. Kesteven, Drs. Russell and Balthazar Foster (Birmingham), Dr. Barlow (Manchester), Dr. Davidson (Liverpool), Dr. Clifford Allbutt, while Dr. Gowers has written an able monograph on the disease.

§ 430. *Etiology.*—This affection is almost but not exclusively confined to infancy. The first symptoms are frequently noticed at the time when the child ought to begin to walk, although it is very probable that the disease is established before that time. The disease begins in a considerable proportion of cases between the ages of five and thirteen years, and cases are recorded where adults have been attacked. It may, however, be doubted whether many of the latter are genuine examples of the disease. In the case described by Auerbach, for instance, the hypertrophy was first noticed in the right arm, and microscopical examination of the affected muscles showed that the disease was more like true hypertrophy. In the case described by Eulenburg, where the patient, a female, was forty-four years of age when the first symptoms appeared, the paralysis began in the right arm, in the form of progressive muscular atrophy, and although there was apparent hypertrophy of the muscles of the lower extremities, the course and progress of the case was different from those of a case of true pseudo-hypertrophic paralysis. In an undoubted instance of the disease under my

re at present the patient is forty-seven years of age, but the fection began at the age of ten. It is much more common boys than girls. Of the thirteen cases collected by Duchenne ıly two were girls, of forty-one cases collected by Webber only ?e were females, and of twenty-three cases mentioned by Dr. ɔwers only five were females. Out of a total of 220 cases therto published 190 were males and thirty females (Gowers). ıe disease also appears to pursue a more chronic course in rls than in boys.

Hereditary predisposition to the disease frequently exists. wo children in the same family are often attacked, as in the ses related by Wernich, Heller, and Kesteven, and sometimes ·en eight children of the same family have been affected, as in e cases related by Meryon. Lutz met with two sisters, a aternal uncle and aunt, and a maternal half-sister, issue of a st marriage, affected with the disease. In another example, ree maternal uncles and aunts were affected; in a second, a aternal uncle and a half-uncle; in a third, three maternal lf-brothers; and in a fourth instance, a maternal half-brother, ree maternal uncles, and other members on the mother's side oore). It is curious to notice that, although the disease is ainly confined to the male sex, yet the descent, so far as is ıown, is always through the mother's side. This disease is t, as a rule, transmitted directly from parents to offspring; ıce by far the greater number of its victims are attacked at early age, and therefore do not become parents, and this nsideration also precludes the idea that it is an example of avism. A certain predisposition is, therefore, transmitted, ıich, with the concurrence of other unfavourable circumstances, ch as an eruptive fever, develops the disease.

The exciting causes of the disease are by no means clear. posure to cold and damp appears to be occasionally the termining cause, while at other times it has followed an ıptive fever, variola, or measles, and several cases have been hered in by convulsions.

§ 431. *Symptoms.*—Feebleness of the lower extremities is ually the first symptom to attract attention, and when the sease begins during infancy it is difficult to fix the exact date

of its origin. The attention of the parents is not directed to the condition of the child until he arrive at the age when other children begin to walk. At this period it is noticed that when the child is placed on his feet he does not instinctively move his legs to walk, but immediately falls down, and in other cases he may have begun to walk, but is soon fatigued and can no longer stand steadily or walk without stumbling. At other times the child may be late in attempting to walk, and is obliged to support himself by holding on to the nearest article of furniture. The parents are not readily alarmed at the inability of the child to walk, inasmuch as the limbs appear to be so well developed. When standing or walking the feet are widely separated from one another, and when they are made to approach each other walking is rendered difficult, and the child may fall. In walking, the body is inclined from side to side, so that the gait resembles the waddling of a duck. When the feet are kept widely apart the centre of gravity at each step must be carried well over to the side of the active leg, in order that the line of gravity may pass through the centre of the arch of the foot planted on the ground. Duchenne thought that the oscillation of the body in walking depended upon weakness of the gluteus medius. But in the case of Charlotte A——, already described (§ 412), the gluteus medius on both sides was paralysed, yet instead of the waddling gait so characteristic of pseudo-hypertrophic paralysis, the head and body were moved forwards during locomotion in a straight line without the normal lateral inclination of them being observed.

In several cases of pseudo-hypertrophic paralysis which I have examined with reference to this point, on placing one of my hands on each side of the pelvis immediately above the trochanters, the gluteus medius on the side of the active leg could be distinctly felt to contract at each successive step. The patients also, when lying on one side, with legs extended, are able to raise the upper leg away from the other, without much apparent difficulty, and when the hand is placed over the gluteus medius during this movement the muscle may be felt to contract powerfully. In an advanced case of the disease which I saw recently along with my friend Dr. John

PLATE III.

1.

2.

3.

4.

own, of Burnley, the patient could not stand or sit erect, yet
ıen lying on his side he could abduct the upper leg, and on
acing my hand above the great trochanter of the femur the
uteus medius was felt to contract. This muscle was, therefore,
ıt likely to have been affected at an early period of the affec-
ɔn. The oscillation of the body in walking, therefore, instead
being caused by paralysis of the gluteus medius, is, in my
ɔinion, mainly effected by contraction of this muscle. The
teral inclination of the body appears, indeed, to be rendered
ɔcessary in the early stage of the affection partly by the legs
ɔing held widely apart, and partly by the inability of the
ıtient to produce dorsal flexion of the foot so as to allow the
ıssive leg to swing forward in locomotion.

In the second stage of the disease, when double talipes
quinus and dorsal curvature are established, other factors co-
ɔerate in the production of the alternate balancings of the
ɔdy. When talipes equinus is once formed, the body at each
ccessive step must be delicately balanced so that the line of
avity will pass through the ball of the foot, and consequently
e slightest displacement of the centre of gravity would cause
e patient to fall. It is therefore necessary that at each step
e body should be inclined well over to the side of the active
g, and the patient aids himself in balancing the body on the
ll of the foot on the side of the active leg by moving his
ms about like a rope dancer.

When the patient is laid down or falls, he raises himself in a
aracteristic manner. If any object be near which he can
nveniently grasp, such as a chair or other article of furniture,
drags himself up by his arms. When the patient has to get
ɔ without extraneous aid, he first raises himself on his hands
d feet. In the first position which he assumes the patient's
ɛt are planted on the ground, the different segments of the
wer extremities are slightly flexed upon one another, the
dy is flexed on the lower extremities, and the head directed
ɔwnwards, and the tips of the fingers of both hands rest on the
ound a little in front of the toes (Plate III., 1). The patient
ɛxt raises his hand, say the left, and places it above
e left knee. The body is now drawn over to the opposite
le so that its weight rests mainly on the right leg, by one

vigorous push of the left arm the left knee-joint is thrust backwards, and the leg and thigh are thus extended one upon another, while the body is at the same time thrust upwards. The feeble extensors of the body on the thigh are now brought into action, and the trunk is partly raised upwards by their contraction, and partly pushed upwards by the left upper extremity, while the right may not require to be placed over the right knee in the early stage of the affection.

But even in the early stage of the disease the action of the extensors of the body on the thighs is greatly aided by the abductors of the thighs, and the patient may be observed to elevate the trunk by a kind of rotatory movement, the body being drawn first to the one side and then to the other.

The following case has afforded me an opportunity of studying the different movements which are made in the act of attaining the erect posture, inasmuch as these are slowly performed; and as the case is of interest in other respects, I shall describe the symptoms in detail. I have received valuable assistance from Dr. A. H. Young in describing the different groups of muscles which are brought into action by the patient in attaining the erect posture.

Peter P——, forty-five years of age, was admitted to the Royal Infirmary, Manchester, February 12th, 1880. He was quite healthy until ten years of age, when he had an attack of typhoid fever. During the attack of fever he suffered from bed-sores, and his recovery was slow and protracted. Subsequent to this period he could ascend a stair without difficulty, and could carry weights like other people. He thinks, however, that his mode of walking was peculiar, and that he was weak on his legs. He could not join in games which required active exercise, as running, and the other boys at school amused themselves by pushing against him and throwing him down. At fifteen years of age he was apprenticed to a joiner, and was then able to ascend a ladder and perform the ordinary work. It was not, indeed, until he was thirty years of age that his present symptoms began to attract attention. At this time his master observed that he was unable to get through his work like the other men, and consequently he was the first to be discharged when work was scarce. The first symptom which attracted his attention was that he was unable to ascend a stair without placing his hand on his knee, while holding on to the banister with the other hand. From that time up to the present, a period of thirteen years, he has become gradually and slowly worse. He was married thirteen years ago, and has three children, all of whom are healthy.

Present Condition.— The patient seems fairly well nourished and healthy

here is a cicatrix two inches in diameter on the prominent part of the crum, and a smaller one over the great trochanter of the femur, on each le, these being left by the bed-sores from which he suffered when ill of phoid fever.

As the patient stands on the floor in the erect posture his feet are inches apart at the heels and 10 inches at the toes. The heels scarcely uch the ground; when he stands on his naked feet a piece of cardboard n be readily passed between the heels and the floor. When he raises his ot off the ground it assumes the position of talipes equino-varus, and he nnot produce dorsal flexion of the foot, but there is no deformity of the es. When the patient is standing the muscles of the calf are hard, tense, mparatively large, and well formed. The muscles of the thigh are small, ft, and flabby, so that the comparatively slender thighs offer a striking ntrast to the large and well formed calves. The buttocks are somewhat ttened, and fibrillary movements are observed in the erector spinæ and e muscles of the back of the thigh. The scapular muscles are unaffected, e deltoids are prominent, and act with great energy on voluntary effort. ne pectoral muscles are decidedly atrophied, and the triceps, biceps, and e coraco-brachialis are wasted to so marked a degree that the slenderness the arm offers a strong contrast to the full and rounded shoulder caused the prominence of the deltoid. The muscles of the forearm are not rophied, and they stand out prominently under the skin, and feel hard d tense when the patient grasps anything strongly, yet his grasp is markably feeble. Dr. Leech, who has made a microscopic examination portions of these muscles withdrawn by his trocar, assures me that ey exhibit morbid changes, but he is unable to say that they are the same those which characterise pseudo-hypertrophy of muscle.

The following measurements were taken: Height 5ft. 7in., circumference the chest 32in., abdomen 31½in., upper part of each thigh 16¾in., middle each thigh 13in., each calf 13½in., upper arm 7¼in., forearm 9¼in. The cumference of the calf exceeds that of the middle of the thigh, while e circumference of the forearm greatly exceeds that of the upper arm. hile standing the pelvis is inclined well forwards, his abdomen is some- at protuberant; while the upper part of his body is dragged backwards, that a deep curve, with its concavity directed backwards, is formed in e lumbo-dorsal region A plumb-line, let fall from the most prominent the spinous processes of the upper dorsal vertebræ, falls three inches hind the sacrum.

Walking.—The gait of the patient is peculiar and characteristic; the dy is alternately drawn from side to side, giving to the walk a duck-like waddling movement. The patient, as already remarked, can neither ace the two heels firmly on the ground at the same time, nor elevate the es by producing dorsal flexion of the foot, and consequently the passive ; cannot swing forwards with the normal pendulum movement.

The difficulty of moving the passive leg forwards is, indeed, increased the fact that the predominant action of the muscles of the calf extends

the foot on the leg when once it is raised off the ground, so that the limb is lengthened instead of being shortened by dorsal flexion of the foot, as in normal locomotion. Under these circumstances the toes of the passive leg are made to clear the ground by a different mechanism from that which obtains in health. The feet are, as already described, held widely apart; and when the passive leg, say the right, is to be moved forwards the body is dragged well over to the left. This movement is mainly effected by the abductors of the thigh on the side of the active leg, and the gluteus medius on that side is felt strongly contracted on placing the hand over it. But the centre of gravity is not only drawn over to the side of the active leg, but it is also drawn somewhat backwards by the action of the gluteal and probably also the hamstring muscles, and the line of gravity in passing through the arch of the left foot approaches the heel, and the latter is now felt to be firmly planted on the ground. During this double but combined movement the line of gravity is in danger of being carried too far to the left and backwards, hence the right arm is thrown outwards and forwards so as to maintain the centre of gravity as far to the right and forwards as possible. During the lateral movement of the body towards the side of the active leg the pelvis on the side of the passive leg is elevated, and thus the length between the head of the femur and ground is increased, and during the backward movement of the body the pelvis is made to assume a more vertical position, so that the flexors of the thigh on the body can act more efficiently on the passive leg. The thigh of the passive leg is now flexed on the body, the abductors also contracting and giving to the thigh an outward inclination, the leg is slightly flexed on the thigh, and the foot is moved slowly forwards and outwards, and when the step is completed the toe comes first to the ground.

The forward and outward projection of the passive leg tends to counteract the tendency of the line of gravity to pass too far to the side of the active leg and backwards. When the passive leg is placed on the ground the abductors of the thigh on that side contract, the body is drawn over to the right, and the line of gravity is slowly transferred to the leg that was passive and which now in its turn becomes active.

Attaining the Erect Posture.—On rising from the recumbent position the patient first gets on his hands and knees, and placing his right foot on the ground, he rests his right elbow above the knee, and inclines his trunk to the right so that the centre of gravity passes through the right foot. When he leans well forwards in this position and presses his right elbow downwards and backwards, it will tend to drag the trunk and with it the right hip-joint forwards, but inasmuch as the right knee is at the same time pressed downwards and backwards, any forward movement of the hip-joint must be accompanied by elevation. The weight of the trunk is, therefore, so applied that it tends to drag the hip-joint forwards and upwards, and thus to extend the trunk on the thighs and to push the right knee-joint downwards and backwards, and thus to extend the leg upon

the thigh, so that the weight of the trunk is so applied as to aid the extensors in erecting the body.

The extensor muscles are now brought into action, and the trunk is slowly elevated to what I may call the second position. In this position the various segments of the right lower extremity are slightly flexed upon one another, the trunk is directed forwards horizontally, and the right elbow rests above the knee, while the left thigh is directed vertically downwards, the left leg is inclined downwards and backwards, and the toe rests on the ground considerably behind the right foot, while the left hand rests lightly on the left thigh immediately above the knee.

After a momentary pause the patient proceeds to attain the third position. The abductors of the right thigh contract and rotate the pelvis so that the left hip-joint is slightly elevated. This movement brings the line of gravity well within the right foot, and takes the weight of the trunk entirely off the left lower extremity. The left foot is now drawn forwards and placed on the ground in a line with the right foot, but slightly removed from it laterally, while the left hand at the same time grasps the left thigh immediately above the knee. By a contraction of the abductors and extensors of the left thigh the line of gravity is now transferred from the right to the left foot, the right shoulder is elevated, and the right hand is quickly transferred to the position previously occupied by the elbow; the abductors and extensors of the left thigh now relax, until the line of gravity passes between the feet, and the third position is attained.

In this position the two sides are symmetrically placed. The feet are placed on the ground and somewhat removed from one another, but the heels do not quite touch the ground; the legs are slightly flexed on the feet, the thighs on the legs, and the trunk on the thighs; both arms pass downwards and backwards, each hand grasping the thigh of the corresponding side close above the knee. When the patient is viewed laterally, the thigh, arm, and trunk are seen to form the three sides of a triangle (Plate III., 3), and the weight of the trunk applied through the arms must tend to push both knees downwards and backwards, while at the same time tending to elevate the hip-joints. The body is inclined forwards and upwards, but owing to the deep dorso-lumbar curve the vertical axis of the pelvis occupies a more horizontal position than might be expected from the upward inclination of the body. The line which joins the anterior superior spine of the ilium and the head of the femur forms nearly a right angle with that which joins the head of the femur and the centre of the arch of the foot; and, consequently, were the gluteus medius and minimus of both sides now to contract, they would act mainly as flexors of the pelvis on the thighs.

The patient, after a little pause to take breath, prepares for a further elevation of the body, the great difficulty he has to encounter is to erect the pelvis on the thighs, while at the same time extending the various segments of the lower extremities upon one another. By transferring the line of gravity from one foot to the other he takes the weight of the body

off each foot alternately, and in this way he is enabled to slip by turns each hand further up the thighs until he grasps them about the junction of the middle with the lower third. The trunk is now dragged over to the left, so that the line of gravity passes through the left foot, and the right hand is removed from the right thigh (Plate III., 4). The right foot is shuffled outwards and backwards, so as to allow the leg to be fully extended on the thigh. This movement is performed with great deliberation, and after it is effected the patient rests for a moment as if to assure himself that the right foot, which now rests on its inner edge considerably behind and removed from the left foot, is firmly planted so as not to slip. The final effort now begins. Apparently by a combined action of the inward rotators of the left and of the outward rotators of the right thigh the pelvis is rotated obliquely from before backwards and from right to left. By this movement the right hip-joint is brought well forwards, and the pelvis is probably also, by a simultaneous action of the extensors of the body on the thigh, made to assume a more vertical position. But whatever may be the nature of the muscular action concerned in this movement, when it is completed the head of the right femur is placed almost vertically below the anterior superior spine of the ilium, instead of being on the same horizontal plane with it as in the third position. The line which joins the anterior superior spine of the ilium and the great trochanter now forms a very obtuse angle with that joining the great trochanter and the middle of the arch of the foot, and in this position the gluteus minimus and medius will act mainly as extensors of the pelvis on the thighs. The great effort of the patient is now directed to transfer the line of gravity from the left to the right foot. This is effected by the trunk being dragged over in a diagonal manner from before backwards and from left to right, partly by the conjoined action of the extensors and abductors of the right thigh, and partly by the left shoulder being pushed upwards and to the opposite side by forces acting upon it from below through the arm. The elevation of the left shoulder is effected by the extension of the different segments of the arm upon one another, and by the elevation of the heel and consequently of the knee by contraction of the muscles of the calf. The upward movement of the left shoulder is not one of simple elevation, but is indeed a very complex act. The left knee is not only elevated by contraction of the muscles of the calf, but a strong contraction of the adductors of the thigh prevents it from being thrust out laterally. The inward rotators of the left arm (the latissimus dorsi, teres major and minor, and infraspinatus), and the abductors of the arm, especially the posterior third of the deltoid, enter into strong contraction. The tendency of the combined action of these muscles is, the arm being fixed by the hand grasping the knee, to thrust the left shoulder to the opposite side, and to rotate the body, so that the left shoulder is pushed forwards in advance of the right one. We have already seen that the pelvis was rotated in such a way that the right was placed in advance of the left hip-joint, and now the left is pushed forwards in advance of

the right shoulder, and consequently the upper part of the body is being rotated in the opposite direction to the lower part; or, in other words, the pelvis is being rotated from right to left through the hip-joints, and from left to right through the vertebral column, the power in the latter case being applied on a level with the brim of the pelvis. If the forces which tend to rotate the pelvis from right to left, and those which tend to rotate it from left to right were applied on the same level, they would tend to neutralise one another, and the pelvis would remain more or less fixed. But, inasmuch as the forces which rotate the pelvis from right to left are applied through the hip-joints, and those which tend to rotate it from left to right through the vertebral column, the consequence is that the former will tend to push the head of the right femur forwards, while the latter will tend to carry the brim of the right ilium backwards. It will be thus seen that the forward rotation of the left shoulder will tend to carry the anterior superior spine of the right ilium backwards, and therefore assists the action of the gluteus medius and minimus of the right side as extensors of the body on the thigh. It may, indeed, be said that the double rotation just described twists or screws the pelvis into a more or less erect position with reference to the right lower extremity, around the hip-joint of which all the movements of the body at present centre.

As the line of gravity approaches the right foot, the left lower extremity is becoming more and more inclined forwards and outwards, its different segments become extended upon one another, and the toe rests on the ground. When once the line of gravity passes through the right foot, the extensors and abductors of the right thigh relax somewhat, while those of the left now suddenly contract; the pelvis is rotated once more in such a way that the head of the left femur is brought forwards under the pelvis. During this movement the left hand is removed from the thigh, the muscles of the calf relax, the heel comes to the ground, and the line of gravity is for a moment transferred to the left foot, but immediately afterwards the weight of the body is borne by both feet, the line of gravity falling between them, and the erect posture is attained.

When the patient reclines on one side he can raise the uppermost leg away from the other with a considerable degree of force, and during this action the gluteus medius can be felt strongly contracted.

When sitting he can cross one leg over the other readily, abduct and adduct his legs with considerable force against a resisting object, but he can only produce dorsal flexion of the foot to a slight extent.

Owing to the feebleness of the gluteus maximus, the patient experiences great difficulty in getting up steps, and the manner in which he ascends a stair is as characteristic as that in which he attains the erect posture. He lays hold of the railing with one hand, say the right, and by the contraction of the muscles of the right upper extremity he drags his body upwards at each

step. The right arm is, however, assisted by the left. The left hand is planted above the left knee, and each time the left leg is raised a step the body is thrust upwards by the various segments of the left arm being extended upon one another.

One of the most constant symptoms of the disease is the existence, during standing or walking, of a remarkable curvature of the spine in the lumbo-sacral region. The shoulders and upper part of the vertebral column are carried backwards, so that a plumb-line let fall from the most prominent spinous process of the vertebræ falls behind the sacrum. I have, however, observed an undoubted example of the disease in which the plumb-line did not clear the sacrum. Duchenne attributes this incurvation to weakness of the erector muscles of the spine; but, as pointed out by Dr. Gowers, weakness of the extensors of the pelvis on the thighs contributes to the formation of the lordosis. Weakness of the extensors allows the pelvis, and with it the lowest lumbar vertebræ, to incline forwards in the erect posture, and a compensatory backward inclination of the dorsal spine is necessary in order to keep the centre of gravity in the normal position.

Another important feature of the disease is that the patient has a difficulty in bringing his heels to the ground; and, as the case advances, a permanent condition of talipes equinus, or equino-varus, is established. The foot becomes more hollow from increase of the plantar arch, while paralysis of the interossei causes the first phalanges to be maintained in a state of exaggerated extension on the metatarsal bones, and the two distal phalanges to be flexed, so that the toes assume the peculiar clawlike appearance, which Duchenne has called *griffe des orteils.*

The apparent hypertrophy of the muscles, which is the most characteristic symptom of the disease, generally begins by enlargement of one calf, the other also becoming affected before very long. This is the usual mode of invasion, but sometimes the muscular enlargement begins in the muscles of the upper extremities, as in a case related by Duchenne, where the deltoids had begun to enlarge many months before the gastrocnemii. The gluteal muscles become affected soon after those of the calf, and then the disease extends in succession to the

lumbo-spinal muscles and to some of the muscles of the thigh, trunk, and upper extremities. Of the muscles of the upper extremities the deltoids are usually the first to suffer. In one case related by Duchenne the apparent hypertrophy had become so general that, with the exception of the pectoral muscles, the latissimus dorsi, and the sterno-mastoids, all the muscles of the limbs, trunk, and even those of the face, especially the temporals, were successively invaded. In a case related by Weir Mitchell not only the muscles of the face, but even those of the tongue, were hypertrophied.

The affected muscles may attain an enormous volume, and stand out so prominently under the skin that Duchenne uses the term "hernial protrusions" to describe their appearance. The muscles also feel hard and resisting to the touch, so that the whole appearance of the child often suggests the idea of Herculean strength instead of the great feebleness which in reality exists. But even amidst all this apparent development of muscular power there are not wanting visible indications of the real nature of the malady. Some of the muscles are always found atrophied, their wasted condition contrasting strongly with the excessive size of the others. Even in the case related by Duchenne, where the child looked like a young Hercules, the pectorals and latissimus dorsi were atrophied. In the majority of cases the muscles of the calves and buttocks, and probably also the deltoids, are enlarged, while the remaining muscles of the arm, forearm, shoulders, and trunk are atrophied; so that the slenderness of the upper part of the body offers a strong contrast to the abnormal development of the inferior extremities. We see, therefore, that all the paralysed muscles do not undergo augmentation of bulk; in fact, atrophy of some of the muscles is a constant symptom of the disease. Another circumstance worth noting is that the degree of paralysis has no direct relation to the amount of hypertrophy. This is well illustrated in the leg where the action of the extensors of the foot, although these are much enlarged, predominates over that of the flexors, as evinced by the elevation of the heel.

The disease now becomes more or less stationary for two or three years, and sometimes for a much longer period, and as the general health is good and the muscular development

apparently very powerful, the parents cannot believe that the affection is incurable. This illusion is, however, after a time destined to be dispelled. The feebleness of the lower extremities gradually increases, so that the child cannot maintain the erect posture, while the muscles of the superior extremities also become both paralysed and atrophied; and even the hypertrophied limbs begin to waste, and to diminish rapidly in size. The patient, now arrived at adolescence, may live on for several years in a condition of almost complete paralysis, until finally death takes place from exhaustion, implication of the respiratory muscles, or more usually from some intercurrent affection.

There are still some minor features of the disease which deserve attention. The statements of different observers, with respect to the electro-muscular contractility, are somewhat contradictory. Except in the very early stages of the disease, the faradic contractility is diminished, while the galvanic contractility may be normal or increased. In the second stage of the disease the quadriceps tendon-reflex is completely abolished.

Very frequently the skin over the affected parts presents a peculiar mottled appearance, the colour varying in different cases, and in the same case according to the degree of exposure. Sometimes it is described as of a roseate hue, again as bright red, and at other times as consisting of patches of purplish colour alternating with white. All of these phenomena, however, indicate capillary congestion, the result of vaso-motor disturbance. This supposition is still further strengthened by the fact that the superficial temperature of the inferior extremities is frequently higher than that of the trunk.

This disease is often associated with a certain amount of mental incapacity. In several instances the subjects of it have been noticed to be slow in acquiring the power of speech, others are described as being obtuse in intelligence, and a considerable number have been idiots. The disease is not accompanied by any suffering, there is no alteration of sensibility, and the functions of the bladder and rectum are not interfered with, while the general health is not much affected until near the terminal period of the affection.

§ 432. *Course and Duration.*—The disease is essentially

chronic. It begins without fever or marked derangement of the functions of digestion, respiration, or circulation. As already stated, it consists of a first stage in which there is progressive enfeeblement of the lower extremities, saddle-back, and waddling gait. This stage may last a few weeks, months, or even a year before the commencement of the next stage. The second period is characterised by apparent hypertrophy of a certain number of muscles, usually beginning in those of the calf, and extending gradually to other muscles of the trunk and upper extremities. Increase in the volume of some muscles is always accompanied by atrophy of others. This stage of muscular hypertrophy continues to increase progressively, and attains its maximum in degree and extent about eighteen months from the beginning of the second stage of the disease; the symptoms then remain stationary for two, or three, and sometimes for many years.

The third stage of the disease is now ushered in by a still further enfeeblement of the muscles already affected, and by the extension of the paralysis to the superior extremities. Abduction and elevation of the arm is at first rendered difficult, then impossible, and by-and-by the paralysis gradually implicates the other movements of the arm.

The child, now probably arrived at the age of puberty, enters upon the last stage of the disease. The slight power of movement of which he was capable during the previous period becomes gradually lost, so that he can only sit in a chair or recline on a couch. The patient may continue to live for a long time in this condition, but eventually death supervenes from exhaustion or some intercurrent malady.

§ 433. *Diagnosis.*—When the disease is thoroughly established there can scarcely be any possibility of mistaking it for any other affection. The diseases which are most nearly related to it are infantile paralysis and progressive muscular atrophy in the infant. True muscular hypertrophy may also be mistaken for the disease, and a likely condition to be confounded with it is a late development of the power of muscular co-ordination and walking in children, especially when combined with a cerebral lesion, as in cases of idiotcy.

The invasion of *infantile paralysis* is sudden and accompanied with fever, and the distribution of the paralysis is totally different from that of the pseudo-hypertrophic variety. Sometimes the paralysis is limited to a few muscles or to an entire limb, at other times it is hemiplegic, crossed, paraplegic, or general. The muscles which are least injured recover completely, while others atrophy, and in the latter there is very early and decided diminution of electro-muscular contractility.

Progressive muscular atrophy in the child usually begins between the age of five and seven. Some of the facial muscles, principally the orbicularis oris and zygomatici, become atrophied. After a stationary period of some years the atrophy extends successively to the muscles of the upper limbs and trunk, and the lower extremities are not affected until a more advanced period. The muscles are invaded irregularly, and as the degree of paralysis is always proportional to the amount of atrophy this gives rise to various deformities of the trunk and limbs. When the atrophy attacks the extensor muscles of the trunk and some of the muscular groups of the lower extremities, as in the case of Charlotte A——, already described, progressive muscular atrophy is by no means easy to distinguish from pseudo-hypertrophic paralysis.

In making a diagnosis, the main reliance must then be placed on the history of the case, the progress of the symptoms, and a microscopic examination of portions of the muscles of the calf withdrawn by the trocar.

Simple muscular hypertrophy may be distinguished from pseudo-hypertrophic paralysis by the history of the case, the absence of paralysis and of the special symptoms of the latter disease, and if necessary by a microscopic examination of the muscle.

In late development of the muscular co-ordination in children the feet are not planted widely apart, and there is no saddle-back or waddling walk. When want of co-ordination is combined with idiotcy there is a flow of saliva from the half-open mouth, and the tendinous reflexes are generally exaggerated in the lower extremities.

§ 434. *Morbid Anatomy.*—The first examination of the condition of the muscles in this disease was made in Germany by

Greisinger and Billroth, who excised in a young living subject a portion of the left deltoid which was completely paralysed and hypertrophied. Duchenne, however, not liking to undertake such a serious operation, invented a small instrument, which he called his "Emporte pièce histologique," and which enabled him to obtain minute portions of muscular tissue from the living subject. A modification of this instrument, first proposed by Dr. Ord (Med. Chir. Transac., vol. lvii., 1874), and made by Hawksley, London, is generally used in this country for the purpose. But after repeatedly using Dr. Ord's trocar in various diseases, I am quite satisfied that the relations which the different elements of the diseased muscle bear to one another are not always accurately represented by the fragment of tissue withdrawn by the instrument. Charcot indeed suggests that Duchenne's instrument will withdraw islets of connective tissue, inasmuch as it will seize the fat cells with greater difficulty; and, judging from my experience of Dr. Ord's trocar, the objection is valid.

The happy idea occurred to Dr. Leech that an instrument might be constructed which would withdraw a portion of the muscle by cutting instead of by tearing; and Hawksley has made one at his suggestion, which answers the purpose admirably. The first muscular change which takes place in this disease consists of an increase of the connective tissue which separates the muscular bundles from one another, so that the sheaths of the muscular bundles become greatly thickened. There is also a corresponding increase of the connective tissue which passes between the fibres themselves. The comparatively thick masses of tissue which now separate the fibres from one another consist of fibres arranged parallel to the long axes of the muscular bundles, mixed with a considerable number of embryonic cells. In this early stage the muscular fibres themselves do not appear to undergo any very manifest changes, except that, according to Duchenne, their transverse striation becomes fainter, while the longitudinal striation becomes more marked. The transverse striation is, however, generally quite distinct until a late period of the disease. Duchenne regarded the proliferation of the connective tissue as the chief cause of the increased size of the muscle; hence he called the

disease "*paralysie myosclérosique;*" but other authors believe that the muscle does not increase much in volume until the second stage of the change occurs. This stage consists of the development of fat cells in the connective tissue and also in the newly-formed fibrous tissue, whereby the muscular fibres become widely separated from one another. The muscular fibres now become atrophied and begin to disappear. They become narrower, and indeed a single fibre varies in diameter at different points in its length. The transverse striation may sometimes disappear in the narrower fibres, and be replaced by granules distributed uniformly through them. Much of the fibrous tissue surrounding the fibres contains oat-shaped nuclei, which are supposed by some to be derived from the empty sheaths of muscular fibres (Clarke, Gowers). After a time both the muscular fibres and the newly-formed fibroid tissue completely disappear, and the entire muscle is represented by fat cells like those of an ordinary lipoma. The fat may subsequently become absorbed, and connective tissue, with perhaps a few traces of muscular fibres, is all that is left.

Condition of the Nervous System.—The brain and spinal cord have been examined in several patients who died from this disease, but the examinations possess no real value except in two or three instances. Even in the case reported by Eulenburg, where the cord was examined with great care by such a competent observer as Cohnheim, it has been justly objected by Charcot that delicate lesions like atrophy of the motor cells might escape detection, inasmuch as the cord was examined in the fresh condition, or only after imperfect hardening. If this objection be valid, when urged against an examination conducted by Cohnheim, how much more true does it become when either no microscopic examination or only a very imperfect one was made. In one of the cases collected by Duchenne the patient died in February, 1871, and his brain and spinal cord were carefully examined, both in the fresh state and after hardening in chromic acid. Portions were forwarded to Charcot, Vulpian, and Lockhart Clarke, and no abnormal appearances were detected. A large number of sections of the cord, at different levels of the cervical and dorsal regions, were made by M. Pierret, and coloured by carmine, but neither Charcot nor

he could detect any trace of disease. M. Barth examined the cord in the case of a man forty-four years old, who suffered from muscular pseudo-hypertrophy, and found partial degeneration of the antero-lateral columns, and diminution of the number of ganglion cells in the anterior horns of the cord. Charcot, however, justly points out that the clinical characters of this case were more like amyotrophic lateral sclerosis than pseudo-hypertrophic paralysis.

The most important case hitherto examined is the one reported by Drs. Lockhart Clarke and Gowers, in which "varied and extensive" lesions of the cord were found. These lesions were so numerous that only the most important of them can be mentioned here. The changes began on a level with the origin of the second cervical pair of nerves, and consisted of "disintegration of the lateral grey network which is so conspicuous in the region between the caput cornu posterioris and the tractus intermedio-lateralis, and through which the spinal accessory nerve makes its way into that tract." "One-half of the anterior white commissure was entirely destroyed." In the lower part of the cervical region there was disintegration of some of the "posterior nerve roots near the entrance into the caput cornu posterioris," and both the lateral and posterior white columns were in many sections damaged by sclerosis. In the upper portion of the dorsal region "the changes were less frequent and extensive, but here and there the anterior white commissure was partially destroyed." The lesions were "most extensive and striking" at the lower part of the dorsal region and the commencement of the lumbar enlargement. The central and lateral parts of the grey substance on each side were severely damaged by softening and disintegration. In the middle part of the lumbar enlargement the lesions were less serious, but in the lower portions and in the conus medullaris the lesions of the grey substance were again more extensive and severe. "The central part of the anterior cornu and the outer part of the cervix cornu posterioris were very much damaged by continuous disintegration." The large nerve cells in the anterior cornua were much diminished in number, and the few remaining cells were atrophied and contained an excess of pigment.

The following abstract of an important case of this disease, in which a post-mortem examination was obtained, I owe to the kindness of Dr. Leech, who is preparing a series of interesting cases of this disease for publication.

R. J——, aged seven, came under Dr. Leech's care at the Manchester Infirmary on the 20th of September, 1877, with the well-known symptoms of pseudo-hypertrophic paralysis. His walk and method of rising from the recumbent posture were quite characteristic, and lordosis was well marked. The calves of the legs were unduly large and firm, the arms and thighs thin, distinctly atrophied, the other parts of the body were badly nourished though not definitely wasted. The pectoral muscles were the most reduced in size and strength, whilst the deltoids were firm and large as compared with the other muscles of the shoulder and arm.

The boy could stand, though not without difficulty, for the heels could only be brought to the ground with effort ; his power of locomotion was of course limited, yet he could easily walk across a wide ward without falling. The boy had appeared quite healthy till he began to walk. When two years old it was noticed that he was not so firm on his legs as his brothers and sisters had been. As he advanced in age muscular weakness became more apparent. He fell constantly and had difficulty in rising ; he could only get upstairs with the aid of his arms. At three years of age the boy's limbs had lost their plumpness. The increase in the size of the calves of the legs was not noticed till six months before he came into the Infirmary. It does not appear that any other member of the family had been similarly affected.

The boy continued under my care two years, and then died of bronchitis. A slight amount of wasting went on in all parts of the body during this time, and the loss of muscular power was considerable.

Eighteen months before he died he became unable to walk or stand, and the rest of his life was passed in a chair or lying down. For the last six months he was unable to extend fully his legs, and sat in a bowed position owing to the weakness of the muscles of his back. The calves of his legs decreased slightly in size, but continued large as compared with the other parts of the body.

A post-mortem was made thirty-six hours after death. The muscles had, for the most part, lost their normal appearance, and were of a light yellowish brown colour. In some places it was difficult to distinguish them from connective tissue. This was specially the case with the pectoral muscles. The gastrocnemius looked on section like dark-coloured fatty tissue. In taking out the spinal cord a very distinct difference was noticed between the condition at the erector spinæ in the lumbar and upper dorsal region. In the former the muscles had a connective-tissue-like appearance ; towards the mid-dorsal region they became darker and redder, and in the upper dorsal region had the ordinary appearance of muscular tissue.

The muscles in the cervical region had the same appearance as those in the upper dorsal. The rhomboids, levator anguli scapulæ, and trapezius were distinctly altered in colour and texture, the upper part of the trapezius being the least affected.

The microscope seemed to show that nearly all the muscular tissue in the body was affected, for even in that taken from the upper dorsal region which looked healthy a distinct increase of connective tissue between the fibres was evident.

In the muscles which appeared to the naked eye most changed, such as the pectorals, the new connective tissue growth was very much more extensive, all the fibres of the primitive fasciculi being separated by it, whilst here and there single fibres ran alone widely separated by connective tissue from their companions.

Here and there rows of fat cells appeared sometimes between muscular

FIG. 172.

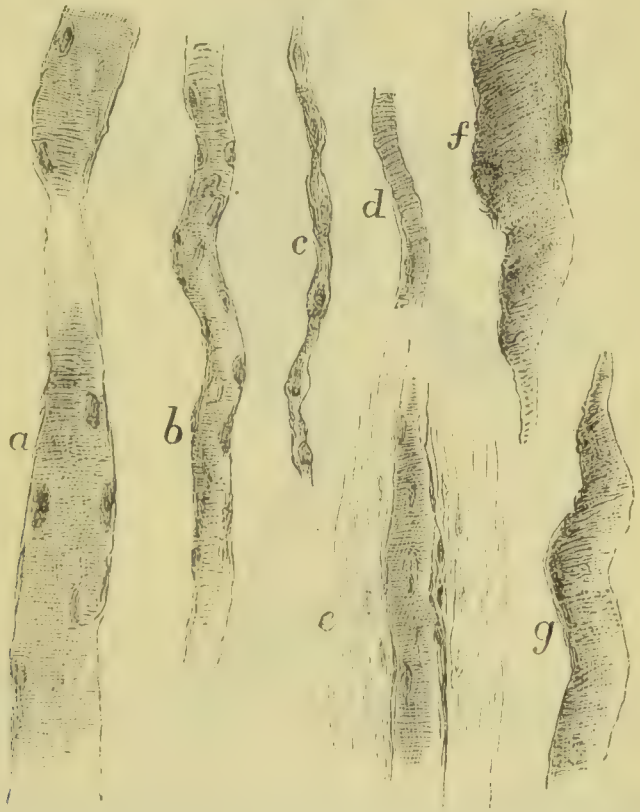


FIG. 172 (Young). *Muscular Fibres in various stages of degeneration, from a case of Pseudo-hypertrophic Paralysis.* — *a*, Muscular fibre only slightly changed, showing increase of the muscle corpuscles, and indistinctness of the transverse striation in certain parts of its length; *b*, the same as *a*, but more atrophied; *c*, muscular fibre greatly atrophied, and presenting nuclei at intervals; *d*, atrophied muscular fibre, with its transverse striation unusually distinct; *e*, atrophied fibre surrounded by a fibrillated connective tissue rich in nuclei; *f* and *g*, muscular fibres from the erector spinæ, which manifested the greatest changes to the naked eye. These fibres appear to have undergone a hyaline change, but their transverse striation is still faintly visible. The fibres often tapered to a point, sometimes at one and sometimes at both ends.

fibres, sometimes surrounded by connective tissue, and in places accumulations of fat cells were met with instead of single rows.

In the gastrocnemius muscle the same condition was present, but the fat cells were much more abundant and formed loose accumulations of adipose tissue amid the muscular fibres and connective tissue.

In all the muscles the fibres were distinctly narrowed, and the nuclei of the sarcolemma were greatly increased in number, but the striation of the fibres was for the most part not interfered with and was often unusually distinct, even in fibres which had undergone a high degree of atrophy (*Fig.* 172, *c*, *d*). The annexed diagram (*Fig.* 172) represents the more usual appearances presented by the altered muscular fibres.

In very few of the fibres was granular change met with, whilst hardly any good examples of true fatty change were seen.

The decrease in the diameter of the muscular fibre seemed most marked in the muscles which were most changed to the naked eye. In the erector

Fig. 173.

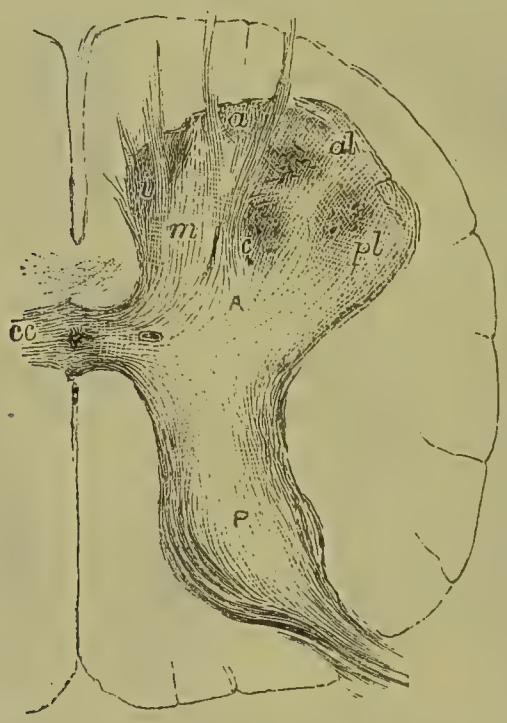


Fig. 173 (Young). *Transverse Section from the lower half of the Lumbar Enlargement of the Spinal Cord, from a case of Pseudo-hypertrophic Paralysis.*—A, Anterior grey horn; P, Posterior grey horn; *cc*, central canal; *i*, internal, *a*, anterior, *al*, antero-lateral, *pl*, postero-lateral, *c*, central group of ganglion cells; *m*, median area.

spinæ from the upper dorsal region, for example, it was hardly manifest; whilst many of the fibres of the pectoral muscle were reduced to one-sixth of their normal diameter. The narrowing of the muscular fibres seemed indeed proportionate to the extent of the development of the new connective tissue.

Dr. Leech kindly sent the spinal cord to me for examination. In the lumbar region the normal loose and spongy texture of the central column was replaced by a somewhat dense and fibrillated tissue, in which no trace of ganglion cells could be found. The blood-vessels were enlarged, and their walls thickened. In the anterior grey horns the ganglion cells had completely disappeared from the median area, the anterior group, and the margins of all the other groups (*Fig.* 173). The ganglion cells could be distinctly seen in the internal group (*Fig.* 173, *i*), but they were atrophied, and only a few of their processes could be discovered, and the central group (*Fig.* 173, *c*) presented one or two cells only which were not distinctly atrophied. The central portions of the antero-lateral and postero-lateral (*Fig.* 173, *al*, *pl*) groups, however, contained some cells which appeared

FIG. 174.

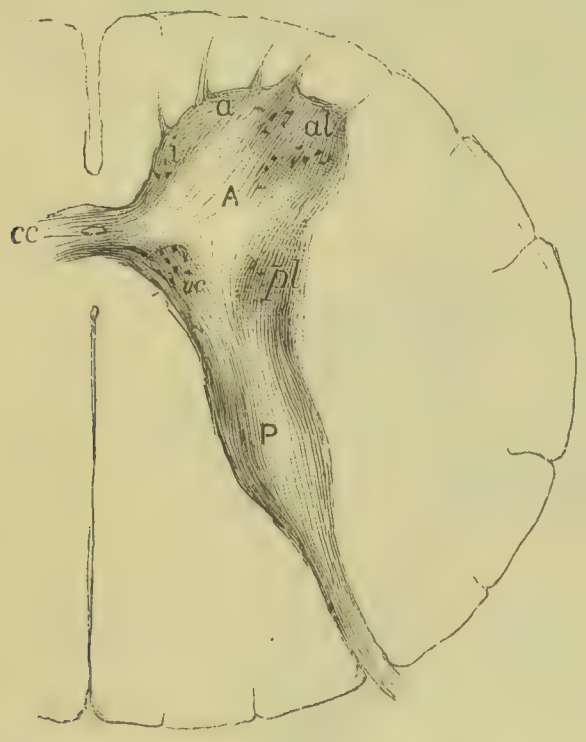


FIG. 174 (Young). *Transverse Section from the middle of the Dorsal Region of the Spinal Cord, from a case of Pseudo-hypertrophic Paralysis.*—*vc*, The vesicular column of Clarke. The other letters indicate the same as the corresponding letters in *Fig.* 173.

in every respect normal, but others contained an excess of pigment, while the marginal cells were decidedly atrophied.

In the dorsal region the central column presented the same general appearances as in the lumbar enlargement. The disease extended into the anterior grey horn in the dorsal region chiefly in the area which lies between the antero-lateral and postero-lateral groups (*Fig.* 174, *al*, *pl*)—the medio-lateral area. The ganglion cells of the postero-lateral group were atrophied and destitute of processes to a very marked degree. The

cells could not be seen in the anterior (*Fig.* 174, *a*) nor in the central group, but those of the internal and antero-lateral groups were distinctly visible. Many of the latter, however, contained an excess of pigment, and appeared to have lost a considerable number of their processes. The cells of the vesicular column of Clarke appeared normal or only slightly altered.

In the cervical region the central column also presented the same general appearances as the other portions of the cord just examined. In the lower half of the cervical enlargement, however, the median area of the anterior horns contained beautiful healthy cells, and it contrasted strongly in this respect with the median area in the lumbar enlargement. The internal group of cells were also healthy, while healthy cells were seen in the anterior group. The marginal cells of the central, antero-lateral, and postero-lateral groups were, however, atrophied, while many of them had disappeared. In the upper end of the cervical enlargement the central and antero-lateral groups appeared to have been more diseased than any other portion of the anterior horn (*Fig.* 175, *c*, *al*).

Fig. 175.

Fig. 175 (Young). *Transverse Section from the upper half of the Cervical Enlargement of the Spinal Cord, from a case of Pseudo-hypertrophic Paralysis.* The letters indicate the same as the corresponding letters in *Fig.* 173.

§ 435. *Pathology.*—This disease is so frequently associated with obtuseness of the mental faculties, or with idiotcy and cretinism, that Duchenne was at first inclined to believe that the muscular changes resulted from cerebral disease. More extended

observation, however, soon showed that this affection frequently exists independently of any cerebral lesion. The dilatation of the capillaries of the skin over the affected muscles, and the frequent elevation of the superficial temperature of the limbs, as compared with that of the trunk, have led some pathologists to think that the primary lesion is situated in the vaso-motor nervous system, but no additional facts have been discovered to verify this supposition. There still remains the question, whether the disease is primarily in the muscles, or in that part of the nervous system which controls their nutrition. Charcot and Friedreich regard the muscular lesion as the essential one. Friedreich, however, looks upon progressive muscular atrophy also as primarily a muscular disease; and, indeed, he considers the two diseases as essentially the same, although each is somewhat modified by circumstances.

Charcot, on the other hand, who believes that progressive muscular atrophy is primarily a nervous disease, regards pseudo-hypertrophic paralysis as a primary disease of the muscular tissue. Charcot grounds his opinion mainly on the fact that he could detect no lesion in the cord which he had examined for Duchenne; and considering how thoroughly competent he is to decide such a point, it must be admitted that the objection against regarding the disease as of nervous origin is an exceedingly strong one.

Passing over the case observed by Barth, as not being an undoubted example of the disease under consideration, the case observed by Drs. Lockhart Clarke and Gowers speaks strongly in favour of the nervous origin of the affection. It is evident, from the description, that extensive changes had occurred in the central column, and the postero-lateral group of cells of the anterior cornua, throughout the greater part of the lumbar enlargement and lower dorsal region of the cord. I observe, however, that even Dr. Gowers has abandoned the nervous theory of pseudo-hypertrophic paralysis, and we must leave the question an open one at present.

In any subsequent case the condition of the ganglion cells of the postero-lateral and medio-lateral groups in the lumbar and dorsal regions of the cord should be carefully examined. In the spinal cord which I examined the changes found in the central

grey column and anterior grey horns corresponded so closely with those observed in progressive muscular atrophy, that I see no reason to question the essential unity of the two diseases.

Even if pseudo-hypertrophic paralysis were proved, beyond doubt, to be a primary disease of the muscular tissue, yet the clinical affinities between it and progressive muscular atrophy are so numerous as to justify us in considering the diseases in the same category.

§ 436. *Prognosis.*—In two cases under the care of Duchenne, the disease was arrested in its first stage by treatment. This shows that the prognosis is not absolutely hopeless. When, however, the second period, or that of apparent hypertrophy of the muscles, has set in, the case is in all probability beyond the reach of treatment, and it is still more surely progressive and fatal in the period of atrophy.

§ 437. *Treatment.*—Duchenne relied mainly on the faradic current in the two cases which he cured. Baths, friction, and shampooing were also employed as subsidiary means. The galvanic current applied over the sympathetic nerves has been recommended by Benedikt, but this method has not been found to possess any advantage in the hands of others. An improvement in motor power took place in a case under the care of Rosenthal by the application of the constant current several times a week to the nerves of the hypertrophied muscles, along with the daily use of rubbing and the cold plunge bath. Gymnastics and mountain air aided the treatment. The iodides of iron or of potassium, or when there are cerebral symptoms the bromide of potassium may be tried; but the remedies which promise to do most good are the so-called tonics. Arsenic, Parrish's chemical food, phosphorus, and cod-liver oil, and in some cases strychnia or nux vomica, may be tried.

CHAPTER IV.

I.—SYSTEM DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

(II.) LEUCOMYELOPATHIES.

1. *Progressive Locomotor Ataxy.*

(Tabes Dorsalis; Grey Degeneration, or Sclerosis of the Posterior Columns.)

§ 438. *Definition.*—Locomotor ataxy is a chronic disease, characterised by the progressive abolition of the power of co-ordinating the general movements concerned in locomotion, and other voluntary actions, but without distinct loss of muscular power.

§ 439. *History.*—An accurate knowledge of this disease, which is now so well known, is of comparatively recent date, although obscure intimations of the affection may even be found in the writings of the Greek physicians. In 1679 Bonnet appears to have revived the name of tabes dorsalis, which had fallen into disuse, and he also associated the symptoms with anatomical changes, principally atrophy of the spinal cord. Individual observations of the disease may be found in the works of Ollivier, Cruveilhier, Hutin, and Stanley, and the phenomena of ataxy were described with great accuracy by Bell, Nasse, and Landry; but W. Horn was the first to give an accurate account of the course and symptoms of ataxy, and to recognise it as a distinct type of disease. Horn's description appeared in 1827, and the classical monograph of Romberg was published in 1840, while the anatomical investigations of W. and E. Horn, Jacoby, Froriep, and, above all, the microscopic researches of Rokitansky and Türck proved that the symptoms were caused by grey degeneration of the posterior columns of the cord. The disease was most accurately described in England in 1847 by Todd, who directed special attention to the fact that the characteristic symptoms were due to want of power in co-ordinating movements, and not to deficiency of voluntary power. French physicians had completely neglected the affection until Duchenne

published his memoir in 1858, and so profound was the impression produced by the exhaustive analysis and accurate descriptions of this author that he was for a long time regarded as the discoverer of the disease. The writings of Duchenne and Trousseau contributed more than any others to obtain for this affection the recognition of the profession. Duchenne did not make any contribution to the pathological anatomy of the disease, but he thought that the deficiency of motor co-ordination must be dependent upon some structural or functional lesion of the cerebellum. He suggested that the central morbid process began in the motor nerves of the eye and the corpora quadrigemina, from which it extended to the superior peduncles of the cerebellum, and lastly to the cerebellum itself. This view was combated by Eisenmann, and a lively discussion ensued which had the effect of settling definitely that the most constant anatomical lesion in locomotor ataxy is grey degeneration or sclerosis of the posterior columns of the spinal cord. Amongst the authors who contributed to establish this view may be mentioned Duménil, Bourdon, Oulmont, Marrotte, Charcot and Vulpian, Luys and Carré in France; Rindfleisch, Westphal, Friedreich, and Leyden in Germany; and Lockhart Clarke in England. In its clinical relations the disease has been investigated amongst others by Jaccoud, Topinard, Axenfeld, Remak, Spaeth, Cyon, Benedikt, and many other authors.

The name of the disease has also undergone frequent changes in accordance with the prevailing doctrines with regard to its nature. When it was regarded as being due to atrophy of the spinal cord, it received the name of "Atrophia Medullæ Spinalis," a name which had to be abandoned on more accurate histological investigation. Wunderlich called it "Progressive Spinal Paralysis," but this name became untenable when it was found that the symptoms did not depend upon paralysis but on want of co-ordination. In the present day three names are employed indifferently to designate the disease. These are "Grey Degeneration or Sclerosis of the Posterior Columns of the Cord," "Progressive Locomotor Ataxy," and "Tabes Dorsalis." None of these names are entirely free from objections but they have got possession of the field, and it would occupy space to very little advantage to discuss here all objections which might be urged against them.

§ 440. *Etiology.*—The etiology of the disease is very obscure and in many cases no definite cause of it can be traced.

Hereditary predisposition undoubtedly exercises a certain amount of influence in its production. Locomotor ataxy is frequently met with in individuals whose nearest relatives are liable to suffer from other nervous diseases, such as monomania, hypochondriasis, epilepsy, migraine, mental diseases, or violent fits of anger and drunkenness. Trousseau

mentions the case of a patient suffering from advanced locomotor ataxy, whose uncle and aunt were insane, and who had one brother ataxic and another hemiplegic. He also mentions the case of another patient who had been ataxic for upwards of twenty years, but whose intellect was perfectly clear. His father committed suicide; and his two sons laboured under peculiar nervous affections, one having singular muscular spasms, and the other being irresistibly compelled to shriek in a most extraordinary manner nearly all day. In other cases the influence of heredity is direct from the parent. Friedreich met with three different families in which several brothers and sisters were attacked with the disease at almost the same age, while the parents themselves were healthy. Carré was informed by an ataxic patient that seventeen other members of her family were affected by the same disease. Dr. Dreschfeld has recorded an instance of a family in which five out of fifteen, and Dr. Gowers one in which five out of nine children were affected with locomotor ataxia. In many cases of tabes no hereditary neuropathic tendency can be traced.

The male is much more liable to be affected with the disease than the female sex, no doubt greatly owing to the fact that men are much more exposed than women to the most powerful exciting causes of the affection, such as exposure to cold and sexual excesses. Out of 149 cases collected by Eulenburg 128 were males and 21 only females, so that the number of the latter affected in proportion to the total number was barely 14 per cent. The following table, given by Eulenburg, shows not only the proportion between the number of males and females, but the number which occurred at various ages:—

	Male.	Female.
From 0 to 10 years	—	1
„ 10 „ 20 „	2	—
„ 20 „ 30 „	35	12
„ 30 „ 40 „	39	7
„ 40 „ 50 „	47	1
„ 50 „ 60 „	5	—
After 60 years	—	—
	128	21

From this table it will be readily seen that locomotor ataxy is a disease of youth and middle age, by far the largest number

of cases occurring from thirty to fifty years of age. The disease is rare before the twentieth and after the fiftieth year.

Those who from the nature of their occupation are obliged to expose themselves to cold and wet and to other bodily hardships, such, for instance, as commercial travellers, engineers, soldiers, and sailors, are very liable to be affected with locomotor ataxy. It is notorious that soldiers are particularly liable to be affected with the disease after bivouacing on damp ground.

Severe bodily and mental exertion both predispose to the disease and act as exciting causes in its production. The severe struggle for existence to which the inhabitants of large towns are subjected explains, perhaps, why the disease is relatively more frequent in large cities than in the country. The largest number of cases of tabes are probably caused by excessive bodily exertion and subsequent exposure to damp and cold, hence the frequency with which tabes occurs in soldiers after forced marches in cold weather.

Emotional disturbances, such as sudden fright, continued anxiety, and repeated anger, appear occasionally to be capable of being the starting point of tabes.

Locomotor ataxy is an occasional sequel of acute diseases, such as typhus, articular rheumatism, acute pneumonia, and above all diphtheria; but it is difficult to determine whether these affections act as predisposing causes or whether they take a direct part in setting up nutritive changes in the cord. Difficult labours and repeated abortions, severe puerperal affections, copious hæmorrhages, and long-continued lactation are mentioned as other causes of this affection. Syphilis is a frequent cause of locomotor ataxy, although probably not so frequent as was at one time supposed.

At one time sexual excess and onanism were regarded as almost the only causes of tabes, and the unfortunate victims of the disease were often unjustly suspected of leading secretly immoral lives. That sexual excess, however, is a very important exciting cause of the affection is shown by the frequency with which it occurs in men during the period of their greatest sexual activity, as well as by the fact that the disease has been known to follow immediately upon great sexual excesses. Frequent pollutions and spermatorrhœa often precede the

outbreak of tabes, but whether these are the cause of the disease or mere results of the primary sensory disturbances which are so common in the early stage of the affection is difficult to determine.

Various traumatic injuries may be the starting point of locomotor ataxy, and instances are recorded in which the disease soon followed a fracture of the thigh, a fall upon the belly, the shock of a gunshot wound, and concussion of the spinal cord (Schulze). Some think that the disease may also be caused by excessive tobacco smoking, but the statement appears to be unfounded. In a great number of cases of tabes no recognisable cause can be traced after the most careful investigation.

§ 441. *Symptoms.*—Locomotor ataxy generally begins with a premonitory stage, which may extend over months or years. The most constant and characteristic premonitory symptoms are *pains* of a very peculiar and distressing kind, which are not only present during the initial stage, but usually accompany the disease throughout its entire course, and which may last for years without any other symptoms being present.

These pains have been described under the names of general neuralgia or neuralgic rheumatism, and are compared by the patients to forked lightning darting through the body (Lightning Pains, § 53). The pains are at other times of a burning character, and are not unfrequently confined to a small well-defined spot of the skin (Hyperæsthetic Spots, § 52). Sometimes the pains may be deeply situated in the soft parts or in the bones, or they may follow certain definite nerve tracts, and are often regarded as rheumatic. The nerve trunks may be sensitive to pressure during a paroxysm. The intensity of the pain varies in different cases, and at times patients suffer the greatest torture from them.

Pain in the back is met with occasionally in tabes, and at times points painful to pressure may be found on the spinous or transverse processes of individual vertebra, but those are rare, and appear also to be quite unimportant. Whenever there is prolonged or severe pain in the back, it may be suspected that the disease is complicated by spinal meningitis.

The feeling of a tight girdle round the thorax or abdomen,

which is so frequent a symptom of many spinal affections, is also a frequent symptom of locomotor ataxy (Girdle Sensation, § 51). Girdle pains may also be felt round the joints of the lower extremities, and it is sometimes described as a feeling like that caused by a garter tied tightly below the knee. Sensations of formication, numbness, or of burning or coldness of the skin are frequently complained of. Some patients feel as if they were walking on wool, cork, or felt soles; while others feel as if they were walking on bladders of water. These paræsthesiæ belong to the earliest stage of locomotor ataxy, and one or other of them is almost constantly found in the initial stage of the affection. Hyperæsthesia of the skin is not uncommon in tabes; and at times there may be hyperæsthesia towards impressions of temperature and anæsthesia of touch, and at other times anæsthesia of the sense of touch may be accompanied by a high degree of hyperæsthesia towards impressions of pain.

But anæsthesia is a much more common symptom of locomotor ataxy than hyperæsthesia. There may be a high degree of anæsthesia without the patients being aware of it; but after a time they find that they no longer feel the floor distinctly, that all articles which they touch have a velvety feel, or that they cannot hold small objects in their fingers. If there is a high degree of anæsthesia, the patients cannot judge of the position of their legs when in the dark. Anæsthetic patches may be found on the soles of the feet, the toes, and back of the feet, and they may be so limited that they can only be recognised by the most careful examination. As a rule, however, the diminution of sensation extends to the thigh, and even the trunk and portions of the upper extremities, although it is generally most marked on the legs. But the cutaneous anæsthesia in locomotor ataxy hardly ever reaches the high grade observed in the later stages of transverse myelitis, and slow compression of the cord.

Every variety of paralyses of sensation and every combination of them are met with in the later stages of the affection; but probably analgesia is the most frequent. Occasionally, however, the sensibility to pain is retained, or even increased, while there is a diminution of sensibility to some or all varieties

of touch; and, again, partial paralysis of the sense of touch may be combined with analgesia and hyperalgesia, or with hyperæsthesia towards impressions of temperature. At a late period of the disease a distinct retardation of the conduction of sensations, especially of impressions of pain, is observed, and this may also be found in the earlier stages of the affection. The prick of a needle frequently gives rise to a double sensation, the first being one of touch which is conveyed with normal rapidity, and the second of pain owing to the slowness with which the impression is conveyed. Hertzberg has demonstrated that in some cases the sensations of touch and of temperature are also retarded, although to a less degree than that of pain. The sensation of pain also continues for a relatively long time, even when the cause which has induced it has been of momentary duration, and the highest degree is not reached until several seconds after the pain has begun (§ 49). Fischer has recently observed that in certain circumscribed cutaneous areas of the foot the patient may feel two points when one only is touching the skin, and when two points are in contact with it four or five may be felt (Polyæsthesia, § 50). Disturbances of the muscular sensibility and muscular sense are frequently observed in this affection. In the first stages the alteration of the muscular sensibility consists of a feeling of unrest in the limbs, which prevents the patient from lying down or sitting still for any length of time, a feeling which has been graphically called the fidgets. It is probable that the feeling of fatigue, which is so frequent at the beginning of the disease, is a parasthesia of the sensitive nerves of the muscles. It must, however, be remembered that even in the early stage of the disease the locomotive movements require a greater amount of attention on the part of the patient, and that a much greater effort is expended in walking than in health.

As the disease advances, the muscular sense becomes diminished, and consequently the power of recognising what muscles are thrown into action is lessened in corresponding degree. When the paralysis of the muscular sense attains a high degree, the patient does not know the position of his lower extremities when his eyes are closed, and is also uncertain with regard to the extent and direction of the movements he

undertakes; hence these movements, not being under due control, become excessive. This condition must, however, be carefully distinguished from the ataxic movements about to be described.

Although disturbances of sensibility are much more constant and marked in the lower than in the upper extremities, yet the latter are frequently involved, especially in the advanced period of the disease. Cases of pronounced ataxia have been described by competent observers, in which the most careful investigation failed to detect the slightest trace of any disturbance of cutaneous or muscular sensibility. On the other hand, cases have been observed in which a high degree of anæsthesia was present, but in which the ataxic symptoms were either entirely absent or little pronounced, so that it may be concluded that there is no constant relation between the degree of ataxy and that of cutaneous or muscular anæsthesia.

The motor disturbances constitute by far the most characteristic features of locomotor ataxy. The motor disturbances were for a long time thought to be of a truly paralytic nature, but Todd and subsequently Duchenne showed that the characteristic gait of ataxy was due to a want of certainty and precision in the execution of movements, especially of combined and complicated movements; while the strength and certainty of simple movements is not at all, or only slightly, diminished. Duchenne indeed gave the name of ataxy to the disease from the recognition of the circumstance that the characteristic gait depends on a want of co-ordination of muscular action and not upon true paralysis. The motor disturbances almost always begin in the lower extremities, and are at first so slight that they can only be recognised by careful examination.

Static Ataxia.—During the early stage of the disease special tests, which have the power of increasing the motor incoordination, are very valuable in enabling us to determine the true nature of the affection. If the patient be asked to stand up and keep his feet closely applied together along their inner edges, he may manage to maintain the erect posture with moderate steadiness when his eyes are open; but when they are closed, he immediately oscillates from side to side, and would fall unless he open his eyes or be supported. As the disorder of

muscular co-ordination increases, standing without support, even when the eyes are open, becomes increasingly difficult, and station becomes by-and-by impossible without the aid of sticks or crutches. When at this stage the patient stands by the support of two sticks, it may be observed that all the extensor muscles of the body are in a state of powerful tonic contraction. The muscles of the calf are strongly contracted and extend the leg upon the foot, so that they form an obtuse angle with the other, the extensors of the leg are contracted and extend the thigh on the leg, the flexors of the thigh are also contracted, and the foot being fixed, they tend to extend the trunk on the thigh, this tendency being greatly increased by contraction of the gluteal muscles and of the erector spinæ. It is evident that if the contractions of these muscles were unantagonised, the patient could not for a moment maintain the erect posture but would fall backwards. The tendency to fall backwards is counteracted by what appears to be a voluntary contraction of the muscles which flex the trunk on the thighs. By this means the body is bent forwards, and the line of gravity falls in front of the line joining the centre of the arches of the feet, while the tendency to fall forwards is counteracted by the support of the two sticks. The attitude assumed by the patient under these circumstances is characteristic. The legs are drawn backwards so as to form an obtuse angle with the feet, the thighs are extended on the legs, and a plumb-line let fall from each trochanter falls considerably behind the heel, while the forward inclination of the body causes the buttocks to project backwards in a marked manner.

Ataxic Gait.—In the early stage of the disease the patient may be observed to stagger a little on getting up, especially after sitting for a long time, the staggering being greatly increased when the patient is in the dark, closes his eyes, or has to turn abruptly round. These phenomena were demonstrated to me in a striking manner a few weeks ago. I was walking on a moonlight night in a garden with a friend who has suffered for upwards of twelve years from the lancinating pains of the disease, and who is now manifesting slight ataxic symptoms. Our walk terminated under the shadow of a high wall and tree covered with thick foliage. So long as my friend was in the

moonlight he walked steadily enough, but when once we got under the deep shadow the staggering became very apparent, and was much aggravated when we turned round.

In order to test at this early stage the degree of precision with which the muscular adjustments of the lower extremities can be performed the patient may be asked to stand on one leg, to run, or to hop, these movements being more difficult to execute than simple walking. He may also be requested to perform some complicated movement with the extremity, such as to describe the outline of a circle on the floor with the toe.

When the ataxia becomes more pronounced, the gait becomes so characteristic that it can be readily recognised without the application of any special tests. The patient has now to direct his eyes to the ground and to his feet while walking, and were he to close them the movements of the legs would become disorderly, and walking would be impossible. The patient assumes in the erect posture the attitude already described, in which the trunk is bent forwards on the thighs, the feet are held well in advance of the buttocks, and the legs are extended on the thighs. It is impossible in this position to advance the passive leg with the pendulum movement characteristic of normal locomotion. And, indeed, owing to the strong tonic contraction of the muscles of the thigh and the extensors of the foot which is present, the various segments of the passive leg cannot be flexed upon one another so as to enable it to clear the ground during its forward movement. Under these circumstances the passive leg is projected forwards in one piece by strong contraction of the flexors of the thigh on the trunk, aided by contraction of the abductors of the thigh. The consequence is that the passive foot is flung forwards and outwards with a rapid jerk, being subsequently brought down with a thump. During this movement the heel is generally the last to leave the ground and the first to touch it. The heel is, however, sometimes lifted from the ground before the toe, as occurs in a case under my care at present. As the passive foot is being elevated a slight flexion occurs at the knee-joint and the heel is elevated before the toe, but no sooner is the latter removed from the ground than the leg becomes suddenly extended on the thigh, the foot is projected forwards and outwards, and the heel is subsequently brought

down with a thump in the usual manner. In order to enable the passive leg to clear the ground during its forward movement, the abductor muscles of the thigh on the side of the active leg enter into strong contraction, and consequently elevate the pelvis on the side of the passive leg. So strong, indeed, does the contraction of the abductors of the thigh on the side of the active leg become that the patient is in danger of carrying his centre of gravity too far to that side. In order to counteract this tendency the upper part of the body is curved to the opposite side by contraction of the erector spinæ, and when the patient is able to walk without sticks, by the arm on the side of the passive leg being thrust out laterally, and during the alternate transference of the line of gravity from one foot to the other in walking, the trunk is moved from side to side and the arms flung about like those of a rope dancer in order to assist the patient to maintain his equilibrium. When the patient walks by the aid of sticks the tendency to too great a lateral displacement of the centre of gravity towards the side of the active leg is counteracted by the patient giving an outward inclination to the sticks, so that he obtains a lateral support from them. Patients who have suffered from a high degree of ataxia have been known to walk long distances without fatigue.

When, however, the disease has made considerable progress, the irregularity and violence of the movements soon exhaust the patient's strength, and he cannot take many steps without panting and being covered with profuse perspiration. After a time the want of co-ordination becomes so great that the maintenance of the erect posture and walking become impossible. If the patient be supported by two persons under the arms whilst he tries to walk, his legs are thrust backwards and forwards to the right and to the left with the utmost disorder, so that they are incapable of giving the least support to the body; they move, as Trousseau remarked, like those of a puppet or a marionnette. The muscles of the trunk may now become affected, the patient is unable even to sit in a chair, and remains confined to his bed. But even in these advanced cases, the patient when laid down may be able to resist passive movements of the limbs, and to perform the simple movements of flexion and extension with scarcely diminished power. When,

under these circumstances, the patient attempts to touch an object with the tip of the foot, the line of motion is irregular and zigzag, and disturbed by lateral movements, while it is quite impossible for him to execute more complicated movements—such, for instance, as are required in describing an imaginary circle with the tip of the great toe.

At a later period the ataxy appears in the upper extremities, and cases have been described by Friedreich in which the ataxy appears in the upper simultaneously with or soon after its first manifestation in the lower extremities. In the more usual form of the disease, however, ataxy of the upper extremities is rare, and belongs to the later manifestations of the affection. Ataxy of the upper extremities first manifests itself in complicated and special movements, such as those required for writing, playing the piano, and other movements requiring delicacy of manipulation. These movements become difficult and uncertain, and the irregularity becomes greater if an attempt is made to perform the necessary actions without the guidance of the eyes. At a later stage the simpler movements also become irregular and ataxic. If the patient now attempts to grasp an object before him, it can only be reached in a roundabout way and with jerky interruptions, and the act of grasping is performed in an uncertain and spasmodic manner. The slighter shades of ataxy of the upper extremities may be tested by instructing the patient to touch with closed eyes some part of the surface of the body, such as the forehead or tip of the nose by the point of the forefinger of each hand alternately, when the ataxic symptoms declare themselves by the inability of the patient to touch the intended spot until after repeated trials. A similar uncertainty of movements is observed when the patient is asked to transfer a small object from one hand to the other. Static ataxy at a later period may be present in the upper extremities, so that the patients can no longer hold their arms still when stretched out horizontally, and are unable to exert uniform pressure with their hands. In a still higher grade patients can no longer dress nor feed themselves, inasmuch as they cannot perform such simple movements as are requisite for carrying a spoon to the mouth, but even under these circumstances they may be able to exert great muscular power in resisting passive movements.

The ataxy may also invade the muscles of the trunk, so that the body makes irregular, swaying movements, owing to the impossibility of maintaining the due balance between the various muscles, contraction of which is necessary for maintaining the erect posture. The muscles of the neck may also be implicated, and the head become the subject of irregular and shaking movements. Speech is sometimes also interfered with. At first the ataxy declares itself by a somewhat indistinct pronunciation of words, but when the affection is more advanced, there is an irregular, stuttering interruption of speech. At times whole sentences are uttered rapidly, and then there is a slight stuttering, and this is repeated in an irregular manner; while the voluntary movements of the lips and tongue are apparently quite unaffected. In the highest grades of the affection articulation may become so defective that speech becomes almost incomprehensible.

The third and final stage of the disease is characterised by decided paralysis, although a certain amount of motor weakness may be shown to be present in the majority of cases of tabes even at a moderately early stage of the ataxic period. Partial and temporary paralysis in the domain of single nerves in the extremities is not of rare occurrence. In the later stages of the disease true motor paralysis increases and ultimately becomes the predominant symptom. The muscles undergo atrophy, or contractures set in, and they finally become more or less completely paralysed. With the appearance of the paralysis the ataxic symptoms are thrown more and more in the background.

Symptoms indicative of motor irritation are not prominent features of locomotor ataxy. In the earlier stages of the disease fibrillary contractions and spasms of single muscles, with slight jerking of a limb, may be observed. At times twitchings of entire extremities may occur in connection with the lancinating pains, and are doubtless reflex in nature.

Muscular tension is also entirely absent from true cases of tabes; the limbs are limp and do not offer the least resistance to passive movements. When, however, the paralytic symptoms supervene muscular tension and contractures also arise, and may ultimately reach so high a grade that the limbs remain im-

movable in the position of extension or flexion as they do in the later stages of other chronic spinal diseases.

The electrical reactions vary at different stages of the disease, and the statements made by different authors with regard to them are not in accord with one another. Erb found the faradic and galvanic excitability to be quite normal in respect both to quality and quantity. In another series of cases he found a slight increase in the faradic and galvanic excitability in the anterior muscles of the leg, without any qualitative alterations, while in other cases he found a more or less distinct diminution of electrical excitability in the anterior muscles of the leg, without qualitative changes. From these cases Erb draws the general conclusion that in the earlier stages of the disease there is an increase and in the later stages a diminution of the electric contractility. But, as Erb confesses, no great advantage is to be gained either for diagnosis or prognosis from electrical examinations.

Reflex Action.—The cutaneous reflex is usually unaffected in locomotor ataxy, at least until a late period of the disease. In some cases, however, the normal interval between the cutaneous excitation and the resulting contraction may be greatly prolonged (Fischer). The absence of the reflex action of the tendons (§ 78) constitutes, as has been pointed out by Westphal, one of the most remarkable features of the affection. The patellar-tendon reflex is usually absent in the premonitory stage of the disease, and often long before the ataxic symptoms make their appearance, and it is consequently one of the most valuable signs of the disease which we possess. It must, however, be remembered that the absence of the patellar reflex is not absolutely pathognomic of the disease. Erb found this reflex absent in forty-eight out of forty-nine cases examined by him, but in the one exception the reaction was very lively. have at present under my care a woman, who developed symptoms of ataxia somewhat suddenly two years ago after a miscarriage, and in whom the patellar-tendon reflex is in excess. have seen another case, which will be subsequently mentioned where symptoms of ataxia were associated with excessive reaction of the patellar-tendon reflex, but the subsequent course of the case showed that it was one of insular sclerosis. I thought for

some time that the case of the woman just alluded to would turn out to be of the same character; but after watching the progress of her case now for eight or nine months, I can come to no other conclusion than that it is one of true locomotor ataxy. The ataxic gait and swaying movements on closing the eyes are well marked in her, it is true that she has not suffered much from lancinating pains, but there is decided diminution of tactile sensibility in the skin over the external aspect of both legs, there has been some dribbling of urine, and there is complete absence of paralysis and muscular tension. But not only does the patellar-tendon reflex remain unaffected or even in excess in cases of true tabes, but it is sometimes absent in those who are otherwise typically healthy, so that some degree of caution is necessary in accepting the absence of this phenomenon as a sign of ataxia. With these reservations, however, the absence of this reflex is a most valuable sign of locomotor ataxy.

§ 442. *Occasional Symptoms.*

Paradoxical Contraction. — Attention has recently been drawn by Westphal to a curious phenomenon, which may be regarded as the opposite of the tendon-reflex contraction of the muscle. As this symptom was unfortunately overlooked by me when the first volume of this work was passing through the press, I shall describe it in detail in this place:—

It consists in the contraction of a muscle induced by suddenly approximating its points of origin and insertion. The curious circumstance that a sudden relaxation of a muscle causes it under certain circumstances to contract has led Westphal to name this phenomenon *paradoxical contraction.* This symptom is best studied in the tibialis anticus, which may in certain diseases of the central nervous system be made to contract by producing sudden or sometimes a gradual dorsal flexion of the foot. When the patient is laid on his back in bed, and the muscles are relaxed, especially if they be paralysed, the feet occupy the position of extension or plantar flexion. If dorsal flexion of one foot be now produced, the tibialis anticus, under certain circumstances, contracts, its tendon becomes prominent, and the foot is maintained for some minutes, sometimes even as long as twenty-seven minutes, in the position of dorsal flexion and adduction. When the muscle is made to contract by direct or indirect excitation or by voluntary effort, the foot may remain in a state of dorsal flexion long after the stimulus has ceased to act, and a constant current

passed through it does not produce relaxation. Distinct resistance is also offered to the passive production of plantar flexion. After a variable length of time the muscle relaxes, either gradually and continuously, or with several intermissions, and the foot falls by its own weight to the position of plantar flexion. The paradoxical contraction sometimes extends to the extensor communis digitorum and extensor brevis pollicis. In one case observed by Westphal, the biceps femoris was seen to contract on the leg being suddenly flexed on the thigh. This kind of contraction may be present when the tendon reflexes are absent or normal, and probably also when they are slightly exaggerated; but the presence of distinct ankle clonus will, of course, prevent the foot from becoming fixed. The phenomenon may also be observed when the cutaneous sensibility of the lower extremities is normal or lowered, and in the absence of any excess of the cutaneous reflex excitability. Paradoxical contraction is generally associated with paresis of the lower extremities, but a spastic rigidity of the muscles is never present, although a slight degree of resistance may be felt to passive movements of the leg and foot. This form of contraction may extend to the muscles of the upper extremities, and in a case observed by Westphal, in which some of them were affected, a certain amount of rigidity subsequently appeared in the muscles of both upper and lower extremities. It is a remarkable circumstance that the paradoxical contraction occurs in muscles like the tibialis anticus, which probably never contract when their tendons are struck; and, conversely, the paradoxical contraction has never been observed in muscles like the quadriceps femoris, which manifest readily the tendon-reflex contraction. Whether the paradoxical contraction is caused by reflex or direct excitation is not known. This phenomenon is sometimes a symptom of locomotor ataxia, but probably never of uncomplicated cases of the disease. Its presence may, perhaps, be regarded as a sign that the lesion in the posterior columns is extending to the lateral columns, and that the paralytic stage of the disease is approaching. This contraction has also been observed by Westphal in paralysis agitans, and in a case of hæmatomyelia at present under my care, in which both lower extremities are completely paralysed, paradoxical contraction is readily induced in the tibialis anticus of the right, but not in that of the left leg.

Paralysis of the Ocular Muscles.—One or more of the ocular muscles are not unfrequently paralysed in tabes dorsalis, and this symptom also is of great value because it may precede by many years the motor inco-ordination of the lower extremities. The motor oculi and abducens are more frequently affected than the trochlear nerve. The paralysis of the ocular nerves is usually transient, and generally lasts for a few days or months. The paralysis, however, often recurs after a longer or shorter time, and may become permanent towards

the later stages of the disease. The temporary paralyses of the ocular muscles in locomotor ataxy often do not give rise to any apparent squint, but cause double vision (diplopia), which is either constantly present, or only when the eyes are turned in particular directions. Distinct squint and ptosis are, however, present in some cases. Out of 64 cases recorded by Eulenburg, 25 had strabismus; of these 25, 19 had divergent strabismus, and 4 had in addition paralytic ptosis, while 6 had convergent strabismus. When paralysis of the third or sixth nerve occurs syphilis is generally suspected to be the cause, and if the paralysis disappear in a few days or weeks under treatment, the diagnosis seems to be placed beyond doubt. Paralysis of these nerves may, however, be the first symptom of locomotor ataxy. In a case of my own, paralysis of the sixth nerve appeared to have been promptly cured by iodide of potassium, and it was not until eighteen months afterwards that the ataxic symptoms declared themselves and the true nature of the case was made apparent.

Mydriasis.—Dilatation of the pupil was observed by Eulenburg in 9 out of 64 cases. In 3 the dilatation was double, in 4 single, and in 2 accompanied by myosis of the other eye. There is no defect of accommodation accompanying this condition; hence it would seem that the pupil is dilated not from paralysis of the third nerve, but from irritation of the cilio-spinal nerves. The pupil frequently dilates during severe paroxysms of lancinating pains (Charcot) and during gastralgic attacks (Grainger Stewart).

Myosis.—Eulenburg found contraction of the pupil in 28 out of 64 cases, 21 showing double and 7 single myosis. The two pupils are indeed seldom of the same size, and the degree of contraction varies greatly in different cases and in the same case at different times. Inequality of the pupils is common in the early stage of the disease; and on the side on which the contraction of the pupil is the more marked there may be redness of the cheek, congestion of the conjunctiva, and local elevation of temperature (Charcot). These symptoms indicate vaso-motor paralysis, and prove that the myosis is due to paralysis of the cilio-spinal nerves.

The Argyll-Robertson Symptom.—This symptom, as already

mentioned (§ 225, 11), consists in the absence of any contraction of the pupil on exposure of the eye to light, while contraction with the accommodation is normally retained. The symptom is generally, although not invariably, associated with myosis.

Nystagmus is, as Friedreich has shown, occasionally present in locomotor ataxy; although, contrary to what occurs in multiple sclerosis, it is an exceedingly rare symptom. The nystagmus only appears when attempts are made to fix the eye on an object. The movements of the nystagmus in tabes do not succeed one another with the same rapidity as the movements in ordinary nystagmus, dependent upon disease of the eye. The movements are, indeed, purely ataxic, and only occur on a voluntary effort at fixation being made. Ataxic nystagmus only occurs in certain cases which possess marked clinical peculiarities, and it is always a late symptom of the disease.

Atrophy of the optic nerve is a frequent and distressing complication of locomotor ataxy. The affection begins with slowly or rapidly advancing diminution in the acuteness of vision, which soon terminates in amaurosis. Colour blindness can usually be demonstrated prior to any limitation in the field of vision. The perception of green is first lost, then that of red, yellow, and blue in succession, although deviations from this order may occasionally occur (Erb). The pupils are usually contracted in such cases and they do not react to light. The blindness is caused by white atrophy of the optic nerve. The rapidity with which blindness supervenes is very variable. At times total blindness may supervene in a few weeks, while at other times years may elapse before the loss of sight is complete, and occasionally the affection may cease to progress after it has lasted for a comparatively long time. The disease may be limited to one eye, but usually both eyes are simultaneously attacked. Atrophy of the optic nerve appears in about thirty per cent of all cases of locomotor ataxy. It is frequently one of the initial symptoms of the disease, and sometimes even precedes the lancinating pains. The amaurosis has been known to have existed ten years before the other symptoms of tabes have made their appearance.

Disturbances of hearing are occasionally observed in tabes. The defect of hearing is sometimes a purely accidental circum-

stance, but at other times it probably depends upon atrophy of the auditory nerve, analogous to that of the optic nerve.

Disturbances of taste and smell have also occasionally been observed, but are of subordinate importance.

The trigeminus at times manifests signs of irritation, giving rise to pain and paræsthesiæ, or it may be either partially or completely paralysed, giving rise to a sense of numbness, or to anæsthesia. Disturbances of taste and smell are always associated with an abnormal condition of the trigeminus.

The facial nerve is very rarely implicated, but irregular twitchings of the facial muscles have occasionally been observed.

The hypoglossal nerve has been rarely affected. The pneumogastric and glosso-pharyngeal nerves are very seldom implicated, unless, indeed, the gastralgic troubles, which are so frequent and distressing in tabes, are the result of irritation of the pneumogastrics.

Psychical disturbances are but seldom observed. Westphal has, indeed, shown that the majority of patients who suffer from progressive paralysis of the insane appear to have degeneration of the spinal cord chiefly limited to the posterior columns. The symptoms of progressive paralysis of the insane may precede the tabes for many years, or may become associated with it after it has existed for many years, or may not arise until the terminal period of locomotor ataxy. In all these cases the tabes is only one of the manifestations of a more widely-diffused degenerative process, just as ataxia may be one of the symptoms of multiple sclerosis.

There are other symptoms of locomotor ataxy which do not form such prominent features of the disease as those already described, but are not on that account less remarkable or of less importance. These are what may be denominated *visceral symptoms.*

Affections of the Bladder and Rectum.—During the early stage of the disease the patient often suffers from frequent and painful micturition, along with neuralgic pains in the depth of the pelvis, in the perinæum, or the neck of the bladder. At a later stage of the disease signs of paresis of the bladder make their appearance, so that emptying the bladder takes a longer time, and there is some subsequent dribbling, or there may be a

moderate degree of incontinence; but complete paralysis of the bladder is exceedingly rare, and only occurs in the last stage of the disease. A certain degree of vesical catarrh may be present during the terminal stages of tabes, but it is seldom of a severe character.

Patients also complain during the early stage of tabes of very peculiar sensations in the rectum. These sensations are at times described by the patient as a feeling similar to that which might be produced by the forcible introduction into the anus and rectum of a long and voluminous body (Charcot). This sensation appears suddenly and soon disappears, and it is usually accompanied by a strong desire to evacuate the contents of the bowels, and occasionally an involuntary evacuation of fecal matters occurs. At a more advanced period of the disease anæsthesia of the anus may be present, so that the patients lose the feeling of approaching evacuations, and hence not unfrequently dirty themselves. True paralysis of the sphincter is rare and only occurs in the terminal period of the disease.

Disturbances of the Sexual Functions.—A certain amount of disturbance of the genital organs is rarely absent in tabes. In the early period of the disease symptoms of irritation are present. Trousseau observed in certain cases a singular aptitude for repeating the venereal act a great many times within a short period, and he mentions the case of a man who was able to have connection as many as eight or nine times in one night. In these cases the appearance of excessive virile power is already conjoined with symptoms indicative of weakness. Very often this excessive desire has been preceded by a certain amount of incontinence of urine and involuntary seminal emissions, and the erections are often imperfect and accompanied by premature ejaculation. Charcot and Bouchard have observed symptoms of genital irritation in women. As the ataxic symptoms manifest themselves weakness of the sexual functions set in, which soon develop into complete impotence, although some tabetic patients retain their sexual power undiminished for a very long time.

A permanent acceleration of the pulse has been mentioned amongst the symptoms of locomotor ataxy. The temperature of the body is often increased during the attacks of lightning pains.

Gastralgic attacks, described by Charcot under the name of

crises gastriques, are frequently observed in the early stage of tabes. I know a gentleman who suffered from these attacks, and lancinating pains, ten years before the appearance of the ataxic symptoms. The gastralgic attack generally begins suddenly during a paroxysm of the lancinating pains. The patient complains of pain, which starts from the groins and appears to pass up each side of the abdomen, so as to become fixed in the epigastric region. At the same time severe pains are felt situated between the shoulders, which radiate round the base of the thorax, under the form of lightning pains. As a rule, the action of the heart is violent, and accelerated during the attacks, which are generally accompanied by extremely painful and incessant vomiting. The food is rejected, then a quantity of watery mucus, which is at first colourless, but may ultimately become mixed with bile and blood. A profound malaise and vertigo are conjoined with the vomiting, while the lightning pains are unusually severe, so that the sufferings of the patient may become truly agonising. These attacks may last without respite for two or three days, and may recur every two weeks; but usually the interval between the attacks is not less than a month. During the interval the functions of the stomach are entirely unaffected. The gastralgic attacks may begin at an early stage of the disease, and may constitute for many years, along with the lightning pains, the only symptom of the approaching malady. These attacks do not, however, always disappear on the ataxic symptoms being established, but may continue to recur until the fatal termination of the disease.

Nephralgic attacks have recently been described by Raynaud which present symptoms almost entirely similar to renal colic, only that there is entire absence of calculi, gravel, or blood from the urine.

Bronchial attacks have been described by Féreol under the name of "*bronchial crises*," consisting of paroxysms of spasmodic cough, difficulty of breathing and swallowing. In cases described by Friedreich vertigo has been a very prominent symptom.

Vaso-motor Disturbances.—Vaso-motor disturbances are not prominent features of locomotor ataxy. Patients not unfrequently complain of cold feet; and sometimes there is a great

tendency to the formation of *cutis anserina*. The skin is at times mottled, and there may be increased or diminished cutaneous secretion. A curious case has recently been described by E. Remak, in which the ataxic symptoms began in the right upper extremity. The muscles of the forearm were somewhat wasted, but there was no distinct loss of motor power, yet the hand was rendered practically useless from the disorderly movements which occurred when any attempt was made to use it. Serious sensory disturbances were observed in the extremity affected with ataxia, and slight analgesia of the sole of the right foot. There were also slight swaying movements on closing the eyes, and the patellar-tendon reflexes were absent, but there were no lancinating pains and no ataxic or paralytic symptoms in the lower extremities. In addition to these symptoms, the patient suffered from unilateral hyperidrosis limited to the right side, redness and slight relative increase of temperature of the right half of the face and side of the head, and myosis of the right pupil, the latter becoming more marked with the increase of the secretion of sweat, and frequently disappearing altogether with complete rest. The secretion of sweat was increased on the right side when the patient took any acid substance into his mouth, and also by faradic excitation of the tongue and mucous membrane of the mouth, or of the skin of the cheek, and trunk of the facial nerve. The conjunctiva is said by Trousseau to be frequently congested in locomotor ataxia.

Trophic Disturbances.—The most common trophic affections are eruptions of the skin, such as herpes, lichen, or the formation of bullæ, like those of pemphigus. Bed-sores usually belong to the terminal period of the disease only. Dr. Buzzard reports a case of locomotor ataxy, in which an eruption of herpes occurred in the right gluteal region during every paroxysm of lightning pain. The patient stated that he must have suffered from fifty or sixty attacks of herpes during the four years previous to the report of his case. The nutrition of the muscles remain for a long time unaffected; and, indeed, the muscles of the lower extremities may undergo a certain amount of hypertrophy during the early part of the ataxic stage, owing to the excessive activity to which they are subjected. During the paralytic stage the muscles may waste rapidly, simply from

disuse, and not from any active atrophy. Occasionally, however, the disease becomes complicated at a comparatively early period with atrophy of certain muscles, such as those of the calves of the legs, or those of the thighs, ball of the thumb, or of one-half of the tongue; and in these cases the atrophy consists of active degeneration, and not merely of the passive degeneration which is caused by functional inactivity. The most remarkable trophic disturbances which occur in tabes are the affections of the joints, which have been described by Charcot under the name of *arthropathies des ataxiques.* It is possible to meet with joints deformed with rheumatic gout, and dry arthritis coincidently with tabes, but the arthropathies of locomotor ataxia develop themselves quite independently of any general affection. This affection always appears during the early stage of tabes, usually during the stage of the lightning pains, although many cases seem to contradict this rule. The joints of the upper extremities may, for instance, become affected at an advanced period of the disease. The disease has, however, only reached an advanced stage in the lower extremities; while the upper extremities are only just beginning to manifest the initial stage of the affection. The joint most frequently affected is the knee-joint; and then in a descending order of frequency, the shoulder, elbow, the hip-joint, and the wrist in succession.

Various luxations of the joints ensue, producing notable deformities.

In locomotor ataxy the bones also sometimes become abnormally friable, so that spontaneous fractures may occur, a change which is no doubt of similar origin to the joint affection.

The following case, reported by Dr. Dreschfeld, will illustrate the arthropathies of locomotor ataxy:—

B. W——, aged fifty, mechanic, married, with no history of either syphilis or alcoholism, had always enjoyed good health till fifteen years ago, when the first symptoms of locomotor ataxy came on. These symptoms consisted in the inability to walk in the dark, and in the presence of lightning-like pains in the legs, in consequence of which he was soon obliged to give up his work. Seven years ago the pains in the right thigh, without losing their lightning-like character, became suddenly much more persistent, and obliged the patient to take to his bed, and to remain in bed for a month, when they left him as suddenly as they came. On trying to get up he found now that his right leg was much shorter than the left,

and that there was a projection on the right hip, which occasionally would suddenly disappear with a peculiar noise. Any movement of the thigh or leg would, however, make this projection very soon reappear. Three years ago the left knee began to give way without any exacerbation of pain, or any sudden swelling of the joint, and very gradually assumed its present position, that of extreme backward dislocation. In consequence of these joint affections, walking, which was already difficult before, became only possible with the help of two sticks. The general health of the patient had remained very good throughout, his eyesight had been bad for some years, but he had never suffered from vomiting or any bladder troubles.

On admission patient looked well and healthy; the thighs were considerably wasted, but the rest of the body was not emaciated. The chest and abdominal organs were perfectly healthy. There was no affection of any of the cerebral nerves, except marked white atrophy of both of the discs. The pupils were contracted and reacted to accommodation, but not to light. The upper extremities were perfectly normal. The lower extremities showed the chief symptoms, and here, as regards (1) sensation, there were diminution of tactile sensibility, analgesia of certain spots, and retardation of sensibility. The sense for temperature and weight was normal; the muscular sense was considerably affected. The patellar-tendon reflex was quite absent. Both lower extremities were often the seat of the lightning pains. As regards (2) trophic changes, (*a*) the muscles of both thighs were flabby and atrophied, but especially the muscles of the left thigh. (*b*) There was marked dislocation backwards of the left thigh, so that when the patient stood the upper surface of the tibia could distinctly be felt under the skin. There was no atrophy of either of the articulating surfaces, nor any new deposit of bone round the joint as far as could be made out on manual examination. The head of the right femur was dislocated on to the dorsum of the ilium, and could be felt as a distinct round projection; it was freely movable and could easily be reduced, but very soon slipped out again from the acetabulum on to the dorsum. Owing to this dislocation the left knee was situated four inches lower than the right knee, which difference disappeared as soon as the reduction of the head of the femur was effected (Plate IV. 1, 2). The head of the dislocated femur did not seem to be atrophied, nor were there any bone deposits to be felt about the joint cavity. A mass of bone, however of more than one inch in length was felt, situated in the sheath of the sartorius muscle, totally unconnected with the joint, but moving freely with the muscles during this contraction. As regards (3) motility, there was considerable diminution of motor power in both legs, but more in the right than in the left; the patient was, however, able to stand and to walk with the help of two sticks; his walk was characteristically ataxic; he was unable to walk with his eyes shut, and with his eyes open his gait was very unsteady, owing to the ataxy and the dislocations.

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§ 443. *Varieties.*—The largest number of cases of locomotor ataxy follow a very uniform course, which may therefore be regarded as forming the type of the disease. The symptoms, however, may deviate considerably from their usual course, or the affection may be observed in combination with other allied forms of disease. Locomotor ataxia may, therefore, be divided into the following varieties (Erb) :—

(1) *The Regular Typical Form of Tabes.*—It usually attacks men during middle age. It begins with lancinating pains, disturbances of the nerves supplying the ocular muscles and of the optic nerves, disturbances of sensibility of the extremities, such as paræsthesia, slight anæsthesia, feeling of a tight girdle, great feeling of fatigue, motor disturbances in the lower extremities, such as swaying on closing the eyes, and disturbances of the urinary and genital organs.

During the second stage of the disease the symptoms of the first stage gradually increase, and now become associated with characteristic ataxia. Patients become more and more helpless, there are well marked disturbances of sensibility, such as partial anæsthesia, retardation of the perception of pain, abolition of tendon-reflex along with increase of vesical and sexual weakness.

In the third or terminal stage true paralysis gradually supervenes in the form of paraplegia; there are also contractures, muscular atrophy, bed-sores, and progressive cachexia.

(2) *The Form of Tabes described by Friedreich.*—Family predisposition forms a prominent feature of the disease, and the female members of these families manifest a tendency to be attacked by preference. The disease generally begins between the age of twelve and eighteen years. Lancinating pains are rare at the beginning, but, on the other hand, the ataxia begins early, and rapidly spreads from the lower to the upper extremities, and sometimes even arises simultaneously in the upper and lower extremities. Co-ordinating disturbances of speech and ataxic nystagmus are generally present. Disturbances of sensibility are absent or insignificant; the sensibility of the skin and muscles remains unaffected for many years. No swaying on closure of the eyes. Tendon-reflex abolished. No disturbances of the bladder or bed-sores, and no psychical disturbances, tremor, or amaurosis. Muscular atrophy, paresis, contractures, weakness of the bladder appear only in the last stage. The affection is usually of remarkably long duration, extending sometimes over thirty-two years.

(3) *Anomalous Forms.*—Sometimes the degenerative changes do not remain limited to the posterior columns, but extend to other segments of the cord, giving rise to a combination of the symptoms of locomotor ataxy with those of one or more of the other systematic diseases of the cord.

(4) *Paraplegic Form.*—In a certain number of cases manifestations of motor weakness come into prominence at an early period of the disease, so that the tabetic symptoms become obscured. The symptoms may in such cases suggest paraplegia, and emaciation and atrophy of the legs may seem to confirm this opinion. In these cases the degenerative changes have no doubt extended to the pyramidal fibres of the lateral columns and to the anterior horns of the grey substance.

(5) *Neuralgic Form.*—In other cases the lancinating pains constitute the most prominent, and for a long time, sometimes upwards of twenty years, the only symptoms of the disease. This form has been called "tabes dolorosa."

(6) *Meningitic Form.*—At times locomotor ataxy becomes complicated with spinal meningitis, and such cases may offer a very variable combination of symptoms. Circumscribed or diffused cutaneous hyperæsthesia, pain in the back, and spinal tenderness are some of the symptoms which are most commonly present. Locomotor ataxy may also be complicated with various psychical disturbances, and these must be subjected to a special investigation.

§ 444. *Course, Duration, and Termination.*—The usual development of the locomotor ataxia is slow and chronic, extending over months or years, although cases have been described under the name of *acute ataxia* which run a rapid course. As a rule, single symptoms arise which remain isolated for a long time, and with which others become after a time associated until in the course of months or years the picture of the disease is complete. The ataxic symptoms, as already mentioned, usually begin in the lower extremities, but a few cases are reported in which the upper extremities were first affected. Occasionally a unilateral development has been observed.

When the disease is fully developed the intensity of the symptoms progressively increases, new symptoms arise, and the condition of the patient gradually grows worse. The disease may, however, remain stationary, or even undergo a marked improvement for months or years; but after a time an unfavourable change usually takes place. The patients feel better in summer and worse in winter, but they generally lose more in cold than they gain in warm weather. In rare cases the improvement may progress to complete recovery. The duration of the disease is always to be counted by years, and sometimes by decades. Even the initial stage, with lancinating pains, may

last over twenty years. In the majority of typical cases of the disease the average duration of life appears to be from eight to twelve years, and in these cases death is caused by bed-sores, cystitis, or bulbar symptoms, or the spinal affection renders the patient less capable of surviving intercurrent attacks of disease, such as pneumonia, the exanthemata, or other fevers.

Recovery is not unusual in the initial stage under appropriate treatment; and even when the disease is fully developed, recovery, or an improvement bordering on recovery, may take place. Often, however, patients must be content with a moderate improvement, or an arrest of the malady. As a rule, the disease is of a progressive character, and the most judicious treatment may fail to bring about even a temporary improvement. A fatal termination may be brought about in various ways. The disease may lead to paraplegia, cystitis, and bed-sores, and the patient dies from the usual symptoms of severe spinal paralysis. During the last few days cerebral symptoms, as coma and delirium, may supervene. The morbid process may, in progressing from below upwards, involve the medulla oblongata, and cause death by interfering with respiration or with the act of deglutition. Very frequently some intercurrent affection, such as typhus, pneumonia, diphtheria, and phthisis, causes a fatal termination.

§ 445. *Morbid Anatomy.*—The spinal pia mater is often thickened, cloudy, and connected by numerous adhesions to the dura mater. The change in the pia mater is generally confined to the posterior aspect of the cord, being circumscribed by the posterior roots on each side. Occasionally, however, the pia mater appears altered over a larger area, and the spinal fluid is almost always increased in quantity. The spinal cord is generally altered in form, being flattened from before backwards over a considerable portion of its extent, caused by a diminution of the volume of the posterior column. On making transverse sections at different levels of the cord, a grey or greyish-yellow discolouration may be observed along the posterior median fissure, extending almost the entire length of the cord. The consistence of the cord is usually increased, but occasionally it may be diminished.

The posterior roots are discoloured, grey, translucent, and atrophied, this condition being particularly well marked in the cauda equina. Dr. Carter, of Liverpool, exhibited the spinal cord from a case of locomotor ataxy at a recent medical meeting in Manchester, in which the ganglia of the posterior roots of the sacral and lumbar nerves were greatly enlarged.

The degeneration is not, as a rule, uniformly distributed over the whole transverse section of the posterior columns. The columns of Goll are usually affected over their entire length, and the posterior root-zones are always affected to a more or less extent, although they are not often degenerated throughout the entire length of the cord. In the inferior portion of lumbar enlargement there is frequently only a slight grey discolouration in the external half of the posterior columns; but on ascending it increases in width, so that in the upper half of the lumbar enlargement the discolouration embraces the entire transverse section of the posterior columns. The whole of the posterior columns are usually affected throughout the entire length of the dorsal region, but its extent diminishes again in the cervical portion, and the degeneration becomes limited in the upper cervical region to the columns of Goll. As a rule, the intensity and extent of the morbid process is greatest in the upper lumbar and the dorsal portions, diminishing both upwards and downwards from these points.

The morbid changes may extend upwards into the medulla oblongata, along the ascending root of the trigeminus (Pierret). The posterior horns of grey matter often appear of a dark grey colour, shrivelled, and distorted, and the vesicular columns of Clarke have been found altered. The discolouration may also extend forwards from the posterior horns to the direct cerebellar fibres and the pyramidal fibres of the lateral column. Such, then, are the morbid appearances which are found in fully-developed cases of the disease, but in cases which have died from an intercurrent affection during the early stages of locomotor ataxia the morbid appearances met with are somewhat different. Charcot and Pierret have shown that, although the columns of Goll are usually implicated in locomotor ataxy, the ataxic symptoms may be present in a high degree in the entire absence of any affection of these columns. Sclerosis

of the posterior root-zones is, according to these authors, the essential morbid alteration of locomotor ataxia, and even the whole width of these zones need not be implicated. A certain amount of alteration of these zones may be detected by microscopic examination, if not by the naked eye, even in those cases that have died by an intercurrent disease during the stage of the lancinating pains before the ataxic symptoms had made their appearance. And on the other hand, in a woman in whom

FIG. 176.

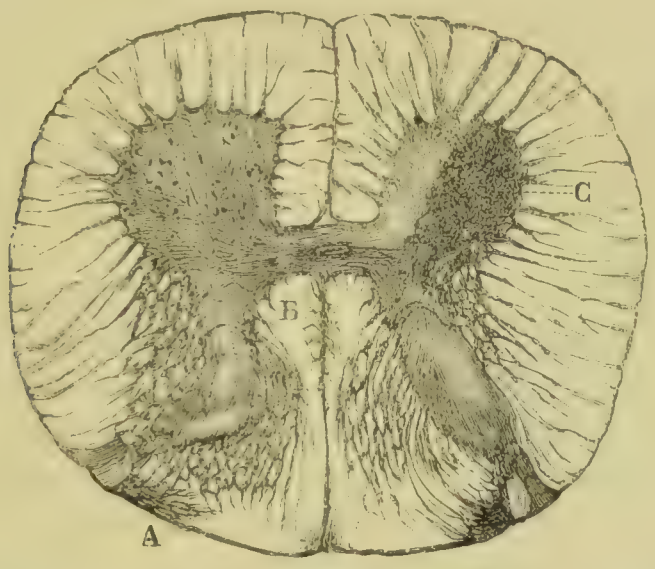


FIG. 176 (Charcot and Pierret). *Transverse Section of the lower portion of the Lumbar Enlargement, from a case of Locomotor Ataxia.*—A, Posterior roots; B, Internal radicular fasciculus, the sclerosis being limited to its course; C, Right anterior grey horn in a state of atrophy.

the disease appeared in a generalised form, the superior, as well as the inferior, extremities being the subject of the lightning pains and motor inco-ordination, the posterior root-zones were found affected the whole length of the cord, while there was complete absence of any affection of the columns of Goll (*Fig.* 176, B).

In very old and protracted cases long portions of the spinal cord appear hardened and atrophied in its entire thickness. On making a transverse section, the whole is found transformed into a grey translucent mass, in which it is difficult to recognise even the distinction between grey and white matter.

The microscopical changes in the cord consist in the early stage of thickening of the interstitial tissue, increase in the number

of nuclei along with the formation of enlarged and highly-developed Deiter's cells.

The nerve fibres dwindle gradually and ultimately disappear. The medullary sheath does not usually undergo fatty degeneration or break down, and no swelling of the axis cylinder is observed; there is simple atrophy and disappearance of the nerve fibres, and numerous granule cells are found. In recent cases the vessels are generally thickened, the nuclei are increased in number, and corpora amylacea are scattered throughout the tissue in greater or lesser number. In the later stages the principal mass of the structure is composed of a firm fibrillar, often wavy, connective tissue, which contains numerous nuclei and is disseminated with innumerable corpora amylacea. Most of the nerve fibres have disappeared, but even in advanced cases some well-preserved but isolated fibres may still be seen scattered through the firm connective tissue.

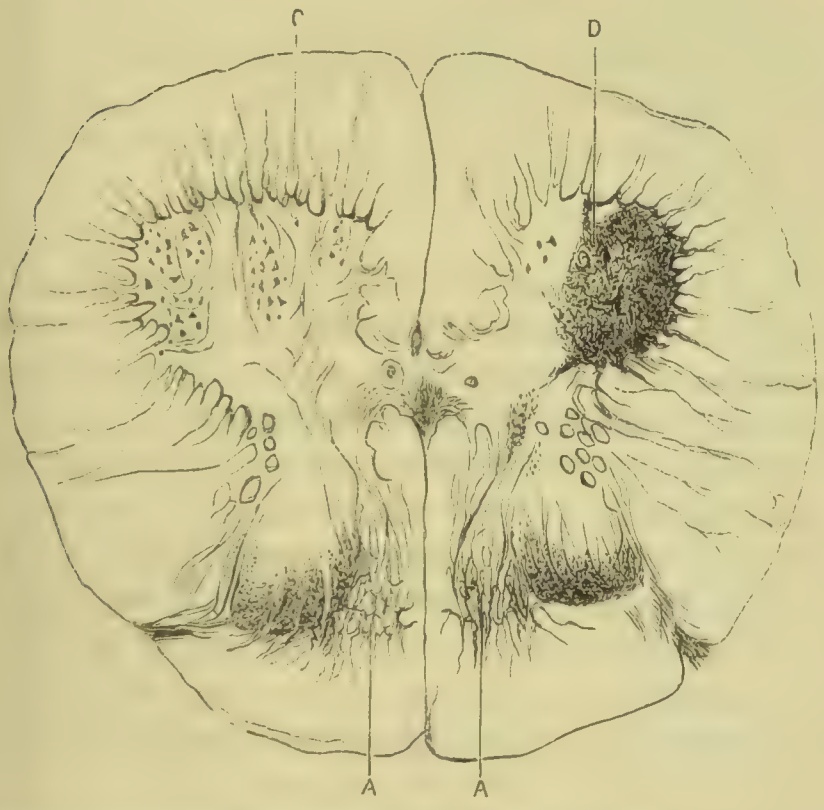
The posterior roots, in their passage through the posterior root-zones to the posterior grey horns—the inner radicular fasciculus—are involved in the degenerative process. Their fibres are broken down and atrophied, some are completely destroyed, while the remains of those left are separated from one another by broad bands of connective tissue.

The posterior horns of grey matter are also implicated in the degeneration. There is thickening of the connective tissue, disappearance of nerve fibres, and the ganglion cells are pigmented but not much changed in other respects. Clarke's columns are also frequently implicated, although their ganglion cells remain tolerably intact.

Sometimes the degenerative process extends to the anterior horns of grey matter, injuring the large ganglion cells, and then the muscles innervated from the diseased grey matter are always in a state of atrophy. This alteration stands, according to Pierret, in connection with sclerosis of the inner radicular fasciculus, and extends from those along the bundles of fibres that radiate into the anterior grey horns. In the annexed diagram the ganglion cells of the right anterior horn (*Fig.* 177, D) are in great part destroyed, and the muscles of the upper and lower extremities of the same side were atrophied.

Most recent observers are of opinion that the degenerative rocess begins within the cord itself, and not in the posterior ots. Leyden thinks that the process from beginning to end nsists of a degeneration; while Charcot, Friedreich, and others ok upon the degenerative changes as the result of chronic flammation. All, however, are agreed that the process egins in the nerve elements themselves, and extends from iem to the interstitial tissue. The disease may, however, egin at times in the pia mater, and spread thence to the osterior root-zones and columns of Goll.

Fig. 177.



. 177 (Charcot and Pierret). *Transverse Section of the Lumbar Region, from a case of Locomotor Ataxia, complicated with Muscular Atrophy.*—A, A, Sclerosis of the posterior root-zone; C, Left anterior grey horn, healthy; D, Right anterior grey horn in a state of atrophy.

The posterior nerve roots are atrophied in the late stages of e disease. They appear as flat, grey, translucent bands, and hibit degenerative atrophy of the nerve fibres and prolifera- n of connective tissue.

The peripheral nerves, the anterior nerve roots, the sympa-

thetic system, and the muscles are generally quite normal. The spinal ganglia of the posterior roots have, however, been found diseased (Carter). Some of the cranial nerves have also been found diseased, grey degeneration of the optic nerves being the most frequent change observed. Morbid changes have been found on rare occasions in the oculo-motorius, abducens, and hypoglossus. The nuclei of these nerves, on the floor of the fourth ventricle, also appear sometimes to be affected.

When arthropathies form a part of the disease there is disappearance of the articular cartilages, and the articular ends of some of the bones are eroded. There is little or no tendency to exostosis. In more recent cases the amount of articular fluid is greatly increased, points of thickening and fungosities are found on the synovial membrane, the surrounding soft parts are swelled and suffused with fluid.

The changes in the skin and viscera are the same as in chronic myelitis.

§ 446. *Morbid Physiology.*—The general opinion amongst pathologists at present is that sclerosis of the posterior root-zones of the spinal cord for a considerable portion of their longitudinal extent is the essential morbid alteration in locomotor ataxia. As the disease extends horizontally towards the posterior median fissure, the columns of Goll become implicated, and when once the fibres of these columns become interrupted in any part of their course the portions above the seat of lesion undergo degeneration, so that sclerosis of the columns of Goll throughout their entire length is usually present, although it does not appear to constitute a necessary part of the morbid change. As the sclerosis spreads outwards, the inner radicular fasciculi and posterior grey horns become occasionally affected; and in many cases, as already remarked, the disease extends to the anterior grey horns and lateral columns.

The lancinating pains may be explained by irritation of the posterior nerve roots, and their prolongations through the posterior columns, while the subsequent anæsthesia is caused by destruction of the posterior root fibres. The absence of the patellar-tendon reflex is caused by disease of the afferent portion of the reflex arc in its passage through the poster

columns. It is probable that irritation of these fibres may in the early stage of this disease give rise to excess of the tendon reflex.

Retardation of painful impressions and analgesia are caused by disease of the grey substance of the posterior horns. Irritation of the grey substance of the posterior horns occasions the cutaneous trophic disturbances. When the morbid change extends to the ganglion cells of the anterior horns, atrophy of the muscles supplied from the diseased part results, and it is also probable that the arthropathies of locomotor ataxia are caused by disease of the ganglion cells of the anterior horns. Disease of the automatic centres in the lumbar region occasions the vesical and sexual disturbances. When the pyramidal tract becomes implicated in the morbid change the paralytic stage of the affection becomes established. Implication of the direct cerebellar tract is not known to produce any symptoms.

It remains now to connect the swaying movements on closing the eyes and the ataxia with the morbid changes in the cord. Are we to connect the motor disturbances in locomotor ataxy with disease of the posterior root-zones themselves, or with disease of the fibres of the posterior roots and of the posterior grey horns with their consequent sensory and reflex disturbances? Vierordt and Heyd have shown that when the soles of the feet in healthy persons are rendered anæsthetic by chloroform or ice, the amplitude of the oscillations of the body is increased. This shows that loss of cutaneous sensibility must exercise some influence in the production of the motor disturbances of locomotor ataxia, and this influence becomes still greater when, as frequently happens, the sensibility of the muscles and articulations is lost.

But there is no constant relation between the degree of ataxia and that of cutaneous and muscular anæsthesia. Numerous cases are recorded in which a high degree of ataxia was present in the absence of any disorder of cutaneous or muscular sensibility, and when both symptoms are present they do not pursue a parallel course. It must, therefore, be concluded that the ataxia is not caused by disease of the fibres of the posterior roots of the posterior grey horn, and that it is caused by disease of the posterior root-zones themselves. These zones, as already

mentioned, consist of looped fibres which co-ordinate afferent impulses before they are transmitted upwards to the cephalic ganglia. But the motor disturbances of locomotor ataxia do not, as we have just seen, result from arrest of cerebro-afferent impulses (anæsthesia), and it may therefore be concluded that it is caused by disease of cerebello-afferent fibres.

§ 447. *Diagnosis.*—Typical cases of locomotor ataxy are easy to recognise; but in those cases in which the morbid process extends beyond its usual limits, the diagnosis is surrounded by many difficulties, and it can only be made by one who has a clear and distinct knowledge of the history of the case and of the symptoms which implication of each segment of the cord occasions. It is also very difficult to diagnose tabes at its commencement, and yet it is of great importance not to overlook the true nature of the case in the initial stage. The most trustworthy symptoms are the lancinating pains, the feeling of a tight girdle, paralysis of the ocular muscles, myosis with the Argyll-Robertson symptom, amaurosis with white atrophy of the disc, paræsthesiæ in the region of the ulnar nerve, great sense of fatigue on slight exertion, slight swaying of the body on the eyes being closed, failure of the patellar-tendon reflex, slight weakness of the bladder, and disturbances of the sexual organs.

The following are the chronic spinal affections which are most likely to be mistaken for tabes:—

Common transverse myelitis, as a rule, presents no difficulties. Paralysis of all the spinal functions, both motor and sensory, characterises this affection, and there are no lancinating pains in the initial stage.

Multiple sclerosis may sometimes be very similar in its symptoms to locomotor ataxy. The following symptoms may be regarded as significant of multiple sclerosis: Great dizziness, headache, psychical disturbances, early nystagmus, the characteristic tremor on voluntary effort, paralysis, muscular tensions, contractures, increased reflex actions of tendons in the lower extremities, and apoplectiform and epileptoid attacks.

Spasmodic spinal paralysis (lateral sclerosis) is characterised by paresis and paralysis with muscular tension and contractures

reatly increased reflex action of the tendons, absence of ataxy, nd of sensory and vesical disturbances, lancinating pains, and ffections of the cranial nerves.

Affections of the cerebellum may sometimes give rise to ymptoms closely simulating locomotor ataxy. The signs of erebellar disease are severe headache especially in the back of ne head, occasional vomiting, general convulsions, staggering ait like that of a drunken man or an impulse to fall back- ards, and amaurosis, while there is an absence of lancinating ains or other disturbances of sensibility, and of the vesical nd sexual functions.

Chronic spinal meningitis is sometimes complicated with abes. The symptoms of meningitis are pains and stiffness in he back, sensitiveness in the spinous processes to pressure, a ertain amount of sensory and motor weakness, and the absence f ataxy.

Progressive cerebral paralysis is distinguished from tabes by ne presence of disorders of speech and psychical disturbances.

§ 448. *Prognosis.* — Locomotor ataxy is a serious disease, lthough the prognosis is not always so serious as it was once hought to be. A certain small per centage end in recovery, nd in many instances the disease may be arrested for years. ts duration is, as a rule, always prolonged, and usually many ears elapse before the fatal termination.

The prognosis is favourably influenced by the absence of any ereditary predisposition to the disease, or of a neuropathic onstitution, by a slow development and moderate intensity of ne symptoms, especially of the sensory disturbances, by the atient being in a comfortable position in life, and by the vourable effect of treatment. If, on the other hand, there be pronounced hereditary predisposition, if the symptoms make pid progress, and there be severe paroxysms of pain, rapid maciation, serious implication of the genito-urinary organs, rthropathies or other trophic disturbances, and implication of he cranial nerves, the prognosis is unfavourable.

The prognosis of individual symptoms is very various. he disturbances of sensibility usually readily improve, but he prognosis of the ataxy is unfavourable. The lancinating

pains and the sexual weakness are usually very obstinate, but the vesical troubles may disappear. The prognosis in paralysis of the muscles of the eye is favourable, but the amaurosis, due to atrophy of the optic nerve, is quite hopeless.

§ 449. *Treatment.*—When there is a manifest predisposition to locomotor ataxy, the members of the family should be subjected to a careful regimen with the view of preventing the development of the disease. Members of such families should be cautioned against exposing themselves to the exciting causes of the disease, such as cold and damp, venereal excesses, and onanism.

Antiphlogistic treatment may be useful in the very early stage of those cases which are complicated by spinal meningitis, but it does no good in any other case. Counter-irritants have been greatly employed in the treatment of tabes, but they have never been found to be of any use, except probably in those cases which were complicated by spinal meningitis. Thermal baths have been much used at one time, but they are probably injurious instead of being beneficial, except in those cases which are attended by lancinating pains of unusual severity, and in which there are general excitability, sleeplessness, and other symptoms of irritation. The temperature should never be above 90° F., and the patient should not remain in the bath longer than from fifteen to twenty minutes, and it should be used only once in two or three days (Erb).

Sulphur baths have been much used in France, and the effects obtained from them have been favourably reported on. They have only, however, been used as adjuncts along with other agents, so it is impossible to tell how much of the effect is to be attributed to them.

Saline thermal baths appear to act favourably on the disease Rheims has long enjoyed a reputation in the treatment of tabes and Erb reports favourably of Nanheim. Chalybeate and mu baths have been employed in the treatment of the disease, bu it is doubtful if they possess any special advantages.

The cold-water cure, in well-conducted hydropathic establish ments, is probably one of the very best methods of treating th disease. Almost all authorities on nervous diseases, with th

exception of Leyden, speak most favourably of the beneficial action of hydro-therapeutics, and Erb recommends his patients to use the cold sponge bath at home all winter. The wet pack should be used with caution, beginning with a temperature of 88° F. and going down to 77° F. Wet rubbings, beginning at a temperature of 77° F. and going down to 65° F. do good. The institutions to which patients affected with tabes are sent should be situated in mountainous regions and provided with convenient walks. Patients should be sent there in the beginning of summer with instructions to remain till autumn.

Electro-therapeutics unquestionably exercise a favourable influence on the progress of the disease. The constant current is usually employed. Both electrodes should be placed on the vertebral column—one in the lumbar region, and the other at the nucha. The direction of the current appears to be an indifferent matter, but an ascending current is generally preferred. One pole, say the lower, should be fixed, and the other slowly moved along the back so as to come in contact with every part of the vertebral column; then the upper pole should be fixed, while the lower one should be slowly moved along the back. Peripheral galvanisation of the nerves of the lower extremities by cathode labile currents may be combined with the galvanic treatment of the spine. In irritable persons with severe pains the currents employed should be very weak, and strong currents should be avoided on all occasions. The galvanic current should be employed daily, but each sitting should not be for more than from three to six minutes' duration. Galvanic treatment should be discontinued if the patients feel fatigued after each sitting, if the pains increase, and if the condition of the patient becomes gradually worse.

Of internal remedies the *nitrate of silver* has acquired the greatest reputation in the treatment of tabes, and most authors are agreed that it frequently produces a beneficial effect. Friedreich has recently drawn attention to the fact that its long-continued use may induce albuminuria, but moderate doses may be taken for a considerable time without producing any deleterious effect. It should be given in doses of one-sixth to one-third of a grain three times a day. It may be continued for three weeks at a time, and then after an interruption for two

weeks its use may be resumed for another three weeks. The use of the medicine may be continued in this way with repeated interruptions for many months without producing any deleterious effects. If the skin shows the slightest discolouration, the medicine must, of course, be interrupted for a longer period.

The *iodide of potassium* given in large doses has sometimes been found of benefit, especially in those cases which are complicated with meningitis. The bromide of potassium in large doses is also said to be of use in mitigating the pains.

Belladonna and *ergot of rye* have been tried in tabes, but neither of them appear to have been productive of good; and the same may be said of arsenic, chloride of gold and sodium, and chloride of barium. Strychnia should on no account be administered, as it always appears to do harm. Phosphorus has been found to have a beneficial effect occasionally, especially in cases where the sensibility is greatly affected.

Cod-liver oil is strongly recommended by many authors, and in many cases its employment is attended with great benefit.

Esmarch made a communication of great importance at a recent surgical congress at Berlin. In a case which was diagnosed by Professor Quinke as locomotor ataxia, and in which the lancinating pains attained their greatest severity in the forearm, Credé, of Dresden, stretched the nerves in the axilla, and the pains and ataxic symptoms disappeared.

The diet and method of life should be carefully regulated in tabes. Patients should have as much fresh air as possible, especially mountain and sea air, and that of forests.

Patients should be particularly careful not to over-exert themselves, although a certain amount of graduated muscular exercise has a beneficial effect. Patients must of course be carefully guarded against exposure to cold and damp; and should, therefore, be particular to wear warm clothing and flannel next to the skin. In old cases patients should be discouraged from all useless attempts at curative treatment. The existence of the patient must be rendered as pleasant as possible, and the treatment may be directed to sustain the general health, but it is quite useless to adopt any special treatment.

In such a long and tedious illness symptomatic treatment will have to be adopted in the course of the disease. For the

ancinating pains a large number of remedies have been em-loyed at various times. Amongst these may be mentioned ynapisms, blisters, warmth, Priessnitz's cold-water compresses, elladonna plasters, rubbing with chloroform, faradisation or alvanisation (stabile anode) of the hyperæsthetic spots, sub-utaneous injections of morphia, large doses of the bromide of otassium, of the hydro-bromide of quinine, and, when there is neningitis, large doses of the iodide of potassium.

Electricity is the only remedy for cutaneous anæsthesia, notor weakness, and atrophy of the muscles.

In vesical weakness faradisation of the bladder, either vith or without the aid of the bladder electrode, is useful. Cystitis must be treated in the same way as chronic myelitis. For the amaurosis no treatment appears to be of any avail.

Constipation is sometimes a very troublesome symptom. The diet should be carefully regulated, with the view of acting upon the bowels, and enemata may be employed as aids to reatment. If necessary, mild aperients may be used; but all purgatives should, as far as possible, be avoided.

In obstinate cases faradisation of the bowels may be of great se.

2. *Sclerosis of the Columns of Goll.*

(*a*) Primary Sclerosis of the Columns of Goll.

§ 450. The most notable example of primary sclerosis of the columns of Goll is a case observed by Pierret. The following is a brief abstract of the symptoms recorded :—Margaret Magnaigat, et. 30 years, experienced numbness, formication, sensations of heat, and deep-seated pain in the limbs, more especially in the upper extremities. There were also obstinate headache, pains in the loins, and a sense of constriction of the thorax. In 1860 she did not feel the ground distinctly with her feet, and she was obliged to walk with a cane, and three years later she entered the Salpêtrière under the care of Charcot. Tactile sensibility was then diminished in the sole of the feet, which she detached with difficulty from the ground. These symptoms were especially marked in the left foot, and she could not walk without the use of a crutch under the right axilla. When she wished to advance she felt as if she were being drawn backwards, but once started

she was impelled forwards by a force she could not control. She could maintain the erect posture with closed eyes, but felt ready to fall at every instant. In 1866 she complained of girdle sensations, and lightning pains passing round the body and down the anterior part of the thighs, while she was readily fatigued, but the muscular sense was unaffected. She died in 1871 from an attack of pneumonia. At the autopsy Pierret found sclerosis of the columns of Goll, and he thinks that disease of these columns explains the tendency to propulsion and retropropulsion experienced by the patient as well as the uncertainty felt in maintaining the erect posture. The posterior root-zones were to some extent implicated in the lesion in the dorsal region, and I should say, from the careful drawings which accompany the case, in the lumbar region also. Implication of the posterior root-zones doubtless explains the lightning pains and other sensory disturbances present during the progress of the case. A case of primary sclerosis of the columns of Goll has been recorded by Ducastel and another by Gowers, but in neither were there symptoms during life which could with probability be attributed to disease of these columns.

(*b*) Secondary Sclerosis of the Columns of Goll.

§ 451. Secondary sclerosis of the columns of Goll occurs in connection with transverse myelitis, and it is then called ascending sclerosis (§ 390, *a*). It is also, as we have just seen, usually associated with sclerosis of the posterior root-zones in locomotor ataxia, and is often observed in many of the compound lesions of the cord. In none of these cases, however, has the affection of the columns of Goll ever been connected with any definite symptoms.

3. *Sclerosis of the Direct Cerebellar Tracts.*

§ 452. Sclerosis of the direct cerebellar tracts is, so far as is known, always secondary and ascending. It occurs in transverse myelitis along with sclerosis of the columns of Goll (§ 390, *a*). These tracts are also diseased in cases of meningomyelitis, or what is called cortical or ring-shaped sclerosis. Disease of these tracts has never been connected with any symptoms during life.

4. *Lateral Sclerosis.*

(*a*) Primary Lateral Sclerosis.

Tabes Dorsalis Spasmodica (Charcot). *Spasmodic Spinal Paralysis.*

§ 453. *Definition.* — The disease is characterised by a progressive paresis advancing gradually from below upwards, accompanied by muscular tension, contractures, and increase of the tendinous reflexes, along with entire absence of sensory and nutritive disturbances.

§ 454. *Etiology.*—No very decided hereditary tendency to the affection has as yet been made out. It appears to occur rather more frequently in males than females.

With respect to age, by far the largest number of cases begin between the ages of thirty and fifty. The disease is occasionally observed in childhood, a fact which might suggest the existence of a congenital defect of some parts of the spinal cord.

The exciting causes of the affection are unknown, although it is very probable that exposure to cold, injuries to the spine, lead poisoning, and syphilis may co-operate as factors in the production of the disease.

§ 455. *Symptoms.*—The first and for a long time the only symptom is a paresis of the inferior extremities, which may be equal in both or more pronounced in one of them, and the only effect of which is to render walking somewhat difficult, especially immediately on getting out of bed in the morning. The patients complain that they are soon fatigued, that their limbs are heavy, and their gait becomes dragging and difficult. It is only in the later stages of the affection that the paresis increases to complete paralysis. Manifestations of motor irritation now ally themselves with the motor weakness. On lying down and especially in bed at night, or after being fatigued, the legs become subject to clonic or tonic spasms. The former produce tremors, which sometimes remain limited to the extremities but are at other times so violent as to be communicated to the entire body. These may be readily excited by pushing against the toes so as to produce dorsal flexion of the foot (Ankle Clonus, § 80). After a time distinct *muscular tension* is developed. On passive movements of the lower extremities

the muscles become tense, but in the early stages of the affection the muscular tension can be readily overcome by increasing the pressure, while it can be considerably diminished by repeated movements. The muscular tension soon shows itself on voluntary movements being made, rendering them difficult and uncertain, and making the degree of paresis appear greater than it is in reality.

After a time the muscular tension increases to permanent rigidity, and a high degree of *contracture* results. The legs are maintained in a position of rigid extension, the thighs being also held rigidly together by contracture of the adductors, the feet are in a position of extreme talipes equino-varus, and the toes are generally strongly flexed. The rigid immobility of the feet is now and then interrupted by clonic trembling, which may extend to the entire leg. The trembling may appear to arise spontaneously, but is nearly always caused either by a reflex or voluntary movement of the foot.

The Spasmodic Gait or Spastic Walk.—The combined paresis, stiffness, and tremors of the lower extremities render the gait quite characteristic. The foot seems to cling to the ground, from which it is detached with difficulty, and as it is made to slide forwards it produces a characteristic scraping noise; while the toes find an obstacle in every elevation of the ground, and the patient readily stumbles and falls. Owing to the contracture of the extensors of the lower extremity the limbs are held in a rigid condition at all the articulations, so that the necessary elevation of the passive leg is obtained by an upward rotation of the pelvis, caused by contraction of the abductors of the thigh on the side of the active leg. The body is consequently strongly inclined at each step to the side of the active leg. The movement of the passive foot is not, however, directly forwards. The predominant contraction of the adductors of the thigh over the abductors causes the legs to be drawn energetically towards one another, while the foot is sometimes though not always inverted, owing to the strong contracture of the inward rotators of the thigh. The consequence is that the toe of the leg about to be moved forwards often gets entangled against the heel of the active leg, and the trunk has to be strongly inclined towards that side so as to give additional

urchase to the abductors of the opposite thigh, and thus nable them to move the foot outwards and away from the ther. The passive leg is, therefore, moved outwards and orwards in a semicircle, and when it is brought to the ground t generally crosses over to the opposite side in front of the ther foot. It will be observed that at each step the body is trongly inclined towards the side of the active leg, and consequently the gait is somewhat "waddling," and is in this espect, as Hammond remarks, like that of a woman with a vide pelvis..

At this period the weight of the body suffices to arrest the lonic contractions of the muscles of the calf of the active leg, ut the passive leg is often agitated by tremors, which greatly dd to the difficulty of progression. As the disease, however, ncreases, the spasmodic rigidity of the muscles of the calf beomes so great that the patient rests upon the tips of his feet; hile the body is inclined forwards, the arms being propped up y crutches, or supported by two sticks, which are held well in ront of the patient with an outward inclination. The contracure of the muscles of the calf is now so pronounced that the veight of the body does not suffice to prevent ankle clonus from aking place, and, consequently, when the patient first attains he erect posture, his heels become strongly elevated, probable o the extent of 6 inches from the ground. After the first levation the heel is in some cases almost immediately lowered o the extent of about 1 to 1½ inches, and this in its turn is ucceeded by another elevation and so on in rhythmical sequence. The heels and with them the whole body are thus elevated and lepressed 7 or 8 times or more in rapid succession, the number f these elevations which take place in a second of time correponding to those of the ankle clonus already described.

After a time the upward and downward movements of the ody cease, the heels come closer to the ground although hey do not come in contact with it, and the patient now ndeavours to move forwards one leg, say the right. The first tep may be performed with tolerable facility, but when once he right foot is projected forwards it crosses over to the other ide, and is brought to the ground in front of the left foot. When the left has now to be advanced the greatest difficulty is

experienced in disengaging the toe from the heel of the right foot, and in the effort to do so the muscles of the calves of both legs become strongly contracted, the patient is elevated on tip-toes, and every effort to abduct the left foot so as to move it away from the other may induce clonus of the right ankle, and consequent elevations and depressions of the body. When at last the left foot is disengaged, and is being moved forwards in the semicircular manner already described, it is generally seized with trembling (partly consisting of ankle clonus and partly of tendinous reflex contractions of the muscles), which extends to the trunk, and throws the whole body into violent agitation. This description only applies of course to the severer cases, and if the muscular contracture increases beyond this point walking becomes impossible. In less aggravated cases one sudden elevation of the heel of the active leg may be followed by a depression without subsequent elevation, so that the gait has a peculiar hopping character.

The disease extends slowly and gradually upwards until the superior extremities are implicated. The lumbar and abdominal muscles are also affected, the abdomen becomes prominent, hard, and separated from the base of the thorax by a horizontal fold of more or less depth, while at the same time a kind of lordosis is produced.

When the upper extremities are affected, the paretic condition of the hands manifests itself by the inaptitude of the patient to seize small objects. The digits from time to time become flexed involuntarily into the palm of the hand; while at a later period of the disease the fingers become permanently flexed. The muscles acting on the wrist and elbow are successively affected, and the forearm and hand become rigid in a condition of extension and pronation. The superior extremities are now rigid and immobile, and more or less strongly drawn to each side of the body, but the tremors are never so pronounced in them as in the lower extremities. Although this is the usual course of the disease, yet occasionally the symptoms are developed in a different order. At times the affection passes first from one lower extremity to the upper extremity of the same side, and this hemiplegic condition may persist for many years before the other lower extremity is

attacked. Sometimes the disease begins in the upper extremities, progresses downwards, and does not involve the lower extremities until a later period.

The paralysis after a time becomes complete, the contractures increase in intensity, the patients grow stiff and immovable, and are doomed to keep their beds. But even in patients who are bedridden for years the general health is good, and it does not appear that the disease ever directly causes death, which generally results from an intercurrent affection.

The tendinous and periosteal reflexes are greatly exaggerated in this disease. The patellar-tendon reflex and ankle clonus can be elicited in the usual way with undue readiness. The quadriceps femoris and the adductors of the thigh may be excited to contract by tapping the broad upper end of the tibia, and the contractions may extend even to the adductors of the opposite thigh. The adductors of the thigh may also often be made to contract by tapping over the region of the lumbar vertebræ. Tendon reflexes also occur in the tibialis posticus, semi-tendinosus, and other muscles.

The tendon reflexes are in like manner increased in the upper extremities when they become implicated. They can be elicited in the biceps and triceps by striking the tendons, while the former may be made to contract by tapping the lower end of the radius, and the latter by tapping the lower end of the ulna. The posterior portion of the deltoid often contracts along with the triceps, when the lower end of the ulna is lightly struck. The flexors of the fingers, the extensors of the wrist, and the supinator longus can each be made to contract by tapping their tendons at the wrist; while the interossei may sometimes be made to contract by tapping the ends of the metacarpal bones. The deltoid may be made to contract by tapping the spine of the scapula, and the pectoralis major by tapping the sternum.

The cutaneous reflex appears to be occasionally increased, but it is generally normal or diminished.

The electrical excitability of the motor nerves may manifest slight quantitative but never any qualitative changes. The faradic and galvanic excitability of the muscles is generally diminished (Erb).

Sensory disturbances are entirely absent in this disease,

the various forms of cutaneous and muscular sensibility being normal. The patient does not complain of paræsthesiæ, girdle sensations, lancinating pains, or the affections of the cranial nerves, which are so common in locomotor ataxia. The functions of the bladder, rectum, and sexual organs are entirely unaffected. Vaso-motor disturbances are absent, and there are no nutritive affections of the muscles or skin, and no bed-sores.

§ 456. *Course, Duration, and Terminations.*—The course of the disease is generally very chronic. It comes on in a very insidious manner, and months or years may elapse before the affection can be recognised with certainty. Occasionally the symptoms become developed in a typical manner in a comparatively brief space of time, and it may then remain stationary for a long period.

The duration of the disease is nearly always long, extending over many years. Complicated cases may run a comparatively rapid course.

The disease occasionally terminates in recovery. Heuck reports a case of spastic spinal paralysis which began suddenly with acute and violent pains in the back, and terminated, after a duration of five weeks, in complete recovery. Death generally occurs from accidental causes or intercurrent diseases.

(*b*) Compound Lateral Sclerosis.

(i.) amyotrophic lateral sclerosis.

Sclérose Latérale Amyotrophique (Charcot).

§ 457. *Symptoms.*—Spasmodic spinal paralysis may be complicated with every degree of progressive muscular atrophy; but the highest degree of this combination is presented by those cases which have been described by Charcot, under the name of *sclérose latérale amyotrophique.*

The disease generally begins, according to Charcot, in the upper extremities by motor weakness, accompanied by a rapid muscular wasting, which extends uniformly to all the muscles of the affected limbs. The symptoms of paresis may be preceded by formication and numbness in the upper extremities, and the atrophy is accompanied by fibrillary twitchings of the

ffected muscles. Muscular tension and contractures are soon uperadded to the paresis and atrophy, and the affected extremities are brought into permanently deformed positions. 'he arm is applied to the trunk, the forearm is semi-flexed and ronated, and it is not possible to supinate or extend it without mploying a considerable degree of force and causing pain, the and is flexed on the forearm, and the different segments of the ngers are flexed upon one another and upon the metacarpal

Fig. 178.

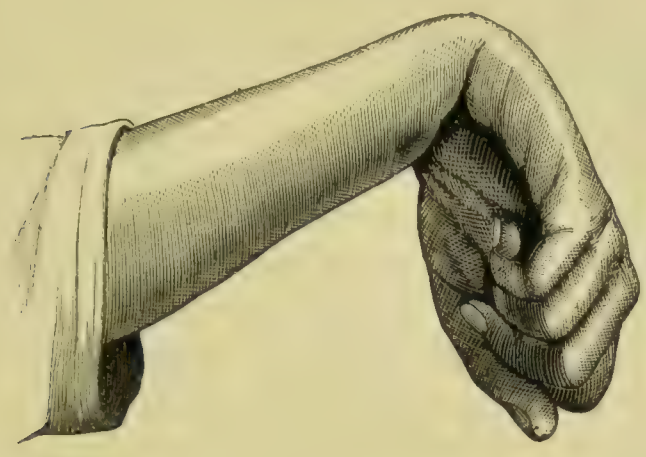


Fig. 178 (After Charcot). *Attitude of the Hand and Forearm in Amyotrophic Lateral Sclerosis.*

ones. When the patient elevates the arm by a voluntary ffort, the extremity becomes agitated by tremors, not unlike hose which occur in sclerosis in patches. In advanced cases he thenar and hypothenar eminences become flattened, the alm of the hand becomes excavated, and the muscular atrophy ay proceed so far that the forearm and arm are reduced almost o a skeleton. Under these circumstances the spasmodic rigidity ecomes less pronounced, but the limbs may even then maintain the forced attitudes in which they have been held so long.

In some patients the head is fixed by spasmodic rigidity of he muscles of the neck so that they cannot move it in any irection. The contracture may also extend to the temporal uscles so that the mouth can only be opened to a limited egree (Charcot). The muscular atrophy may occasionally be asked by a pseudo-hypertrophy of the affected muscles.

After a period of from two to six or nine months the lower extremities become affected, first by paresis, which may be preceded or accompanied for a longer or shorter time by formication and numbness of the limbs. The paresis of the lower extremities is not, as occurs in the upper extremities, necessarily accompanied by atrophy of the muscles. It is, however, accompanied by muscular tension, permanent contractures which maintain the extremities rigid in the position of extension, by tremors, ankle clonus, and increased reflex action of tendons. These symptoms are, indeed, those which have already been described as belonging to primary lateral sclerosis, and they soon increase to such a degree in the amyotrophic varieties as to render walking impossible. At first the muscles of the lower extremities are tense and firm, and do not show any trace of atrophy, but after a time fibrillary contractions occur, diffused atrophy of the muscles supervene, and the contractures diminish.

The third stage of the disease is characterised by the appearance of bulbar paralysis, consisting in paralysis of the tongue and lips, and of the pharyngeal and laryngeal muscles. The nuclei of the pneumogastric nerves appear finally to be invaded, giving rise to disturbances of circulation and respiration which before long induce death. The disease develops rapidly, and, according to Charcot, always causes death in from one to three years; differing in this respect greatly from progressive muscular atrophy, which may extend over a period of from eight to twenty years.

(ii.) COMBINED SCLEROSIS OF THE POSTERIOR AND LATERAL COLUMNS.

§ 458. The symptoms of locomotor ataxy and of primary lateral sclerosis may be present in every possible combination those of the former predominating at one time and of the latter at another. The symptoms which indicate that the lateral columns are being gradually invaded in locomotor ataxy are spontaneous jerkings in the lower extremities, gradual loss of power to perform simple movements of extension and flexion muscular tension, and contractures.

When, on the other hand, the symptoms of lateral sclerosis predominate, the signs by which a complication of locomot

axy may be suspected are the presence of lancinating pains, rdle pains, and other sensory disturbances, vesical weakness, ght swaying on closing the eyes, and, above all, the absence the patellar-tendon reflex and ankle clonus.

(c) Secondary Lateral Sclerosis.

§ 459. Lateral sclerosis occurs as a secondary disease in nsverse myelitis and in various diseases of the medulla longata, pons, and brain. It always occurs below the seat of e lesion, and pursues a *descending* course (§ 390, *b*). The mptoms caused by secondary lateral sclerosis are the same as ose caused by the primary form of the affection, being only odified by the symptoms of the primary lesion with which it associated. These symptoms are, briefly, voluntary paralysis, uscular tension and contracture, excess of the deep reflexes, d generally also of the cutaneous reflexes. The symptoms secondary degeneration of the pyramidal tracts will be more nutely described when the primary lesions with which it is sociated are under discussion.

§ 460. *Morbid Anatomy.*—The morbid anatomy of primary eral sclerosis has given rise to a good deal of discussion, and post-mortem examination of an uncomplicated case of the sease has hitherto been published. But what we know the functions of the lateral columns renders it probable at the anatomical basis of the affection consists of sym-etrical sclerosis of the lateral columns. Leyden has, however, vanced powerful arguments against this opinion. Two cases ve been published by Charcot and Pitres, one of which was gnosed during life as amyotrophic lateral sclerosis, and e other as primary lateral sclerosis, and the post-mortem amination showed that both were anomalous cases of sclerosis patches. The French authors, however, acknowledge that the case which was diagnosed during life as primary lateral erosis the symptoms were not quite characteristic. Symptoms, instance, of vesical weakness and slight sensory disturbances re present, and the authors think that had sufficient weight en given to the presence of these symptoms the diagnosis of

primary lateral sclerosis would not have been made. And, again, although a focus of disease was found in the posterior columns in the cervical region of the cord, the other foci were found in the pyramidal tracts in their passage through the crusta, anterior pyramids of the medulla, and lateral columns of the cord. Charcot, therefore, thinks that this case confirms to some extent the theory of symmetrical sclerosis of the lateral columns. Dr. Carl Ritter von Stofella has published a case in which the typical symptoms of spasmodic paralysis were present during life. The autopsy was conducted by Prof. Klob, who found symmetrical sclerosis of the posterior portion of the lateral columns. Prof. Klob, however, mentions that the sclerosis extended in the thoracic and lumbar regions to the pia mater, so that, as pointed out by Leyden, the direct cerebellar tract must have been affected, and the case cannot be quoted as an example of symmetrical sclerosis of the pyramidal tracts. No microscopic examination of the cord was made, and this fact of itself would render the case almost valueless with respect to the morbid anatomy of the affection.

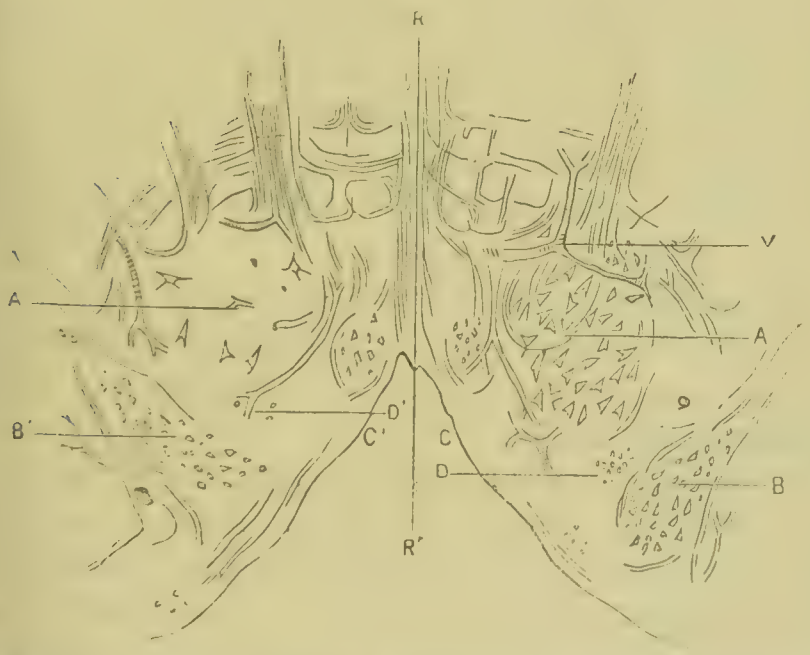
Dr. R. Schulz communicates three cases in which the symptoms of spasmodic paralysis were present during life, but in which the post-mortem examination decided against a primary sclerosis of the pyramidal tracts. In the first case a tumour o the medulla oblongata was found, accompanied by descendin sclerosis of the pyramidal tracts; in the second a tumour wa found between the right lobe of the cerebellum and pons, bu without a trace of descending sclerosis; while the third cas was one of chronic hydrocephalus internus, also without a trac of descending sclerosis of the cord.

A patient under the care of Dr. Morgan, in the Manchest Royal Infirmary, who presented the typical symptoms of prima lateral sclerosis, died from some intercurrent disease. T spinal cord having been hardened in bichromate of ammon symmetrical sclerosis of the pyramidal tracts of the late columns of the cord, from the medulla oblongata to the con medullaris, was found.

Dr. Dreschfeld, who made a microscopic examination of t cord, assures me that no other lesion exists in the co Aufrecht has recorded a somewhat similar case.

In the amyotrophic variety of the affection Charcot has veral times proved the presence of symmetrical sclerosis of e pyramidal tracts of the lateral columns of the cord, and of e anterior pyramids of the medulla oblongata, along with generative atrophy of the anterior grey horns and loss of the rge ganglion cells of the cord and of the motor nuclei in the edulla oblongata. The condition of the bulbar nuclei in this fection is represented in *Fig.* 179, borrowed from Charcot, the seased nuclei being shown to the left of a fictitious line (R, R′), id the healthy one, for the sake of comparison, to the right of iat line. The part which Charcot calls the fasciculus teres ally consists of a group of small cells, and is the same as that hich I have called the external accessory nucleus of the facial. is seen to be diseased on the left side (D′) of the figure. The ternal accessory facial nucleus is, however, apparently healthy. hese observations have also been confirmed by Joffroy, omboult, and others. Valuable contributions to the morbid

FIG. 179.



IG. 179 (From Charcot). *Transverse Section of the Medulla Oblongata on a level with the middle of the Nucleus of the Hypoglossal.*—R, R′, median raphé; A, B, represents the normal condition, and A′, B′ the parts as they appear in amyotrophic lateral sclerosis; C, C′, the floor of the fourth ventricle; V, a vessel which bounds the nucleus of the hypoglossal anteriorly and externally; D, fasciculus teres? and D′, the corresponding part on the diseased side; A, healthy nucleus of the hypoglossal; and A′, the diseased nucleus. B, the healthy nucleus of the pneumogastric nerves; and B′, the nucleus on the diseased side, which is seen not to be much affected.

anatomy of the cord, in cases where the symptoms of locomotor ataxy and of spasmodic paralysis were combined, have been made by Westphal and others.

§ 461. *Morbid Physiology.*—Little need be added to what has already been said with regard to the connection which exists between the morbid anatomy and the symptoms in this disease. The fibres of the pyramidal tract convey voluntary impulses from the cortex of the brain; and, consequently, when these fibres are diseased, and conduction through them is interrupted, voluntary paralysis must result. It is more difficult to explain why voluntary paralysis should be associated with muscular tension and contractures, but I agree with those pathologists who believe that these symptoms are caused by the unantagonised influence of the cerebellum on the paralysed muscles.

§ 462. *Diagnosis.*—Spasmodic spinal paralysis is liable to be mistaken for *transverse myelitis*, inasmuch as the lateral columns are generally implicated in the latter disease. In transverse myelitis there are various disturbances of sensibility, vesical weakness, and bed-sores, the paraplegia is developed more rapidly and completely, while the upper boundary of the paralytic manifestations remains stationary. Reflex action of the skin is, as a rule, considerably increased.

In the cases of *locomotor ataxy* which are complicated with sclerosis of the lateral columns, the diagnosis must largely depend upon the history of the case, especially with respect to the sensory disturbances.

This disease may be distinguished from *poliomyelitis anterior chronica* by the fact that in the latter the paralysed muscles become rapidly atrophied, the reflex action of the tendons and the faradic excitability of the muscles are lost, the muscles generally manifest the reaction of degeneration, and the deformities are of the nature of paralytic contractions.

When *multiple sclerosis* first makes its appearance in the lateral columns, it may be impossible for a time to distinguish between it and spasmodic spinal paralysis. As the form of disease progresses, its symptoms become so characteristic that the diagnosis between the two diseases is easy.

Spasmodic spinal paralysis may be distinguished from paralyses arising from lesions of the cauda equina and other peripheral paralyses by the absence in the former of disturbances of sensibility and muscular atrophy, the retention of electrical excitability, and the increase of the reflex action of the tendons.

The hemiplegic form of primary lateral sclerosis may be mistaken for hemiplegia of cerebral origin. In the former disease the lower extremity is first affected, and the paralysis only advances very slowly to the upper extremity, and muscular tension and contractures are early developed. There are no disturbances of sensibility and no symptoms referable to the cerebrum or to the cranial nerves. A focal lesion affecting the pyramidal tract symmetrically in the anterior pyramids of the medulla oblongata, followed by descending sclerosis, may cause symptoms which cannot perhaps be distinguished from those of primary lateral sclerosis.

§ 463. *Prognosis.*—Spasmodic spinal paralysis is probably the most protracted of all the chronic affections of the spine. Uncomplicated cases do not appear to shorten life at all, owing to the entire absence of disturbances of the bladder, bed-sores, and any affection of the respiratory centres. In some few cases the disease is capable of considerable improvement or even complete recovery.

Where the disease is complicated with muscular atrophy and bulbar symptoms, the prognosis is of course very grave.

§ 464. *Treatment.*—The treatment of spasmodic spinal paralysis is generally the same as that of chronic myelitis and of tabes. The galvanic current is by far the most trustworthy remedy. Carefully conducted hydropathic treatment may be beneficial, and gaseous thermal springs have been employed with apparent advantage. The nitrate of silver has been used internally, but it does not appear to be so useful in this affection as in tabes.

The rules with regard to diet, exercise, and general regimen are the same as for other forms of chronic myelitis.

CHAPTER V.

II.—MIXED DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

(I.) PARALYSIS ASCENDENS ACUTA.

Acute Ascending Paralysis—Landry's Paralysis.

§ 465. *Definition.*—Acute ascending paralysis is characterised by a motor paralysis which generally begins in the lower extremities, spreads pretty rapidly over the trunk to the upper extremities, and usually involves the medulla oblongata, the general sensibility and also slightly the functions of the bladder and rectum; but there is no decided atrophy of the muscles and no alteration of their electrical excitability.

§ 466. *History.*—Cases of this disease were described by Ollivier, Walford, and others, and it appears that Cuvier died of it in 1832. The disease, however, was not recognised as a separate affection until 1859, when Landry described some cases under the name of "Paralysie Ascendante Aigue." Kussmaul also described two cases in the same year. Since Landry's publication reports of cases have multiplied; although at times instances of other diseases, such as of acute central myelitis, and of subacute anterior poliomyelitis, have been described under this name.

§ 467. *Etiology.*—Very little is known with respect to the causation of this disease; and at times it arises in the absence of recognisable predisposing or exciting causes. Most of the reported cases have occurred between the ages of twenty and forty, and men are more frequently attacked than women.

Exposure to cold is probably the most frequent exciting cause. Some cases have occurred during convalescence from acute diseases, as typhoid fever, pleurisy, or variola, and a few have followed suppression of the menses. Various authors regard

philis as a frequent cause of the disease, but it is doubtful ow far this opinion is correct. In the case of a woman of enty years of age, who died of the disease in the Royal In- mary, within a week from the commencement, there were ep cicatrices in the left groin, but no further evidence of philis could be detected. The interior of the uterus was lined ith a layer of blood. I did not see the case during life, but e symptoms as reported to me were very characteristic.

§ 468. *Symptoms.*—The paralytic phenomena are generally, ough not always, preceded by various premonitory symptoms, ch as slight fever, shooting pains in the back and limbs, rmication and numbness in the feet and finger tips, and a eling of great weariness, debility, and general discomfort. hese may last for one or several days, and they have occa- onally existed for six weeks.

The characteristic symptoms of the disease now make their pearance. Great weakness of the lower extremities is soon mplained of, which increases to such an extent as to render anding and walking impossible. The patient can for a short ne execute when lying down the individual movements of the gs, but this power is soon lost. The paralysis appears first the muscles which move the feet, then in those which move e legs, and at last in the muscles of the thighs, and thus, in e course of a few days, the lower extremities may be com- etely paralysed. The legs now lie flaccid and powerless, there no resistance to passive movements of them, and there is mplete absence of muscular tension and contractures.

The paralysis advances steadily upwards, the muscles of the unk are invaded, and sitting up is rendered impossible; while e acts of coughing, sneezing, and defecation are weak and effective through paralysis of the abdominal muscles.

The muscles of the upper extremities are now attacked; ey are implicated, indeed, before the abdominal muscles, and on become completely paralysed. The hands first grow eak, and finer actions, as writing, become impossible. The ovements of the forearm become more and more difficult, and ose of the shoulder-joint are soon implicated, the arms, like e legs, being completely relaxed and immovable.

Disturbances of respiration now appear, owing to paralysis of the intercostal and other respiratory muscles of the trunk.

The disturbances of sensibility are quite subordinate to the motor paralysis, although they are not entirely wanting. Patients frequently complain of numbness and formication in the fingers and toes, a diminution of feeling in the soles of the feet, extending occasionally over the whole of the lower extremities, and pain may be complained of at the beginning of the disease, although it is never a prominent symptom. Cutaneous sensibility is usually normal, but occasionally it is distinctly lowered towards the periphery of the extremities, and in some few cases there is almost complete anæsthesia, while rarely hyperalgesia has been observed.

A considerable amount of emaciation may appear just as occurs during the course of any other acute disease, but the paralysed muscles do not undergo rapidly progressive atrophy, and the electrical excitability of the paralysed nerves and muscles remains normal. The nutrition of the skin is not affected, and bed-sores do not occur.

In a case reported by Eisenlohr transitory œdema of the skin with redness of the integument over various joints is mentioned as having been present, and in some other cases a profuse secretion of sweat has been noticed, but no other vasomotor disturbances.

Reflex action is preserved during the first few days of the disease, it then diminishes more or less rapidly, and is finally extinguished. In Eisenlohr's case an increase of reflex action was observed. In one case examined by Westphal the reflex excitability of the tendons was abolished as well as that of the skin.

The functions of the bladder and rectum are usually unaffected. In some few cases slight disturbances of the bladder have been met with, but the severe paralysis of the bladder and rectum, which occurs in other forms of central myelitis, has never been observed. The bowels are usually constipated.

The general health is as a rule good, and in the majority of cases there is no fever. In some, however, the general health is disturbed, and febrile symptoms appear, which occasionally may be severe. The brain is entirely unaffected throughout

ıe whole course of the disease; even the cerebral motor nerves re not implicated until the terminal period.

As the disease advances upwards, patients complain of pain nd stiffness in the neck, and the muscles of that region ecome paralysed, and sometimes there is paresis of the facial ıuscles. The medulla oblongata is soon implicated, and then he functions of articulation, mastication, deglutition, and ultiıately respiration are interfered with; evidences of hyperæmia nd hypostatic congestion of the lungs appear, and the patient ies from asphyxia. Sometimes the pupils have been unequal, nd the pulse may become very frequent.

The duration of the disease is somewhat variable. In some ases it runs its course and ends in death in two or three days, vhile occasionally it lasts from two to four weeks. The average luration of fatal cases is from eight to twelve days.

But the disease may end in recovery. It may cease to proress at any stage of its development. This usually takes place ›efore the nerves of the medulla oblongata are involved, but ecovery has been known to take place even after disturbances f respiration, deglutition, and mastication had commenced.

In cases which run a favourable course improvement takes ›lace at an early period of the disease, the parts last attacked ›y the paralysis being the first to show signs of improvement. The patients first begin to use their hands, after a time they ıre able to sit up, and finally after another considerable interval hey are able to stand and walk. The period of recovery occuies many weeks, although the duration varies much in indi-idual cases. Fluctuations and relapses may occur during ecovery, and the patients complain for a long time of debility. The disease occasionally begins in the bulbar nerves, and the paralysis progresses downwards within the cord. Cuvier, as reported by Pellegrini-Levi, died of this acute descending paralysis.

The following, according to Landry, is the order in which the muscles are affected by paralysis:—

1. The muscles which move the toes and foot, then the posterior muscles of the thigh and pelvis, and lastly the anterior and internal muscles of the thigh.

2. The muscles which move the fingers, those which move the hand, and the arm upon the scapula, and lastly the muscles which move the forearm upon the arm.
3. The muscles of the trunk.
4. The muscles of respiration, then those of the tongue, pharynx, and œsophagus.

It will thus be seen that although the paralysis pursues a general ascending course, yet the various groups of muscles are not affected in the same relative order in which they are innervated from the cord. The muscles of the hand, for instance, are paralysed before those of the abdomen, yet the former are innervated from the cervical and the latter from the dorsal region of the cord.

§ 469. *Diagnosis.*—It may not be possible to arrive at a positive diagnosis during the first days of the disease, but when it is fully developed the diagnosis presents no difficulty.

Acute anterior poliomyelitis may be distinguished from this affection by the circumstance that it has no progressive character, rarely attacks the medulla, and hardly ever directly causes death. It is also ushered in by fever, and there is rapid muscular atrophy and loss of faradic excitability. Even the temporary form of acute anterior myelitis may be distinguished from acute ascending paralysis by the loss of reflex excitability and lowering of faradic excitability, and by the fact that the paralysis is not progressive.

Subacute anterior poliomyelitis, when it pursues a tolerably rapid ascending course, may very readily be mistaken for acute ascending paralysis, but the latter disease is not attended with muscular atrophy, and electrical excitability is preserved. In subacute anterior poliomyelitis reflex action is earlier abolished than in this affection, there is almost entire absence of disturbances of sensibility and the functions of the bladder, and bulbar symptoms only appear at a late period, and the disease is never so rapidly fatal.

In *acute central myelitis* there is always a high degree of disturbances of sensibility, reflex action is early abolished, and in addition, there is paralysis of the sphincters, fever, acute bed

ores, a lowering of faradic excitability, and a rapidly fatal ermination.

The spinal form of syphilis, when it assumes the form of cute ascending paralysis, may be distinguished by the previous istory or evidences of still existing syphilis, and by the results f antisyphilitic treatment.

Acute multiple neuritis may be distinguished from acute scending paralysis by the severe pains limited to single nerve-oots, by the limitation of the anæsthesia and paralysis, and by he rapid lowering of electrical excitability.

§ 470. *Morbid Anatomy.*—All the examinations which have itherto been made have yielded completely negative results oth as regards the spinal cord, medulla oblongata, brain, sym-athetic nerves, peripheral nerve trunks, and muscles. The ames of Vulpian, Cornil and Ranvier, Bernhardt, Westphal, Déjerine, and Goetz, who have conducted the examinations, ufficiently attest the competency of the observers. Déjerine nd Goetz state that they observed changes in the anterior oots of the nerves. The altered fibres presented the ordi-ary characteristics of parenchymatous neuritis or degenerative trophy, such as are observed in the peripheral segment of a erve after section.

The following case appears to me to have been an example f Landry's paralysis; but, as I did not see the patient during ife, the diagnosis must, perhaps, be regarded as somewhat loubtful. The symptoms were reported to me by Mr. Wartenburg, who was then House Surgeon at the Royal nfirmary, and who took charge of the case in the absence of he House Physician :—

Henrietta R——, æt. twenty-one years, was admitted into the Royal nfirmary, under the care of Dr. Browne, on January 26th, 1877, and died he following day. On admission the lower extremities were completely aralysed, and there was partial paralysis of the upper extremities. The aralysis of the upper extremities became rapidly more pronounced, the espiratory muscles were soon implicated, and the patient died from sphyxia about thirty hours after admission. No sensory disturbances, culo-pupillary phenomena, vesical troubles, or bed-sores were noted. The istory obtained on admission was that the patient had had a slight blow n the back of the neck four days previously, and that she soon afterwards

became paralysed in the lower extremities. There were no contusions or other signs of injury.

The autopsy was conducted by me thirty-six hours after death, and the following is an abstract of the report: Three linear and deep cicatrices are observed in the left groin. The skin over the sacrum and trochanters is not ulcerated; the muscles are plump, and none of them present any signs of atrophy. The spinal cord was somewhat softer than usual in the lower half of the cervical and dorsal regions, and in the lower half of the lumbar enlargement and conus medullaris, the remaining portions being normal. The other morbid appearances noted were unimportant. I have repeatedly examined sections of the spinal cord, and always found the greater portion of the ganglion cells of the anterior horns so beautifully defined and healthy that I came to regard the cord as being typically healthy. I observed decided pathological changes in the central column, but regarded them as accidental, or at least of no importance so far as the functional disturbances present during life were concerned. When, however, embryological considerations forced upon me the conclusion that the central columns were endowed with important functions, my judgment of the significance of the morbid changes observed in this cord became altered. A section of the dorsal region is represented in *Fig.* 180; the

FIG. 180.

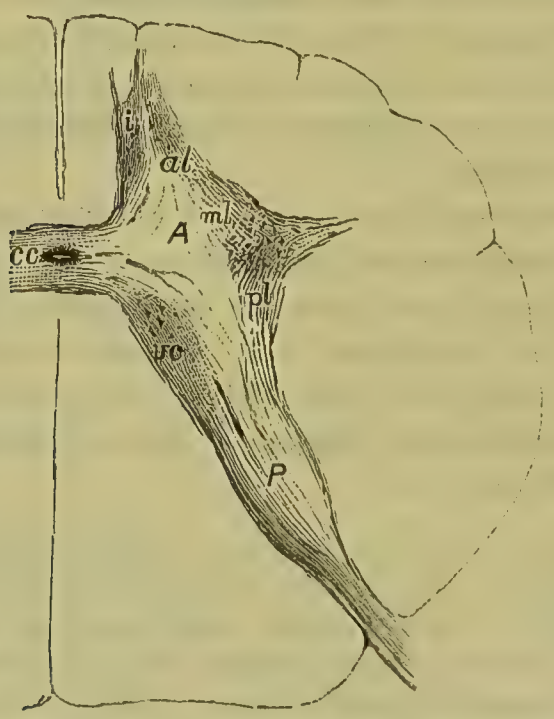
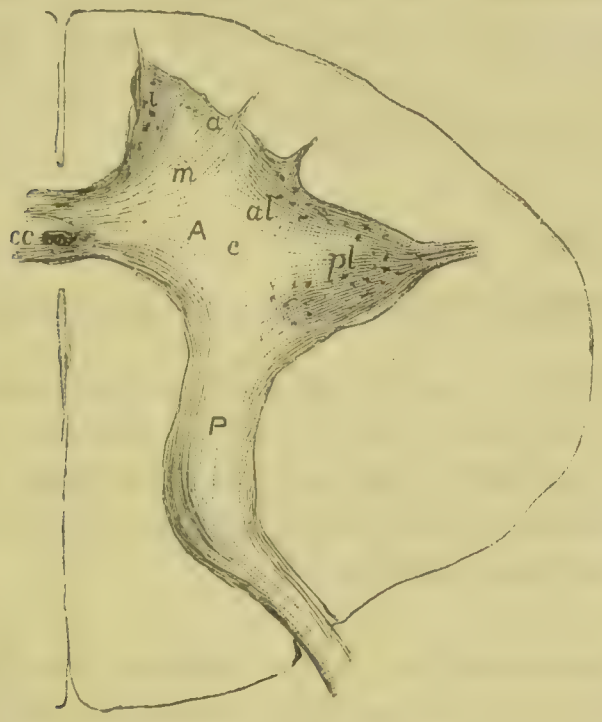


FIG. 180 (Young). *Section of the Upper Dorsal Region of the Spinal Cord, from case of Acute Ascending Paralysis.—A*, Anterior horns; *P*, posterior horns; *c* central canal; *vc*, vesicular column of Clarke; *i*, internal, *al*, antero-lateral *pl*, postero-lateral group of cells; *ml*, the medio-lateral area. The diseased portion is represented by the lightly-shaded area which occupies the central grey column and its extensions between the antero-lateral and postero-lateral groups, and between the internal and antero-lateral groups.

ernal, antero-lateral, and postero-lateral groups are normal, while the ıtral column and medio-lateral area are diseased. A section of the ddle of the cervical enlargement is represented in *Fig.* 181; the internal, terior, antero-lateral, and postero-lateral groups are normal, while the ıtral column, the median area, and the central group of ganglion cells ; diseased. The diseased areas showed granular degeneration of Ger-h's nerve network, complete disappearance of the ganglion cells, increase nuclei, and dilatation and congestion of blood-vessels.

Fig. 181.



G. 181 (Young). *Section of the middle of the Cervical Enlargement of the Spinal Cord, from a case of Acute Ascending Paralysis.*— *c*, Central group, and *a*, anterior group of ganglion cells; *m*, median area. The remaining letters indicate the same as the corresponding letters in *Fig.* 180. The diseased area is represented by the lightly-shaded portion which represents the central grey column and its extensions into the median area (*m*), between the antero-lateral and postero-lateral groups of cells, and round the central group.

§ 471. *Morbid Physiology.*—The pathology of this affection exceedingly obscure, and Westphal considers it probable that e disease is due to some intoxication, and a similar opinion d been maintained by Landry. In many respects this disease like *tetanus*. Acute ascending paralysis manifests itself, as name implies, by loss of motor power, while tetanus is mani-ted by symptoms of motor irritation, but both affections e similar in their mode of invasion, in their rapid course, and equently rapid fatal termination.

§ 472. *Prognosis.*—The prognosis is always very serious. The more rapid the ascending course of the disease, the earlier respiration has been attacked, and the more pronounced the signs of bulbar paralysis, the graver does the prognosis become. When once the progress of the disease is arrested and improvement begins, the prognosis becomes more hopeful, but even then there is danger of a relapse.

§ 473. *Treatment.*—At an early stage of the affection Chapman's ice-bag may be applied to the spine. The constant current has been employed in the later stages of the cases which have terminated favourably.

(II.) ACUTE DIFFUSED MYELITIS.

Acute Diffused Inflammation of the Spinal Cord.

§ 474. *Definition.*—Acute diffused myelitis comprises all the affections of the spinal cord which are attended by fever, and which lead in a short time to serious functional disturbances, with the exception of the acute system diseases of the cord, which have just been considered.

§ 475. *Etiology.*—Many cases apparently originate spontaneously without any recognisable exciting cause. The male sex appears to be more subject to the disease than the female sex. The greater number of cases occurs between the ages of ten and thirty, with the exception of infantile spinal paralysis. Sexual excesses, and severe bodily exertion, act as predisposing causes of the disease.

The most usual exciting causes of the affection are injuries such as those produced by puncturing and cutting instruments, fractures and luxations of the vertebræ, contusions, slow compression of the cord, and inflammatory processes transmitted from neighbouring organs. Exposure to cold, especially when the body is overheated, or after severe bodily exertion, and sleeping on the damp earth or in snow, are the most frequent causes of the affection.

Acute myelitis is not unfrequently developed as a complication

sequel of acute diseases, such as typhus, the acute exanthe-
ata, acute rheumatism, severe puerperal diseases, and more
pecially of variola.

Myelitis, running a very rapid course, is observed with un-
ual frequency amongst syphilitic patients. Suppression of
e menses and hæmorrhoidal bleeding play a more or less
ubtful rôle in the etiology of the disease.

Violent emotions appear sometimes to have caused the
fection, and several cases are recorded in which the first
mptoms of myelitis showed themselves immediately after
eat fright, anxiety, or anger.

Irritative lesions of peripheral organs give rise to acute
yelitis. A proportion of the so-called reflex paraplegias, which
e developed in connection with diseases of the digestive and
nito-urinary organs, or the joints, should be classed as acute
yelitis. Feinberg has recently succeeded in exciting an acute
lammation of the spinal cord in rabbits by varnishing the
in, but the mechanism of its production is unknown.

§ 476. *Symptoms.*—The symptoms of acute diffused myelitis
fer greatly in each individual case, so that it is difficult to
scribe the generic features of the affection. The onset of
e disease is very variable. It is sometimes preceded by
neral malaise, slight pyrexia with or without a feeling
chilliness, and the usual febrile accompaniments, headache,
neral depression, aching pains in the limbs, and loss of
petite. In many cases the spinal symptoms make their
pearance at once, disturbances of sensation being those
ich usually first attract the attention of the patient. The
mptoms of sensory irritation assume the greatest prominence,
t in some few cases they are entirely wanting, and the vio-
t shooting pains of meningitis are rarely present. The pains
myelitis consist of neuralgic pains surrounding the trunk
a variable height like a girdle, dragging, tearing, boring,
burning sensations in the limbs which are not increased by
essure or movement, and pain in the back extending over a
re or less extensive area. Several of the spinous processes
e often tender to pressure. The tender spot can sometimes
best elicited by passing hot and cold sponges or the cathode

of the galvanic current along the vertebral column. Various paræsthesiæ are almost constantly present in the disease, the most common and constant of these being the sensation of constriction, like a girdle, which is felt both in the trunk and in the extremities and joints. Feelings of tension or swelling, and of cold or heat, or pricking sensations and formication are experienced over more or less extensive areas of skin, especially that of the lower extremities. True hyperæsthesia is rarely present in acute myelitis, and when it occurs it is probably due to a complication with meningitis. It, however, occurs on the same side as the motor paralysis in unilateral circumscribed myelitis. As the disease advances the feelings of numbness and furriness become more and more prominent, and these are soon followed by the diffused painful and vibratory sensations which Charcot has named dysæsthesiæ, and which are produced by touching the skin of circumscribed areas, or of the entire surface of one or both extremities.

As the disease advances, the feelings of numbness and furriness give place to complete loss of sensation. The anæsthesia may be partial, or may manifest itself in the form of retardation of sensory conduction. At other times it may be more or less diffused and complete, although the parts deprived of sensation may be subject to severe pains—*anæsthesia dolorosa*. Shooting pains and spasmodic twitchings of the muscles are very common in the paralysed parts. Patients complain at times of painful, dragging sensations in the bladder and rectum, gastralgic attacks, and neuralgic pains in the other viscera. In all severe cases there is complete anæsthesia of the lower half of the body up to a certain height, the anæsthetic being marked off from the normal skin by a pretty sharply defined line.

The motor disturbances consist of both irritative and paralytic phenomena. In children the onset of the disease is marked by general convulsions. The symptoms of motor irritation of spinal origin are twitchings of individual muscles or of entire extremities, while at times the spasmodic contractions of the muscles may increase to a condition of tetanic rigidity. The paralytic symptoms are, however, much more constant and important. They may sometimes be developed with such rapidity that we speak of *apoplectiform* myelitis. In some

ses the paralysis may be developed in the course of a night, even in less than an hour, while in the hæmorrhagic form it ay develop in the course of a few minutes. When the paratic symptoms are rapidly developed the muscles are perfectly ccid, and offer no resistance to passive movements of the nts, and when the limbs are raised, they fall like those of a ad body. Symptoms of motor irritation may reappear in the fected limbs at a later period of the disease, if the patient rvive. Isolated spontaneous twitchings of the muscles may en be observed in the paralysed limbs, these being generally companied by severe shooting pains.

Spasmodic tonic contractions of the muscles occur, which e excited by a voluntary effort to move the affected limb, by irritation of the sensitive nerves. Ultimately severe ntractures are produced, which fix the legs in an extended or xed position, and are frequently rendered more intense by tempts at active or passive movements of the paralysed nbs. These symptoms are, however, more frequently obrved in the subacute and chronic than in the acute forms of yelitis.

The most common forms of paralysis are paraplegia, hemiraplegia, monoplegia, and paralysis of the cervical muscles, nile complete paralysis of all four extremities and of the eater number of the muscles of the trunk not unfrequently curs.

The *reflex excitability* varies according to the seat of the sease. The reflex activity of both the skin and muscles ay be diminished, destroyed, or increased. In some cases is abolished at an early period of the disease, and imediately after the development of the paralysis, so that flex actions cannot be excited even by severe irritants. At her times it is not entirely abolished, but a longer time is quired for the production of reflex movements; while in other ses it undergoes a considerable increase, so that slight irritions call forth active reflex muscular twitchings, which may crease to a convulsive jerking of the paralysed parts. In her cases the reflex excitability is unaltered, or it is slightly creased for a time and then begins to diminish and gradually comes weaker, and finally disappears.

The sphincters are frequently involved. Vesical paralysis may be one of the earliest, or even a premonitory symptom of acute myelitis. In severe cases complete retention of urine is usually present, so that the use of the catheter is rendered necessary. In other cases there is merely incontinence of urine, while in the beginning the symptoms are those of irritation, such as spasmodic closure of the sphincter, with increased desire to urinate. In severe cases the urine after the seventh or eighth day becomes alkaline and sometimes bloody; it contains numerous crystals of the triple-phosphates, and there is a muco-purulent deposit. Paralysis of the sphincter ani is generally present. *Priapism* is a not uncommon symptom in acute myelitis, the erections generally being incomplete, but often persisting for days, with slight variations in degree.

The *vaso-motor disturbances* are variable. Engelken found in one case a rise in the temperature of the paralysed parts, but most authors speak of the extremities as being cold. Diffuse œdema of the paralysed lower limbs is often observed. The perspiration is sometimes increased, sometimes diminished. The trophic disturbances consist of gangrenous inflammation of the skin over the sacrum, trochanters and other exposed situations, and these usually progress rapidly and prove fatal by septic fever. When the lesion is unilateral the bed-sore is situated on the opposite side of the body to the motor paralysis. Acute bed-sore may make its appearance as early as from the second to the fifth day of the disease.

The case may terminate so rapidly that there is no time for the development of trophic changes in the nerves and muscles, but traces of degenerative atrophy have been found even in rapidly fatal cases of central myelitis. The atrophy of the muscles is generally well marked when the disease has been of somewhat longer duration. In these cases there is loss of faradic irritability of the muscles and nerves along with the development of the reaction of degeneration. There are cases of acute myelitis, however, in which there is no change in the electric irritability, and others in which only slight quantitative changes such as slight increase or diminution can be demonstrated.

Cerebral phenomena are not often present in acute myelitis, t in children there may be headache, delirium, and general ıvulsions at the beginning. Headache and delirium some- ıes occur in the adult, but they are probably due to the ːompanying fever or to the simultaneous presence of sep- æmia or uræmia.

When the inflammatory process extends to the brain graver ·ebral symptoms become developed. In cervical myelitis ılo-pupillary symptoms are sometimes observed, and when ː medulla oblongata is implicated speaking and swallowing ıy be interfered with. Disturbances of the optic and ocular rves, such as occur in cases of locomotor ataxia, have not en observed.

The bowels are obstinately constipated, and at a later period ː abdomen may be greatly distended with flatus.

The pulse is increased in frequency, and when the myelitis ːends to the cervical portion of the cord the acceleration of ː pulse may be very great. Many patients suffer from palpi- ion and irregularity in the action of the heart, accompanied unpleasant sensations in the cardiac region.

When the abdominal muscles become paralysed, all forcible ɔiratory acts, as coughing, are rendered feeble, and conse- ently the air passages cannot be cleared of mucus. The ːctores spinæ are probably paralysed even before the ab- minal muscles.

As the disease ascends still further the intercostal muscles ɔome paralysed, and the patients breathe only with the ıphragm. When the latter muscle is involved in the para- is the dyspnœa becomes intense, inspiration being only ·ried on by means of the cervical muscles, and death by ɔhyxia is imminent.

The formation of bed-sores and cystitis cause sleeplessness d loss of appetite, which are soon followed by marked dis- ·bance of nutrition, great emaciation, and exhaustion.

Fever is generally present in the commencement of acute ·elitis. It may at times run very high and remain per- tently high throughout the entire course of the disease. At ıer times it occurs in isolated sharp attacks, and an excessive e of temperature is not unfrequently observed immediately

before death. In other cases the fever is slight, never attains a high grade, and may disappear entirely during the subsequent course of the disease. An exhaustive symptomatic fever occurs in the latter stages of the affection, caused by the bed-sores, cystitis, pyelo-nephritis, and consequent septic infection.

§ 477. *Course, Terminations, and Duration.*—The course of acute myelitis is exceedingly variable, but it is always rapidly developed, and it is this feature which entitles it to be regarded as an acute affection. Cases which take more than ten days to develop may be regarded as subacute. The paralysis may at times be developed in an apoplectiform manner almost without premonitory symptoms, and it may attain considerable intensity in an hour or even less. As a rule, however, there is a premonitory stage of variable length, and days may elapse before the paralysis develops into pronounced paraplegia. At times the development of the disease is interrupted by remissions instead of being continuous.

In central myelitis and hæmatomyelitis the paralysis rapidly ascends, symptoms of asphyxia appear, and death takes place in a few days; or the fatal termination is brought about by the violent fever and septicæmia caused by the acute bed-sores and cystitis, and life may then be prolonged for a period of a few weeks. In the less severe cases, particularly when the entire lumbar enlargement is affected with or without the dorsal portion of the cord, the course is somewhat slower. There is complete paraplegia with paralysis of the bladder, cystitis, decubitus, fever, cachexia, and exhaustion, and the patient succumbs after several weeks or months.

In all other cases chronic myelitis is developed, and the symptoms are then complete motor paralysis with incomplete paralysis of sensation and of the bladder. The symptoms may then remain stationary for months or years. After the disease has persisted for a variable time cystitis and bed-sores may develop, but they never become very severe and are susceptible of being at least partly cured. Death finally results from exhaustion or from some intercurrent disease.

In other cases the disease ceases and the general health soon completely restored. The disorders of sensation and

bladder, and the trophic disorders of the skin, are usually irely wanting. The disease then terminates in imperfect overy, the only traces left being paralysis and atrophy of e or more muscular groups.

Complete recovery takes place in rare cases, and in these nptoms of improvement set in early. After paralysis, fever, l other symptoms of a mild attack of acute myelitis have sisted for one or two weeks they undergo a slow and gradual rogression, and in a few weeks all the functions of the body e completely restored, although convalescence is somewhat otracted.

§ 478. *Morbid Anatomy.*—After acute inflammation the nal cord is generally softened, but the appearances presented the diseased parts differ according to the stage of the mye-is. Leyden divides the inflammatory softenings of the spinal rd into (1) red, (2) yellow, (3) white, (4) grey, and (5) green purulent softening.

(1) *Red Softening.*—The stage of hyperæmia and com-encing exudation is not often met with post mortem. It ay, however, be observed in cases of traumatic and central yelitis which run a rapidly fatal course. The affected spot ay be found swollen, the transverse markings on section ing blurred and indistinct, and the cut surface presenting a riegated marbled appearance. The colour may vary from a sy injection to a deep red, reddish brown, or chocolate colour, d numerous capillary hæmorrhages may be observed. The flamed spots are moist and soft, and swell up above the level the cut surface, and at times the tissue becomes diffluent. rare instances a slight increase of consistency is observed in e stage of hyperæmia. The membranes in the neighbourhood the affected spot also frequently present the signs of hyper-mia and inflammation.

(2) *Yellow Softening.*—As the disease progresses the affected rts become paler and softer, hence this stage may be called at of yellow softening. The change of colour from red to llow is due partly to the diffusion and alteration of the louring matter of the blood, and partly to the fatty degene-tion of the medullary sheaths, and the formation of masses fat-granules.

which undergo a granular degeneration and are finally absorbed. The axis cylinders are swollen, often attaining enormous dimensions. They present large spindle-shaped swellings, appear cloudy, and are in a state of granular degeneration.

In the second stage the nerve fibres are broken down into fragments, the medullary sheath is in a state of fatty degeneration, and the axis cylinders altered or destroyed.

(4) *The Ganglion Cells.*—The nerve cells are involved in the inflammatory changes. They are swollen, often to a considerable size, their substance is clouded, and the nucleus and nucleolus may at times be observed in process of division, while at other times they undergo vacuolation. Their processes are also swollen, cloudy, irregular in shape, and in part destroyed. At a later period the cells shrivel, lose their processes, and become reduced to small angular masses without structure, while some may entirely disappear.

When a cicatrix has formed, the affected spot is occupied by a dense connective tissue containing numerous nuclei and neuroglia cells; numerous Deiter's cells may be observed, and they are often large and possess numerous processes. The vessels are dilated, their walls are thickened, and the ganglion cells, if any remain, are atrophic. When cysts are formed they are surrounded by a more or less dense layer of connective tissue, and they are generally traversed by a loose connective tissue network. Nothing can be detected in the fluid except detritus. When the case has become chronic and sclerosis has occurred, the increase of the interstitial tissue becomes particularly distinct. This condition will, however, be described under chronic myelitis. *The peripheral nerves* have been found in different stages of degenerative atrophy, more especially in central myelitis. *The muscles* also manifest the characteristic appearances of the first stage of atrophy.

The mucous membrane of the bladder is often found swollen and covered with mucus. Hæmorrhagic spots are frequently observed in it, or it may present sloughs, or be covered with diphtheritic exudation. Similar changes are often found in the ureter, extending to the pelvis of the kidney. The kidneys are filled with metastatic abscesses. The lungs are frequently congested, and often present the appearances which characterise

pneumonia; the pleuræ and pericardium are generally studded with small hæmorrhagic spots.

The large sloughs characteristic of acute bed-sores are usually found on the parts exposed to mechanical pressure, as the sacrum, nates, and trochanters.

§ 481. *Morbid Physiology.*—The initial symptoms of irritation must be ascribed to the increase of the irritability of the nerve cells and fibres, caused by increased nutritive activity during the early stages of the inflammatory process, and the later symptoms of paralysis to the destruction of these elements and their compression by the inflammatory exudation. The girdle pains depend upon implication of the posterior roots in the inflamed focus, and the paræsthesiæ and neuralgic pains of the inferior half of the body by irritation of the sensory tracts situated in the grey substance and in the posterior white columns of the cord. Since the sensory tracts either lie within the grey substance or pass through it for a certain distance the intense anæsthesia in acute central myelitis may be readily accounted for. In the circumscribed, disseminated, and cortical forms of myelitis, the degree of disturbance of sensation will depend solely on the extent to which the posterior roots and sensory tracts are involved in pathological changes.

The motor disturbances are first caused by irritation and subsequently by loss of function of the motor centres and conducting fibres in the cord. When the anterior horns of grey matter are affected, paralysis ensues with complete flaccidity of the limbs. When the pyramidal tract is affected, loss of voluntary power ensues, followed, if the patient survive some weeks, by muscular tension and contractures.

The condition of the reflex excitability furnishes a valuable indication of the state of the grey substance. So long as the grey substance is unaffected, so long is there a continuance of reflex actions, unless indeed the reflex arcs be interrupted in their passage through the posterior and anterior roots and their continuations through the white columns to reach the grey substance. When a portion of the grey substance is separated from its connection with the brain by a myelitis situated higher up the cord, reflex actions become increased. In transverse

myelitis of the dorsal region, the lumbar portion of the grey matter is separated from the brain, but being itself healthy the reflex of the sole is increased. When the disease is situated in the cervical region, the cremasteric, gluteal, abdominal, epigastric, dorsal, and scapular reflexes are increased. When the grey substance is destroyed by disease, the reflexes are abolished, hence their disappearance in myelitis of the lumbar enlargement, and more especially in diffuse central myelitis, in which all reflex action is usually abolished from the very commencement. When inflammation gradually extends downwards, the reflex activity, which may have been normal or increased at first, subsequently becomes weaker and gradually disappears. Important conclusions can be drawn from the condition of the reflex excitability with regard to the localisation of the myelitis in the grey substance. The reflex actions can also be modified by disease of the anterior roots, and disease of the lateral columns appears also to exert an important influence on them, especially on the tendon reflexes.

When the myelitis affects the lumbar region, the sphincters of the bladder and rectum become involved, beginning with spasmodic closure of them and ending in paralysis. The priapism which is so often present must be regarded as a sign of irritation produced either by irritation of the nerve tracts passing from the brain to the lumbar portion of the cord, or by reflex excitations proceeding from the bladder or skin.

The trophic affections of the muscles and bones are caused by lesion of the ganglion cells of the anterior horns, while the cutaneous trophic affections and bed-sores appear to be caused by disease of the posterior horns. The vaso-motor disturbances are caused by interference with the vaso-motor centres of the cord, but the mechanisms in the cord which preside over these functions are not yet clearly ascertained.

§ 482. *Varieties of Acute Diffused Myelitis.*

The following forms of acute diffused myelitis may be distinguished:—1, Acute central myelitis; 2, Hyper-acute central myelitis or hæmatomyelitis; 3, Acute bulbar myelitis; 4, Acute transverse myelitis; 5, Acute hemilateral myelitis; 6, Acute myelo-meningitis; 7, Acute disseminated myelitis.

1. *Acute Central Myelitis.*—This affection begins suddenly by disturbances of sensation, which are soon followed, often in the course of a few hours, by complete anæsthesia and paralysis of the lower extremities, with flaccidity of the limbs, and paralysis of the bladder and rectum. The reflex excitability is abolished, and the paralysed muscles undergo early and rapid atrophy, with loss of faradic contractility. The further progress of the disease is marked by acute bed-sores, cystitis, and ammoniacal urine, œdema of the paralysed limbs, arthropathies, more or less intense fever, progressive advance of the paralysis upwards, and early death by asphyxia.

Morbid Anatomy.—In this form of myelitis the softening affects principally the grey substance, which often flows out or becomes depressed under the surface of section when the cord is divided transversely. On microscopic examination the disease is seen to extend in the lumbar region forwards from the central column into the anterior grey horns, the ganglion cells of which may be entirely destroyed, and also backwards into the posterior grey horns. In the cervical and upper dorsal regions of the cord and in the medulla oblongata, however, the disease is restricted to the central grey columns, and the accessory cells of the anterior horns and their continuations through the medulla, while the fundamental cells in these regions may be almost entirely unaffected.

2. *Hæmatomyelitis* is a variety of acute central myelitis, and is distinguished from the latter clinically only by the sudden onset of the symptoms. In hæmatomyelitis the paralysis becomes complete in the course of a few minutes, or at most in the course of an hour or two, and the more rapid is the development of the disease the more predominant is the hæmorrhagic element in the morbid process likely to be. Hæmatomyelitis can only be distinguished from hæmorrhage into the substance of the cord in those cases in which the paralytic symptoms are preceded by paræsthesiæ, girdle sensations, vesical weakness, muscular twitchings, and fever.

Morbid Anatomy.—The morbid anatomy is the same as that of acute central myelitis, except that in hæmatomyelitis the central softened mass assumes a hæmorrhagic character.

3. *Acute Bulbar Myelitis.*—This disease always begins suddenly by violent headache, giddiness, vomiting, distressing hiccough, various paræsthesiæ, difficulty in swallowing and speaking. Consciousness is, however, preserved contrary to what occurs in an apoplectic attack. After a short time severe symptoms of bulbar paralysis make their appearance. The degree in which the inferior muscles of the face, the muscles of the tongue and soft palate, and those of deglutition are affected varies greatly in different cases. Disturbances of respiration appear at an early period of the disease. The respirations are hurried and irregular, and there may be an intense feeling of oppression, dyspnœa, and cyanosis. The pulse is small, quick, and irregular, or intermittent. When the anterior pyramids

are affected, one, two, or all four extremities may be paralysed, or there may be complete absence of paralysis of the limbs. The patient may complain of pains and formication in the limbs, but there is no anæsthesia. The bladder and rectum may become paralysed towards the terminal period of the disease. The disturbances of circulation and respiration increase, the patient becomes unconscious, and death results from asphyxia.

Morbid Anatomy.—In three cases of acute bulbar myelitis, observed by Leyden, small centres of softening without definite boundaries were found in the medulla oblongata. In two cases the centres of softening occupied the internal portion of the medulla from the floor of the fourth ventricle to the anterior pyramids; while in the third a small centre of softening was found situated between the restiform and olivary bodies, and roots of the hypoglossal nerve.

4. *Acute Transverse Myelitis.*—This variety of acute inflammation of the cord is represented by the form of the disease which develops after severe injuries of the spinal cord and vertebral column. The symptoms vary greatly according to the level at which the cord is diseased. For practical purposes acute transverse myelitis may be subdivided into: (*a*) Acute dorso-lumbar transverse myelitis; (*b*) Acute dorsal transverse myelitis; and (*c*) Acute cervical transverse myelitis.

(*a*) *Acute Dorso-lumbar Transverse Myelitis.*—This variety usually begins by fever, formication, numbness, and pain in the lower extremities. These symptoms are accompanied or soon followed by startings of the limbs, but the phenomena of sensory and motor irritation soon give place to those of paralysis. The paralysed limbs are, rigid, and the reflexes both cutaneous and deep, are exaggerated, and there is a tonic spasm of the sphincters. After a time the urine becomes altered, acute bed-sores appear over the sacrum and trochanters, intermittent fever supervenes, and the patient dies from marasmus.

Sometimes the onset and progress of the disease is so rapid that it deserves to be called *hyper-acute* or *apoplectiform.* Hayem has reported two cases, in which death occurred in the one in five days, and in the other in twelve.

(*b*) *Acute Dorsal Transverse Myelitis.*—This form of the disease is the least dangerous of the three. The sensory and motor disturbances are more or less similar to those which occur in the dorso-lumbar form. In acute dorsal transverse myelitis, however, there is complete absence of bed-sores, the bladder is unaffected, and consequently the septicæmia and marasmus are absent. Cases of this kind may completely recover, but, as a rule, the affection assumes the chronic form. A large number of cases of chronic paraplegia belong to this variety.

The patient may at times recover from a first attack, and may even partially recover from a second and third attack, and ultimately succumb to the disease many months after the first attack (Pierret).

(*c*) *Acute Cervical Transverse Myelitis.*—This form of the affection

generally begins with pain in the back of the neck, followed by *spasmodic* rigidity of the muscles of the back of the neck, which may at times be more pronounced on one side, so that the head is rotated as well as retracted. The pain and spasmodic contractions soon extend to the upper extremities, and these symptoms are succeeded by paralysis, muscular atrophy, and various other trophic disturbances, such as œdema and arthropathies. The hands assume characteristic positions, but these will be subsequently described when we come to speak of the chronic varieties of the disease. The lower extremities become implicated after a time, and if the case do not terminate fatally in a brief space of time, the muscles become the subjects of contractions, and the deep reflexes are exaggerated. The pupils are in some cases contracted, in others dilated, and there may be great pallor or flushing of the face, but in other cases oculo-pupillary and vaso-motor phenomena have been absent.

The paralytic phenomena may be preceded for some time by anomalous symptoms, such as coughs, dyspnœa, sweats, wasting, pains between the shoulders, severe attacks of vomiting recurring at regular intervals, difficulty of deglutition, hiccough, and great slowness of the pulse, which may fall to twenty-eight beats (Gull), or even to fifteen beats per minute (Eulenburg). The inflammatory process generally ascends to the medulla, and death results from arrest of respiration.

Morbid Anatomy.—Little need be said with regard to the morbid appearances presented in cases of acute transverse myelitis. The main centre of the lesion is found in various stages of softening, and the cord may be so diffluent that the softened mass flows out on section. If the case has assumed a more or less chronic form, the usual ascending and descending degenerations are observed above and below the primary seat of disease.

In cases which are rapidly fatal there is no time for the secondary degenerations to occur; but I have always observed that the central grey substance is affected both above and below the primary lesion. In the neighbourhood of the primary lesion all the ganglion cells of the anterior horns may be destroyed; but in the portions of the cord remote from the latter, the disease of the grey substance becomes more and more limited to the central grey column, and the margins of the groups of the ganglion cells of the anterior horns. Although *Fig.* 146 has been taken from a somewhat protracted case of myelitis, I have observed similar appearances in the cervical region of the cord from a case of fracture of the vertebral column in the dorsal region, which proved fatal in five days. The continuation of the central column upwards into the medulla, and the accessory nuclei of the latter, are generally implicated.

5. *Acute Hemilateral Myelitis.*—When the lesion is limited to one-half of the cord there are motor paralysis, hyperæsthesia, and elevation of temperature on the side of the lesion, and more or less anæsthesia on the opposite side. The symptoms and morbid anatomy of this affection will be more fully described hereafter.

6. *Acute Myelo-Meningitis.*—Acute myelitis is frequently complicated with meningitis. We shall hereafter speak of those cases in which myelitis is developed simultaneously with or as a complication of acute meningitis, but at present we wish to limit our remarks to the cases in which meningeal changes are superadded to acute myelitis. This complication is not very important, since the addition of meningitis to an acute inflammatory affection of the cord does not appear to add to the gravity of the latter. The symptoms which indicate meningitis are pain and stiffness in the back and neck, pronounced hyperæsthesia, and diffused pains. When these symptoms are superadded to those of acute myelitis, the case is likely to be one of myelo-meningitis.

Morbid Anatomy.—When the membranes are affected they become congested, and sometimes capillary extravasations may be observed in them, or they are infiltrated with serum and cellular elements, the spinal fluid is increased in quantity, is cloudy or reddish, and in more protracted cases the membranes become thickened and loosely adherent. On microscopical examination the membranes are found thickened and infiltrated with cellular elements, while in the cortical layer of the spinal cord itself a large number of the nerve fibres are destroyed, the connective tissue septa being thickened, and a great increase in the number of Deiter's cells having taken place. In carmine preparations, after hardening, the cord is surrounded by a highly-stained border which extends into the substance of the cord to a greater or lesser depth, according to the extent of the disease. This form of disease has consequently been called *peripheric* or *cortical* myelitis.

7. *Acute Disseminated Myelitis.*—The symptoms of this affection are paraplegia, sometimes associated with spastic symptoms. The bladder is generally paralysed. The state of the sensibility is variable, but it is generally more or less impaired. The reflex and electric excitability is also variable, both being generally diminished. The grouping of the symptoms and the exacerbations show sometimes during life that several centres of disease exist. The disease may be suspected if the symptoms of acute myelitis supervene during an attack of variola, or if they develop suddenly in phthisical patients.

Morbid Anatomy.—This form of myelitis occurs in small spots scattered through the substance of the cord, and appears to form an intermediate grade between the acute and chronic forms of myelitis. The essential characteristic of the morbid process appears to be an increase in the interstitial tissue, which becomes unusually dense, and rich in nuclei. The septa are swollen, the walls of the vessels thickened, and some granule cells are observed. The consistence of the cord is often increased.

§ 483. *Diagnosis.*—Typical cases of acute myelitis are easily recognised, but the less pronounced cases and those complicated by the presence of other affections are difficult to unravel. The

characteristic symptoms are, the sudden onset of the affection, the presence of more or less marked signs of sensory and motor irritation, the rapid development of complete paralysis of some of the limbs and of the bladder, the rapid formation of bed-sores, and the presence of fever.

Acute myelitis may be mistaken for the following diseases:—

Acute ascending paralysis resembles diffuse central myelitis so closely that it is scarcely possible to distinguish the two affections. Landry's paralysis is characterised by the absence of convulsive movements at the onset of the affection, absence of trophic disturbances, slight degree of sensory disturbances, and the preservation of the faradic contractility of the paralysed muscles.

Acute meningitis of the cord is characterised by high fever, severe pain, dorsal and cervical rigidity, contractures, slight symptoms of paralysis, especially of the sphincters, absence of severe trophic disturbances, and pronounced hyperæsthesia. The two diseases are often combined, and then the difficulties of diagnosis become greater.

Hæmatomyelia or simple hæmorrhage into the spinal cord is very difficult to distinguish from central myelitis, especially from the hæmorrhagic form of the affection. In the former the paralysis is developed suddenly without fever or prodromata, and the paralysis is stationary instead of being progressive as in the latter.

Hæmatorrhachis or hæmorrhage into the meninges of the cord is characterised by a very abrupt development without premonitory symptoms or fever, symptoms of severe meningeal irritation, violent pains, dorsal rigidity, a comparatively slight degree of paralysis, and particularly by the slight intensity of the anæsthesia.

Hyperæmia of the cord is characterised by the absence of fever, the slight intensity of the sensory and motor disturbances, the frequent and rapid variations in the symptoms, and the absence of vesical weakness and of bed-sores.

The diagnosis of hysterical paralysis from acute myelitis will hereafter be considered. Several poisons produce symptoms which resemble closely those of acute central myelitis.

The seat of the disease in the cord and its *extension* in the transverse and vertical directions can be determined from the area of the paralytic phenomena, the state of the reflex irritability, and the trophic disorders.

§ 484. *Prognosis.*—The prognosis is generally unfavourable, but there are exceptions to this rule. Perfect recovery is rare. In many cases a chronic disease is induced; whilst in others there is arrest of the disease, but incurable defects remain.

The prognosis depends on the locality and extent of the inflammatory process. The higher up the disease is situated in the dorsal and cervical regions, the greater the liability of the respiratory tracts to become affected and the greater is the danger to life. Dorsal myelitis is, however, other things being equal, more favourable than dorso-lumbar myelitis, because the genito-urinary automatic centres are unaffected in the former.

The prognosis is the more unfavourable the greater the extent of the transverse section of the cord involved in the process. It is rendered very unfavourable when the central and posterior portions of the grey substance are involved, owing to the consequent cystitis and acute bed-sores. It is doubtful how far implication of the white columns influences the prognosis.

The prognosis also becomes worse in proportion to the longitudinal extent of the disease. A circumscribed transverse myelitis is not so dangerous as the same affection when it extends over a greater length of the cord. The progressive ascending forms of the disease, and particularly of central ascending myelitis, have a particularly unfavourable prognosis. A longitudinal extension of the disease in the white columns is not so dangerous.

A rapid development and great intensity of the paralysis, complete paralysis of the sphincters, early formation of acute bed-sores, progressive advance of the disease upwards, high fever, impairment of the general health, dyspnœa, cyanosis, and other disorders of respiration influence the prognosis unfavourably. A moderate degree of paralysis, absence of trophic and sensory disturbances, implication of the bladder to only a slight extent, absence of fever and of marked impairment of the general health, and commencing improvement of some of the nervous

symptoms are all favourable signs. The nature of the causes of the affection and the possibility of their removal, the occurrence of relapses, the state of the general health, and the effects produced by treatment are other elements which should be taken into account in forming a prognosis.

§ 485. *Treatment.*—In acute diffused myelitis the patient should maintain absolute rest in bed, and lie as much as possible on his side or abdomen, while Chapman's ice-bag is applied to the spine. It is also desirable to place the patient on a water bed, and the parts exposed to pressure should be sponged with brandy or whisky, and dusted with an absorbent powder, in order to prevent, if possible, the formation of bed-sores.

Internal remedies do not appear to be of much use in the treatment of acute myelitis; but ergot or belladonna is supposed to do good in the early stage of the disease, and iodide of potassium may be given to promote absorption when the fever has subsided. A saline mixture may be given in order to maintain the activity of the skin and kidneys, while the bowels should be gently acted on by mineral waters containing sulphate of magnesia, or by senna, rhubarb, or castor oil. When myelitis becomes developed in the course of syphilis, energetic mercurial treatment in combination with large doses of iodide of potassium is to be adopted.

Counter irritation to the spine should be used in great moderation, for fear of favouring the formation of bed-sores. In cases where the disease is situated in the dorsal and upper cervical regions a blister may be applied, or a hot iron may be used; but counter irritants should never be applied in cases where the skin is anæsthetic, and all the parts exposed to continuous pressure should be avoided in their application.

The galvanic current should never be employed in the acute stage of myelitis, but it is useful in the treatment of the sequelæ of the affection, and in the treatment of the more chronic forms. The diet should be easily digestible and nourishing, and no tea, coffee, or alcohol should be allowed.

Great care should be used in introducing the catheter for fear of setting up or aggravating the cystitis. The catheter should always be washed in carbolic acid lotion, and lubricated

with carbolic oil, in order to prevent the introduction of bacteria into the bladder. When the disease has become subacute or chronic, the treatment must be modified accordingly.

(III.) CHRONIC DIFFUSED MYELITIS.

Chronic Inflammation of the Spinal Cord.

§ 486. *Definition.*—Chronic diffused myelitis comprises all those slowly-developing and diffused processes in the spinal cord which run a lingering course without fever, and which are at present ascribed to chronic inflammation.

§ 487. *Etiology.*—It is very probable that individuals inheriting a neuropathic constitution are predisposed to myelitis, but no statistical proof has as yet been forthcoming. The other predisposing causes of the disease are mental and bodily over-exertion, dissipation, sexual excesses, syphilis, emotional excitement, especially the depressing emotions. The disease is most common during youth and middle age, and in the male sex.

Chronic myelitis can develop from the acute form, although the former can be produced primarily from the same causes as the acute.

The following are the usual exciting causes of the affection :— Exposure to cold, long sojourn in damp and cold localities, sleeping on damp earth, bodily over-exertion, especially when combined with exposure to cold, as occurs during campaigns. Simple concussion of the cord, without direct injury, and gradual compression, may also give rise to chronic myelitis, and sexual excess may act both as a predisposing and exciting cause.

Syphilis is a fruitful source of chronic myelitis. Syphilitic neoplasms are not here in question, but cases of chronic myelitis which arise in the course of secondary syphilis, or in persons who had previously suffered from the disease, and where no specific lesion can be discovered to account for the affection. It is probable, therefore, that we have here to do with simple myelitis in persons predisposed by syphilis to chronic inflammations.

Chronic myelitis is occasionally developed as a sequel of various acute and chronic diseases, such as lepra, chronic

alcoholism, and chronic lead poisoning. It may also be developed from irritation and diseases of peripheral organs, and most of the cases called reflex paralysis belong to the category of subacute and chronic myelitis.

§ 488. *Symptoms.*—All the diseases comprised under the term chronic myelitis cannot be included in one general description. The following description applies more particularly to transverse myelitis, where one large focus of disease exists at any height in the cord, or where there are several foci, one of which, however, determines the chief clinical features of the affection. In the majority of the cases belonging to this class the symptoms develop slowly and gradually. Sensory disturbances are first complained of, consisting of abnormal sensations and anæsthesia in the lower, more rarely in the upper extremities, and these may entirely disappear for a time. They are frequently accompanied by a girdle sensation in the trunk, or perhaps also in the extremities. Painful sensations, severe pains, and hyperæsthesia are rarely complained of. The symptoms are variable and inconstant, and only attain a high degree of intensity very slowly and gradually.

Of the motor disturbances which occur in the early stage of the affection those of motor irritation are of subordinate importance. These usually consist of slight twitching movements of the legs, or the patient may experience slight muscular contractions and trembling of the legs after prolonged exertion. The paralytic symptoms are usually more prominent and important. The first motor symptoms to attract the patient's attention are a feeling of weakness and heaviness of the limbs and an undue sense of fatigue on slight exertion. These symptoms may at first be relieved by walking, patients often feeling stiffer and more fatigued at starting than after they have walked for some time. The affection occasionally begins by vesical weakness manifested by slight incontinence or retention, while on rare occasions the onset may be marked by pronounced paralysis of the bladder.

As the disease advances the symptoms become slowly and progressively aggravated, or become suddenly worse under the influence of one of the exciting causes of the affection.

The stiffness and weakness of the lower extremities now become more marked, the legs feel as if they were made of lead, and the feet are slowly dragged along the ground, the toes catching readily in every inequality. The movements of the toes, and of the fingers if the upper extremities be implicated in the paralysis, are slowly performed, each of them being accompanied by a large number of associated movements. The paralysis generally assumes the typical form of spinal paraplegia, although occasionally it appears as spinal hemiplegia, and still more rarely the arms may be paralysed, while the legs remain very little or not affected.

Anæsthesia of varying forms and degrees of intensity, usually involving the lower extremities and the trunk up to a variable height, is rarely wanting. Retardation of sensory conduction and various paræsthesiæ are often observed, and the patients occasionally complain of dysæsthesiæ.

The *reflex activity* is increased in the majority of cases, and a variety of reflex movements may be produced by irritation of different sensitive surfaces. Tickling the soles of the feet causes active jerking and kicking movements in the paralysed legs; while the introduction of a catheter, or the act of dressing a bed-sore, may also excite movements in them. Evacuation of urine may be produced by irritating the skin of the feet or by introducing the finger into the rectum, erections may be produced by rubbing the inside of the thighs, and a discharge of fœces often occurs during the dressing of a bed-sore.

The *tendon reflexes* are as a rule abnormally active, the reactions obtained being similar to those described under spastic spinal paralysis. In some cases the various reflexes are diminished or even entirely abolished. In these cases the grey substance is extensively involved, or the conducting fibres of the various reflex arcs are destroyed as they pass through the nerve roots.

The *vaso-motor* disturbances are as a rule not very prominent. The patients complain of coldness of the feet, and the limbs are often of a cyanotic or bluish-red colour.

The *electric* irritability of the nerves and muscles is generally preserved, both quantitatively and qualitatively, when the reflex actions persist, and in some cases both faradic and galvanic

irritability may be increased. When the reflexes, however, are abolished, in consequence of extensive destruction of the grey matter, the muscles undergo atrophy accompanied by loss of their faradic excitability and the reaction of degeneration. The distribution of the atrophy is very variable. At times the upper extremities are alone or chiefly affected by the atrophy, the lower limbs presenting simple paralysis without atrophy.

In transverse myelitis bed-sores are sooner or later developed in the usual places. The bed-sores usually assume the chronic form, although they may occasionally pursue an acute course. In some rare cases large sloughs may become permanently cicatrised, even though there be no noticeable improvement in the other symptoms of the disease.

The sphincters are almost always involved to a more or less extent, and the vesical functions are particularly liable to be impaired at an early period, but there are some exceptional cases in which the bladder remains unaffected thoughout the entire course of the disease.

Sexual power is usually impaired at an early period. It diminishes with more or less rapidity, and finally disappears entirely. In cases of incomplete paraplegia sexual power may be preserved for a long time.

The general health may remain perfect for a long time; but in all the more severe cases a constantly increasing disturbance of general nutrition is observed, which becomes more marked as soon as cystitis and bed-sores are developed. Loss of appetite, fever, progressive emaciation, and exhaustion constitute the final symptoms, provided life be not terminated sooner by some acute intercurrent disease.

The cerebral nerves are, as a rule, not implicated in cases of chronic transverse myelitis, except towards the termination of the disease. In some cases the morbid process ascends until it at last reaches the medulla oblongata, giving rise to disorders of deglutition, speech, circulation and respiration, and ultimately causing death by asphyxia.

§ 489. *Course, Duration, and Terminations.*—When the chronic is preceded by the acute form, the onset of the disease is of course sudden. In most cases, however, the disease super-

venes slowly and gradually, so that the first symptoms attract little or no attention. The development, instead of being continuous, is sometimes marked by intervals of more or less improvement, which alternate with exacerbations and slight relapses.

The disease on attaining a certain height may remain stationary for many months or even years, or it may slowly progress and terminate in different ways. The duration of the affection generally extends over many years, and in some cases it may last a lifetime without producing dangerous symptoms.

Complete recovery is rare, and even in those rare cases relapses are liable to occur. Various symptoms, such as paralysis, atrophies, partial anæsthesiæ, deformities, vesical disorders, and other affections, often remain behind permanently. The usual course of the disease is a slowly progressive one, leading gradually and in different ways to a fatal termination. The process may slowly creep upwards until disturbances of deglutition and respiration occur, and death results from asphyxia. At other times the secondary affections caused by the myelitis, such as bed-sores, cystitis, pyæmia, and septicæmia, destroy the vital powers of the patient and lead to death by exhaustion and marasmus. In a large number of cases death is caused by an intercurrent affection, such as pneumonia.

§ 490. *Morbid Anatomy.*—The morbid appearances presented by the spinal cord are often so slight that they cannot be detected by the naked eye. In many cases, however, changes occur in the consistence, colour, and form of the cord, which can be readily detected.

It is unnecessary to repeat here what has already been said with regard to sclerosis or grey degeneration (§ 387). It will suffice to remind the reader that in subacute cases, or in the earlier stages of a chronic myelitis which has developed from the acute form, the tissue, instead of being found in a state of sclerosis or grey degeneration, may have undergone softening, or the morbid process may have led to the formation of cavities. It must also be remembered that a chronic myelitis often terminates by an acute attack, and that softening may be found associated with sclerosis.

In chronic myelitis the cord undergoes various changes of form, according to the extent and localisation of the lesion. An increase of volume of the whole or circumscribed parts of the cord is rare, but a diminution of volume or atrophy is common. The atrophy may be general and affecting the transverse area of the cord equally in all directions, or it may take place in particular directions. The cord may be flattened in the antero-posterior direction, so that it presents the appearance of a band, or the surface may be depressed the entire length of certain columns, as the posterior columns in locomotor ataxy, or the surface may be depressed in isolated and circumscribed spots.

Microscopic Examination.—Changes have been observed in the (1) connective tissue, (2) the nervous tissues, and (3) in the blood-vessels.

(1) *Connective Tissue or the Neuroglia.*—The connective tissue septa become thickened, the neuroglia cells enlarge and their nuclei undergo proliferation, while Deiter's cells become greatly increased in size and number. After a time the normal neuroglia becomes converted into a dense, fibrillated connective tissue in which a large number of nuclei may be observed.

(2) *Nervous Tissues.*—The *nerve fibres* undergo changes which correspond to some extent with the secondary degeneration of the fibres of peripheral nerves. The medullary sheath becomes irregularly thickened, then undergoes granular and fatty degeneration, and is finally absorbed. The axis cylinders become at first much swollen, so that they may attain two or three times their normal dimensions, but when they are viewed longitudinally they are seen to present spindle-shaped enlargements. After the disappearance of the medullary sheath the naked axis cylinders may often be observed for a considerable time longer, but after a time they also undergo atrophy and disappear, so that nothing remains but a dense fibrillated connective tissue. The ganglion cells are cloudy and swollen at first, but after a time they undergo various forms of atrophy and vacuolation.

(3) *Changes in the Vessels.*—The walls of the small arteries and veins, and probably of the capillaries also, become thickened, while their calibre is lessened. The walls of the vessels are

often adherent to the indurated connective tissue, so that the lymph spaces are destroyed. In other cases the perivascular spaces contain collections of fat and pigment granules, and occasionally granule cells also. Granule cells and corpora amylacea are observed scattered through the diseased tissue.

§ 491. *Morbid Physiology.*—The connection between the morbid lesions and the symptoms is the same in the chronic as in the acute varieties of myelitis, so that it is unnecessary to repeat what has already been said with regard to the morbid physiology of the disease.

§ 492. *Varieties of Chronic Diffused Myelitis.*

The following varieties of chronic diffused myelitis may be distinguished:—1, Chronic central myelitis; 2, Chronic transverse myelitis; 3, Universal progressive myelitis; 4, Chronic bulbar myelitis; 5, Chronic myelo-meningitis; and 6, Chronic disseminated myelitis or multiple sclerosis.

1. Chronic Central Myelitis.—(*a*) *The subacute general spinal paralysis* of Duchenne is probably a typical example of inflammation of the central grey column, with extensions of the process into the anterior horns, and pursuing a gradually ascending course. Chronic atrophic paralysis is, indeed, closely similar in its symptoms to Landry's paralysis, of which it may be regarded as the chronic form. Hallopeau has shown that this form of paralysis sometimes pursues a descending course. In these cases the upper extremities, especially the muscles of the forearm, become first paralysed; they undergo atrophy soon afterwards, and the hands assume deformed postures. It is often seven or eight months or longer before either of the lower extremities are affected, and it may be several months longer before both of them are implicated in the paralysis. But even in these cases the disease pursues an ascending course as well, bulbar symptoms supervene after a time, and death results from paralysis of the respiratory centres.

(*b*) *Peri-ependymal Myelitis*—the "myélite péri-épendymaire" of Hallopeau—is another variety of chronic central myelitis. The symptoms are characterised by paralysis followed by diminution of the faradic contractility, and atrophy of the affected muscles.

The paralysis comes on somewhat suddenly; the patient finds that he is not able to move the fingers, hands, or more rarely the entire limb. After a time the muscles lose their faradic contractility and become atrophied, so that the affected extremities assume deformed positions. Fibrillary contractions are either absent or only present in a slight

degree. The patient may complain of vague pains along the vertebral column, but other sensory disturbances are usually absent. The sphincters also remain unaffected. The course of the affection is slow, and it may be temporarily arrested for a long time. The muscles most frequently affected are the flexors of the foot on the leg, and of the thigh on the pelvis in the lower extremities; and the extensors of the fingers and of the hand, then the muscles of the hand, and lastly the flexors and other muscles of the arm and shoulders. This affection may pursue a descending or an ascending course, and in the latter cases bulbar paralysis supervenes and death is caused by respiratory paralysis. Many cases of syringomyelia and hydromyelia are only forms of chronic central myelitis. Peri-ependymal myelitis is closely allied, on the one hand, with the subacute general spinal paralysis of Duchenne, and with progressive muscular atrophy on the other. And if we compare the symptoms of Landry's paralysis, subacute general spinal paralysis, peri-ependymal myelitis, and progressive muscular atrophy, it is impossible not to be struck with the essential unity which underlies them, while their differences are no less instructive.

All these diseases are characterised by a progressively-invading paralysis, which may pursue an ascending or a descending course, and by almost entire absence of sensory disturbances, bed-sores, and paralysis of the sphincters. The most striking differences between them are found in the time occupied in development of the symptoms, and in the course of the disease. Landry's paralysis is sudden in its onset and rapid in its progress; while, on the contrary, progressive muscular is gradual in its onset and slow in its progress, and the other two diseases occupy intermediate positions between these with respect to their development in time. In Landry's paralysis there is no decided muscular atrophy, and the faradic contractility is nearly normal; in subacute general spinal paralysis there is decided muscular atrophy, rapid loss of faradic contractility, and the reaction of degeneration; in peri-ependymal myelitis there is also pronounced atrophy, and the faradic contractility becomes slowly and gradually diminished, while in progressive muscular atrophy the atrophy and paralysis proceed usually side by side, and the faradic contractility is generally normal so long as any muscle remains.

We shall hereafter show that the morbid anatomy of these diseases tends to explain the differences just described, on the supposition that all are only different kinds of inflammation of the central grey columns of the cord, with anterior and lateral extensions of the disease into the anterior grey horns.

(*c*) *Chronic Central Dorso-Lumbar Myelitis.*—In this variety of the disease the symptoms correspond to the affection already described as acute central myelitis. The lower extremities are paralysed, the limbs are flaccid, the muscles undergo atrophy, and the faradic contractility of the paralysed muscles is soon lost; the reaction of degeneration is present, and the paralysis may gradually ascend to the muscles of the trunk and

those of the upper extremities. So far then the symptoms of this affection correspond to those of subacute general spinal paralysis of Duchenne, but other symptoms are observed in the former which do not occur in the latter. These symptoms are œdema of the lower extremities, paralysis of the sphincters, bed-sores, and arthropathies. Sensory disturbances also exist, consisting of various paræsthesiæ and hyperalgesia, followed by varying degrees of analgesia. If the myelitis be limited to the grey substance, tactile sensibility and the sense of locality and of temperature remain unaffected. If the lower portion of the cord be not implicated in the disease, the reflex of the sole may be exaggerated in the early stage of the disease. This form of myelitis is seldom if ever chronic from the commencement, and results usually as the sequel of an acute attack.

Morbid Anatomy.—It is unnecessary to say much at present with regard to the morbid anatomy of the chronic forms of central myelitis. It appears to me that Landry's paralysis, chronic atrophic spinal paralysis, peri-ependymal myelitis, progressive muscular atrophy, and what I have termed chronic central dorso-lumbar myelitis are only different forms of inflammation of the central grey column of the cord. In Landry's paralysis the inflammatory process is very acute, and keeps limited to the central column and the embryonic areas (the medio-lateral area in the dorsal and upper cervical regions, and the median areas and margins of the groups of ganglion cells in the lumbar and cervical enlargements) of the anterior grey horns. The muscles thus still maintain their connection with the fundamental cells, and their faradic contractility and nutrition remain comparatively unaffected. In chronic atrophic paralysis the morbid process pursues the same ascending or descending course, but the fundamental ganglion cells are invaded and destroyed, hence muscular atrophy and loss of faradic contractility result. Peri-ependymal myelitis pursues a somewhat similar course, but the fundamental cells are not invaded at so early a period as in the chronic atrophic variety. Progressive muscular atrophy pursues a still more chronic course. The disease, indeed, appears to be a parenchymatous one, and to spread from fibre to fibre and from cell to cell. It appears to begin in the small cells and fine fibres which lie near the central artery, and to spread gradually upwards and downwards and laterally. In its lateral extension the ganglion cells which were last developed become first affected, and the process gradually spreads to the more fundamental cells. It will be apparent that in such a gradual process as this the muscular paralysis and atrophy will pursue a parallel course, and that the faradic contractility will be maintained so long as the fundamental cells are able to perform their functions even imperfectly, and long after they have become partially diseased.

In the form of the disease which I have named chronic central dorso-lumbar myelitis the affection apparently begins as an acute central myelitis, and then assumes a chronic form. It pursues an ascending course, but is not so surely invading as Landry's paralysis and

chronic atrophic paralysis. The morbid process, on the other hand, extends backwards to the posterior grey horns, and thus gives rise to analgesia; but, if it remain limited to the grey matter, the tactile sensibility is comparatively unaffected. The sphincters also become paralysed, and bed-sores appear on the sacrum. Recovery may take place even after bed-sores have formed; but if the affection has become chronic, many of the muscles of the lower extremities remain permanently atrophied. The disease may extend in the lumbar region into the white substance; and if death supervenes, a transverse myelitis, with ascending sclerosis of the columns of Goll, and the direct cerebellar tract and descending sclerosis of the pyramidal tracts, will be found associated with the morbid appearances usually observed in central myelitis. Such were the conditions observed in a case under the care of Dr. Simpson, in which I conducted the post-mortem examination. The prominent symptoms during life were paralysis with flaccidity of limbs, muscular atrophy, and loss of the faradic and reflex excitability, ultimately associated with anæsthesia and paralysis of the sphincters. At no time did the symptoms indicate a spastic condition of the muscles. On microscopic examination the lower dorsal region of the cord was disorganised in the whole of its transverse diameter, there was ascending sclerosis of the columns of Goll, and of the direct cerebellar tracts, while the pyramidal tracts were sclerosed in the lumbar region. The central grey column was diseased from the conus medullaris up to the lower end of the medulla. In the upper dorsal and cervical regions, however, the fundamental cells appeared quite healthy, while the accessory cells had disappeared. The condition of the grey substance in this cord in the cervical region is, indeed, represented in *Fig.* 146.

In a case of what I must regard as in the main a central myelitis, under the care of Dr. Morgan, the morbid appearances discovered differed from those observed in Dr. Simpson's case. The patient under Dr. Morgan was run over and had his sciatic nerve injured. This was followed by paraplegia, characterised by flaccidity of limbs, and muscular atrophy. A few weeks after the accident spontaneous fracture of the neck of the right femur occurred. After a time there was anæsthesia of the lower extremities, paralysis of the sphincters, bed-sores, and death from pyæmia and peritonitis. The posterior columns of the cord could be seen with the naked eye to be gelatinous in appearance. On microscopic examination the presence of sclerosis of the posterior columns of the cord was confirmed. The portion of the posterior columns which adjoins the posterior commissure was healthy in the lumbar region, the whole of the columns were diseased in the dorsal region, but in the lower cervical region the portions which adjoin the posterior grey horns were healthy, and the healthy areas gradually increased in size until in the upper cervical region the sclerosis was limited to the columns of Goll. In addition to the sclerosis of the posterior columns, the central grey columns were found diseased throughout the entire length of the cord, accompanied with the

usual lateral extensions into the embryonic areas of the anterior grey horns. I have observed similar morbid appearances—posterior sclerosis and central myelitis—in a case of tumour pressing upon the cauda equina.

2. Chronic Transverse Myelitis—The symptoms vary according to the level at which the cord is diseased. This affection may be subdivided into (*a*) chronic dorso-lumbar, (*b*) chronic dorsal, (*c*) chronic cervical transverse myelitis, and (*d*) compression myelitis.

(*a*) *Chronic Dorso-Lumbar Transverse Myelitis.*—The paralytic symptoms are often preceded by girdle pains, formication, numbness, and various other paræsthesiæ, and by lancinating pains in the lower extremities. The patient often complains of muscular cramps, especially in the calves of the legs; he is soon fatigued, and the movements are stiff and constrained. After a time the lower extremities become rigid by contractures, the gait assumes the spastic form, and both the superficial and deep reflexes are exaggerated. If the lesion be situated below the origin of the sixth lumbar nerves the patellar-tendon reflex is abolished. The sensory disturbances also increase, the patient cannot feel the floor, and he finds it necessary to direct his eyes to the ground; tactile sensibility and the sense of locality and temperature are generally impaired, while in advanced cases there may be retardation of sensory conduction and analgesia. The general health may be unaffected for a long time, but by-and-by the paraplegia becomes complete, and the various forms of sensibility become more profoundly affected, so that at last there may be complete anæsthesia of all forms of sensibility. Muscular atrophy, accompanied by loss of the reflex and faradic contractility, may now supervene, the sphincters are paralysed, bed-sores form over the parts exposed to mechanical pressure, and the patient dies from pyæmia and marasmus.

(*b*) *Chronic Dorsal Transverse Myelitis.*—In this form both the sensory and motor paralysis extends higher than in the dorso-lumbar variety. The condition of the lower extremities with regard to paralysis, contractures, spastic walk, and exaggerated reflexes is the same as in the dorso-lumbar form of the disease. When the lesion is situated in the upper portion of the dorsal region, the muscles of the back and abdomen are involved in the disease. The paralysis of the abdominal muscles renders urination, defecation, and forcible expiratory acts difficult, so that the patient is much troubled by constipation, and he cannot clear the bronchial tubes thoroughly from mucus. Death is therefore liable to be caused by slight bronchial catarrh. Valuable information may be obtained with regard to the level at which the cord is diseased by an examination of the cremasteric, gluteal, abdominal, epigastric, dorsal, and scapular reflexes. Erb has recently described a case in which a spontaneous subacute dorsal transverse myelitis was preceded by double optic neuritis.

(*c*) *Chronic Cervical Transverse Myelitis.*—The initial symptoms now generally begin in the upper extremities which become paralysed some

time before the lower extremities. As the disease increases all the four extremities become paralysed. The muscles of the upper extremities after a time become atrophied and lose their reflex and faradic contractility, while those of the lower extremities are in a state of contracture, their faradic contractility being preserved and the superficial and deep reflexes increased. Various oculo-pupillary symptoms are also commonly present. When the upper cervical region of the cord is diseased, all four extremities are affected, but the nutrition of the muscles of the upper as well as those of the lower extremities remains unaffected, and their reflex activity is increased. The diaphragm becomes paralysed, and the patient suffers from dyspnœa, impairment of speech, vomiting, and hiccough.

Morbid Anatomy.—The morbid anatomy of chronic transverse myelitis varies according to the level at which the cord is affected. At the level of the principal focus of disease both the grey and white substances are affected, and this portion of the cord may be softened instead of being in a state of sclerosis. Above the level of the main lesion the columns of Goll and the direct cerebellar tracts undergo ascending sclerosis; while the pyramidal tracts undergo descending sclerosis below the level of the lesion. If the lesion is situated low down, the ascending sclerosis may be limited to the columns of Goll and the descending to the pyramidal tracts of the lateral columns. If, on the other hand, the lesion be situated high up in the cord, both the columns of Goll and the direct cerebellar tracts are affected with ascending sclerosis, and the pyramidal tracts and the columns of Türck with descending sclerosis. In addition to these changes transverse myelitis is frequently accompanied by central myelitis, which may extend up into the medulla oblongata. Near the main lesion the central myelitis may extend forwards so as to destroy all the ganglion cells of the anterior horns, but in the upper portion of the cord and in the medulla oblongata the accessory ganglion cells are alone destroyed, while the fundamental cells remain more or less healthy.

(*d*) *Compression Myelitis.*—This is a very common form of myelitis, and may occur along with any disease of the vertebral column or membranes which causes a slow compression of the cord. The symptoms of this affection correspond in the main with those of spontaneous transverse myelitis. The characteristic feature of the affection is the existence of symptoms which indicate local disease of the cord for some time previous to the development of definite paraplegia. The most usual of these symptoms are severe pains along the course of nerves which issue from a particular level of the cord, cramps in the muscles supplied by those nerves, local paralysis and muscular atrophy, local hyperæsthesia or anæsthesia, and pain and stiffness of the back.

Morbid Anatomy.—The morbid anatomy of compression myelitis is the same as that of the transverse varieties. The structure of the cord is destroyed at the level where the compression has been applied; while there is ascending sclerosis above the level of the lesion, and descending sclerosis below it. Central myelitis is frequently present also.

3. Universal Progressive Myelitis.—In this form of the disease the myelitis gradually extends until it involves the whole breadth of the cord. It is characterised by progressive weakness of the muscles of the lower extremities followed by complete paralysis. The disease generally begins in the lower extremities, and pursues an ascending course; but occasionally it begins in the upper extremities, and then its course is descending. There may at first be contracture of the muscles, and this is followed by gradual atrophy as the grey substance becomes involved. The reflexes and the faradic contractility also gradually diminish and ultimately disappear. There may be more or less violent pains in the back, trunk, and extremities, the patient complains of paræsthesiæ and dysæsthesiæ, and after a time there is complete anæsthesia, paralysis of the sphincters, and bed-sores.

Morbid Anatomy.—In the lumbar and lower dorsal regions the entire transverse area of the cord is diseased, but in the upper dorsal and cervical regions the sclerosis may be limited to the columns of Goll and the direct cerebellar tract, associated sometimes with more or less of chronic central myelitis.

4. Chronic Bulbar Myelitis.— The most characteristic form of chronic bulbar myelitis has already been described as *chronic progressive bulbar paralysis*, but cases of bulbar paralysis are occasionally observed which do not possess a distinctly progressive character. When the latter class of cases is caused by a bulbar myelitis, the affection is probably in all cases produced by the extension of inflammation from morbid changes taking place in the neighbourhood of the grey nuclei of the medulla oblongata. Inflammation of the bulbar nuclei may in this manner be set up by tumours of the neighbouring structures, or they may be involved in the inflammatory zone which frequently surrounds hæmorrhagic foci and centres of necrotic softening.

5. Chronic Myelo-meningitis.—This form of myelitis has been termed peripheric or cortical myelitis by Vulpian, because it consists of a chronic inflammation of the cord extending inwards from the pia mater. It is not easy to recognise this form of myelitis during life; but its presence may be suspected when the symptoms of chronic meningitis are accompanied by an unwonted degree of both sensory and motor paralysis. The paralysis is generally of the spastic variety and muscular atrophy does not occur. It may, however, be assumed that, should the anterior roots become involved in the disease, muscular atrophy will inevitably take place. In some cases the posterior columns are more involved than the lateral tracts, and then ataxic symptoms predominate over those of paralysis.

Morbid Anatomy.—The most characteristic feature of the morbid anatomy of this affection is that the affected portion of the cord is surrounded more or less completely by a ring of sclerosis. Sometimes, however, the sclerosis appears to spread inwards in the posterior columns to a greater

extent than elsewhere, while at other times the anterior are affected to a greater degree than the posterior or lateral columns.

6. Chronic Disseminated Myelitis.—As this disease is not usually limited to the spinal cord, but extends as a rule to the cerebrum and cerebellum, its description will fall more naturally with the encephalo-spinal than the spinal diseases.

§ 493. *Diagnosis.*—Simple chronic transverse myelitis is characterised by slow development of paraplegia with relatively slight irritative motor symptoms, and by the presence of more or less marked sensory and vesical disturbances, contractures, increased reflexes, and bed-sores. The affection is but slightly progressive in character, and runs a tedious course. Transverse myelitis can as a rule be readily distinguished from the systematic affections of the cord. In some cases of the latter, however, the lesion of one of the functional systems of the cord extends to neighbouring systems, and combinations are thus produced which are sometimes very difficult to distinguish from transverse myelitis. Numerous combinations of the system-diseases are possible, and every case of the kind requires separate study and a special diagnosis.

§ 494. *Prognosis.*—The prognosis is generally unfavourable in simple transverse myelitis. The affection is always severe and dangerous, and the most that can usually be hoped for is arrest of the morbid process with considerable loss of power in the lower extremities. Complete recovery is exceptional.

A slow progressive course must be looked for in the majority of cases, and the disease generally terminates in death in a few years. The prognosis will be determined by the ascending tendency of the disease, the occurrence of relapses, and by the presence or absence of cystitis and bed-sores.

§ 495. *Treatment.*—The treatment of the early stages of chronic myelitis must be conducted on the same general principles as are applicable to the acute varieties. So long as any active symptoms of irritation are present, the patient should maintain the recumbent posture, while all the usual precautions against the formation of bed-sores and cystitis must be

adopted. Ergot, belladonna, and the iodide of potassium are the internal remedies which have been found of most use. If syphilis be suspected, active antisyphilitic treatment must be employed. Nitrate of silver was first recommended by Wunderlich in the treatment of chronic myelitis, but it is probably of more use in locomotor ataxia than in any other variety of the disease. It should not be administered in cases associated with spasmodic rigidity of the muscles. Arsenic, phosphorus, and cod-liver oil may sometimes be found useful, but strychnine is positively injurious.

Counter irritation was at one time used too indiscriminately, and the results obtained were not very gratifying. The use of the milder counter-irritants, such as flying blisters, may be employed with advantage. Brown-Séquard praises highly the application of a hot douche, from 98° to 104° F., for two or three minutes at a time. The stream should be nearly an inch in diameter. The greatest reliance must be placed on baths, hydropathy, and galvanism.

Baths.—Erb strongly recommends the thermal brine baths (Rehme, Nanheim). The temperature should not be above 86°—78° F., and the immersion not too prolonged, and the water should not contain an excessive quantity of carbonic acid. Ordinary brine baths, chalybeate, and mud baths are much extolled by many authors.

Cold-water cure is very useful. All severe and strongly-exciting procedures, such as the use of water at a very low temperature, cold douches, and sharp slappings, should be avoided, and even wet packs of the entire body have proved injurious (Erb). Simple rubbing with wet-cloths, foot-baths, sponging the back, hip-baths, and local compresses to the back left on till they become warm, appear to be the most useful measures. The treatment should begin with baths of moderate temperatures (68°—77° F., never below 60°—53° F.), and should not be too prolonged. When an insufficient reaction follows a bath, and chilliness and discomfort are produced, the treatment should be discontinued.

The galvanic current is one of the most important therapeutic agents for the treatment of chronic myelitis. The electrodes must be applied differently according to the differences in the

position and extent of the forms of disease. It is best to let both poles act successively, either with a stabile or with a slowly labile current. The currents used should not be very strong, and each application should be of short duration. The treatment should be continued for months, being occasionally interrupted only to be recommenced after a brief pause. It is expedient to alternate or combine galvanisation with other methods of treatment. Some few cases do not bear well the application of galvanism, and with these the use of it must be discontinued.

The general management, the diet, and the mode of life of the patient are of the utmost consequence. Rest and a regular mode of life are essential. Over-exertion of any kind, mental as well as bodily, must be avoided. Sexual intercourse should be confined within the strictest limits or completely stopped. All excitement and violent emotions must be prevented as much as possible. The diet must be simple, nutritious, and easy of digestion, and cod-liver oil is often beneficial. Alcoholic beverages, coffee, tea, and tobacco should be used with great moderation. Residence in a mountainous region at a moderate elevation, or at the sea-side, will be useful; and it is advisable that the winters should be passed in the south.

When the patient is completely paraplegic, a wheeled chair may be used so as to permit the enjoyment of fresh air, and when patients are bedridden, care should be taken that the recumbent posture is not always maintained, but occasionally replaced by the lateral or abdominal position.

Pain must also be relieved by various means, the subcutaneous injection of morphia being the readiest and most effectual method. Other serviceable drugs are bromide of potassium, quinine, bromide of quinine, zinc, and valerian. Cutaneous irritation, electricity either in the form of the faradic brush or galvanic current, Preissnitz's compresses, applications of chloroform, and frictions with veratrine ointment and similar agents, often do good service. For the paralysis, atrophies, and anæsthesia, which persist after the disease has run its course, electricity is the best remedy.

(IV.) MYELOMALACIA.

§ 496. *Simple Softening of the Spinal Cord.*—Softening of the spinal cord may result from non-inflammatory processes.

§ 497. *Symptoms.*—The symptoms of simple softening are exceedingly obscure. The patient first complains of feelings of numbness and feebleness, generally of the lower extremities, which gradually increase in severity, until ultimately anæsthesia of the lower extremities and paraplegia are fully established; the reflex excitability is also gradually diminished and ultimately abolished. To these symptoms are added progressive paralysis of the sphincters of the bladder and anus, and in the final stage bed-sores, marasmus, and pyæmia.

Brown-Séquard and Hammond assert that at no time in the course of the disease are there any pains, hyperæsthesia, spasms, or increase of the reflex excitability.

§ 498. *Morbid Anatomy.*—The post-mortem appearances of simple softening are similar to those of white softening caused by previous inflammation. When fatty degeneration assumes special prominence, the softened spot may assume a yellowish colour, and present the appearances of yellow softening. The softened spot usually merges imperceptibly into the normal tissue. The microscopical characters are not well known; but it may be presumed that the nuclei of the neuroglia do not multiply to such an extent, and that the corpuscular elements and granule cells are less abundant in simple than inflammatory softening.

§ 499. *Morbid Physiology.*—The symptoms of simple softening are caused by the gradual destruction of the nerve elements without previous irritation. Softening is probably in all cases caused by disease of the vessels, thrombosis, and embolism.

§ 500. The *diagnosis* must rest mainly on the absence of all symptoms of sensory and motor irritation during the entire course of the affection. The *prognosis* of the affection is decidedly unfavourable when the centres of softening are at all extensive.

§ 501. *The treatment* must be carried on according to the rules laid down for the chronic forms of myelitis. Brown-Séquard recommends, besides iron and quinine, iodide of potassium in a bitter infusion, and strychnine used with the greatest caution, while he avoids ergot and belladonna. He also thinks that the cold douche or shower bath to the back is indicated, and maintenance of the dorsal position. The food should be abundant and nourishing, and when possible moderate exercise should be taken. The use of the galvanic current will be found advantageous.

CHAPTER VI.

III.—VASCULAR DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

(I.) ANÆMIA, THROMBOSIS, AND EMBOLISM OF THE SPINAL CORD AND MEDULLA OBLONGATA.

1. *Anæmia of the Cord.*

§ 502. Anæmia of the spinal cord consists of a diminution in the amount of blood contained in it. This condition may be due to causes special to the cord itself, and then it is called *spinal ischæmia;* or to general causes, such as oligæmia and hyrdæmia, and then it is called *dyscrasic spinal anæmia* (Jaccoud).

§ 503. *Etiology.*—The predisposing causes of spinal anæmia are congenital narrowness of the calibre of the vessels, weakness of the heart, and undue excitability of the vaso-motor nerves. The female sex is strongly predisposed to spinal anæmia. Diseases of the vessels of the cord, such as atheroma or the fibrosis which accompanies Bright's disease, also produce spinal anæmia. The exciting causes of the affection are arrested or diminished circulation, as may be produced by compression, thrombosis, or embolism of the abdominal aorta above the point of origin of the lumbar arteries.

Owing to the numerous anastomoses of the spinal arteries, thrombosis and embolism of one or more of them only lead to circumscribed ischæmia. It is probable that a considerable number of the so-called reflex paralyses are caused by a reflex spasm of the spinal arteries, and direct irritation of the vaso-motor conducting paths in the cord may likewise produce spasm of these vessels.

The cases of spinal anæmia which arise from general causes have been described by Jaccoud under the name of *paraplégies dyscrasiques.*

Spinal paralysis occurs only rarely in dyscrasic anæmia of the cord, but paraplegia has been known to follow great losses of blood, as in parturition, menorrhagia, and hæmorrhage from the kidneys and intestines; and weakness of the lower extremities is a not unfrequent symptom of chlorosis and severe acute diseases.

§ 504. *Symptoms.*—It has been proved experimentally in animals that, when the aorta is compressed, motor and sensory paralysis of the lower extremities immediately occurs, the reflex acts cease, and the bladder and rectum are paralysed. When the circulation is restored, improvement in the symptoms is slow in proportion to the duration of the compression. The same symptoms occur in man after embolism of the aorta. Gull observed paralysis to occur in a few minutes from embolism of the abdominal aorta in a case of aneurism. If the contraction of the aorta occurs gradually, the symptoms develop gradually, and are less severe in nature. They consist of a feeling of numbness and weakness of the lower extremities, which become easily fatigued on slight exertion. Nothing is known of the symptoms caused by ischæmia of the cervical region of the cord.

Vaso-motor ischæmia or anæmia from reflex irritation is characterised by the same kind of symptoms as those caused by aortic obstruction, although they are much less severe in the former than in the latter. In vaso-motor ischæmia some source of peripheral irritation can be discovered, and the paralytic symptoms generally disappear if the source of irritation can be removed. The intensity of the paralytic symptoms is said to vary in degree according to the severity of the peripheral irritation.

In the spinal anæmia produced by general causes, motor weakness, tremor on the slightest exertion, and in some cases complete paralysis are the most constant symptoms. These symptoms begin in the lower extremities, and extend to the trunk and arms. The sensibility is usually intact, but paræs-

thesiæ, pain, hyperæsthesia, and even slight anæsthesia may occasionally be present. The reflex actions are often exaggerated, and the sphincters are not, as a rule, affected. It is said that the symptoms improve on lying down, and, on the contrary, are made worse when the patient assumes the erect posture. The paralytic symptoms are associated with the usual signs of general anæmia or of chlorosis.

§ 505. *Course, Duration, and Termination.*—The disease may begin suddenly when it is caused by severe hæmorrhage or embolism, but the onset is more gradual when it results from thrombosis and chlorosis. The patient often recovers rapidly by the establishment of collateral circulation. At other times recovery is slow and gradual, and in cases of embolism the cord may undergo softening, so that recovery becomes impossible.

§ 506. *Pathological Anatomy.*—Anæmic portions of the cord look pale and bloodless. The grey substance is dull in colour, and sinks below the level of section; while the white is soft, and protrudes slightly above the surface of the section. The membranes are pale, and their vessels are empty. The anæmic portions contrast strongly in colour and consistence with those which are healthy. In thrombosis and embolism of the small spinal vessels it is often possible to find the point of occlusion. Red softening exists in the region supplied by the plugged artery and collateral fluxion in its vicinity. If the ischæmia be protracted, white and yellow softening of the corresponding portion of the cord may occur.

§ 507. *Diagnosis.*—The diagnosis must rest mainly on the concomitant symptoms. The symptoms of the acute ischæmic form resemble those due to spinal hæmorrhage, and anæmia can only be inferred to be the cause when the aorta is known to be obstructed or a great loss of blood has recently occurred.

The chronic forms of spinal anæmia resemble chronic myelitis or chronic meningitis, but when severe general anæmia exists it may be inferred that the disease is caused by it. The fact that the horizontal position relieves the symptoms may afford valuable aid in forming a diagnosis (Hammond).

§ 508. *Prognosis.*—Spinal anæmia is not a serious disease taken in itself, but in some of the severer cases softening may occur, and then the prognosis becomes unfavourable.

§ 509. *Treatment.*—The causes of anæmia of the cord must first be removed. This must be done by a tonic and stimulating treatment.

The patient should be laid on his back with his head and legs raised, and this position should be maintained in the night and for a considerable portion of the day.

Special stimulants of the cord itself may be administered, the most powerful and reliable of them being strychnine. The constant current should be applied daily to the vertebral column, especially in the form of the ascending stabile current. Warm applications should be made to the back, such as hot sand bags, or Chapman's spinal bags filled with hot water.

The diet should be generous and moderately stimulating.

2. *Anæmia of the Medulla Oblongata—Thrombosis and Embolism—Necrotic Softening.*

§ 510.—Anæmia of the medulla oblongata is generally accompanied by anæmia of the brain and spinal cord. Some of the symptoms, however, which occur in general anæmia are probably caused by anæmia of the medulla oblongata.

Thrombosis and embolism of the vessels supplying the medulla are not very rare, and the anæmia in such cases is so great that, unless the circulation be quickly restored, the part soon undergoes softening.

Thrombi and *emboli* generally occur in the vertebral and basilar arteries (§§ 353, 354). This subject has received much attention in recent years, and in consequence cases which were at one time classed together as apoplectic bulbar paralysis are now known to have been produced by embolism or thrombosis of the arteries of the medulla oblongata.

§ 511. *Symptoms.*—The symptoms of simple anæmia of the medulla do not require to be separately considered, as they are merged in the symptoms of anæmia of the cord.

The symptoms produced by obstruction of the arteries differ according as the main arteries or the small branches are affected.

The following general symptoms are commonly observed when one or both vertebral arteries, or the basilar artery, are obstructed by thrombosis or embolism. A more or less complete bulbar paralysis occurs suddenly or in a very short time, and without loss of consciousness. The soft palate and tongue are paralysed, the power of articulation and of deglutition is lost, and there is partial paralysis of the muscles supplied by the inferior portion of the facial nerve. These symptoms are sometimes accompanied by paralysis of the ocular and masticatory muscles, dulness of hearing, and noises in the ears. Respiratory, circulatory, and vocal disorders are also of frequent occurrence. Paralysis of one or all of the extremities is simultaneously developed. As a rule, a certain degree of anæsthesia is present.

If death from respiratory paralysis does not at once ensue, the disease is not of a progressive character, and at most only a slight change for the worse takes place during the first few days.

In some cases gradual improvement may take place, the paralysis partially disappears, the muscles of the extremities undergo various degrees of contracture, and the tendon reflexes are exaggerated, but life may be preserved for a comparatively long period.

§ 512. *Varieties.*

(1) *Obstruction of the basilar artery*, as a rule, produces bilateral symptoms, paralysis of all four extremities, and of both sides of the face. But the most important symptom is the cessation of the functions of the vagus and glosso-pharyngeal nuclei, causing severe respiratory disorders, dyspnœa, cyanosis, and usually a rapid death from asphyxia. A rapid and complete obstruction of the basilar artery generally produces profound coma and rapid death. If life last for a few hours complete paralysis of all four extremities is observed.

(2) *If the obstruction extend only to a small portion of the basilar*, or if the thrombosis be merely attached to the walls of the vessel and only cut off the blood from a few branches, the symptoms are often less threatening. Individual cranial nerves are paralysed, and there is weakness or paralysis of the extremities; some of the ocular muscles may be paralysed, as well as those supplied by the facial and trigeminal nerves. If, however, the

circulation continue in the posterior portion of the basilar artery and in the vertebral arteries, respiration is not arrested.

(3) *Simultaneous obstruction of both vertebral arteries* produces exactly the same effects as thrombosis of the basilar artery. The life of the patient is not in such immediate danger if the thrombosis be slowly developed so as to allow time for collateral circulation to be established. Joffroy attaches some importance to the lockjaw which has been observed in such cases.

(4) *Obliteration of one vertebral artery* produces symptoms which assume to some extent the hemiplegic form. The lesion is more frequent in the left artery, from which the anterior spinal artery is often exclusively or in great part given off. The inferior cerebellar artery is also obstructed.

The hemiplegia may either be on the same side as the lesion or on the opposite side, a matter that depends on variable conditions, such as the situation of the obstruction, the point of origin of the anterior spinal artery, the completeness of the decussation of the anterior pyramids, and other circumstances. The hypoglossal and spinal accessory nerves may be paralysed, causing disorders of articulation and deglutition and aphonia, and in addition there may be paralysis of the inferior branches of the facial, and partial paralysis and anæsthesia of the soft palate. These symptoms may be to some extent bilateral, but are generally more pronounced on one side of the body.

The successive occlusion of the different arterial territories may be recognised by the grouping of the symptoms and the order in which they follow one another.

The larger the vessel obstructed, and the more complete the occlusion, the quicker does death ensue. If the circulation can be quickly restored by the disintegration or displacement of the thrombus or embolus, or by sufficient collateral branches, improvement in the symptoms and partial recovery may take place.

(5) *Occlusion of the small arteries of the medulla oblongata* can never be diagnosed with certainty from the symptoms. But as these vessels are terminal their obliteration is surely followed by necrosis of the parts affected, and it only depends on the territory of such artery whether we get symptoms or not. Obstruction of the smaller vessels may produce partial paralysis of the tongue, difficulties of articulation and swallowing, unilateral paralysis of the facial and abducens nerves, respiratory disorders, and perhaps even fits of asthma.

(6) *Obstruction of the superior cerebellar artery* produces paralysis of the third nerve on the side of the occluded vessel and hemiplegia of the opposite side.

§ 513. *Morbid Anatomy.*—Either one or both vertebral arteries may be completely obliterated, and the thrombosis may extend from them into the basilar artery. The basilar artery

may be obstructed, either from one end to the other or for a short distance in its anterior, median, or posterior division, and any one or all of the branches given off from the main arteries may be partially or wholly obstructed by thrombosis.

Most of the arteries of the medulla oblongata are terminal arteries, hence obstruction of one of them always occasions grave disorders of nutrition. The immediate result is to produce intense anæmia in the *vascular territory* affected. The second stage consists of engorgement, accompanied by effusion of blood (hæmorrhagic infarct), which leads to red and afterwards yellow and white softening. The final stage often consists in the formation of a cavity of variable size in the medulla. The walls of the cavity are formed of a soft reticular connective tissue, while delicate threads of the same traverse the interior.

If the obstruction take place in one of the smaller vessels, a number of small wedge-shaped centres of softening or hæmorrhagic infarcts are found, having their apices directed forwards and their bases towards the floor of the fourth ventricle.

§ 514. *Diagnosis.*—When a bulbar paralysis, accompanied either by hemiplegia or paraplegia of the extremities, occurs suddenly, our attention should at once be directed to the pons and medulla oblongata. In fulminant and extremely rapid cases, when the patient sinks into deep coma and universal paralysis sets in with threatening asphyxia, we can only make a guess with regard to the nature and situation of the lesion.

Even when it is possible to determine that the lesion has occurred in the medulla oblongata, it is not always easy to decide whether a hæmorrhage or an embolus has taken place, and the question must be determined from general considerations. When the symptoms come on, one after another and not all at once, obstruction of a vessel must be presumed. Epileptoid convulsions are more frequent in hæmorrhage than in embolus and thrombosis. It is only when the basilar is completely occluded that we meet with a severe apoplectic attack.

The symptoms of embolism are often of a definite character, owing to the regular distribution of the vessels; whilst those of apoplexy are more dependent on chance, so that we meet with

a repetition of exactly the same group of symptoms more frequently in cases of embolus than we do in extravasation.

Striking and rapid improvement, with total disappearance of complete groups of paralytic symptoms, seldom occurs in cases of hæmorrhage. The frequent anomalous distribution of the vessels often renders it impossible to diagnose the particular artery which has been obstructed. Other symptoms may help us to a diagnosis. An unusually full pulse in the carotids is said to point to obstruction of the basilar artery.

§ 515. *Prognosis.*—The prognosis is always of the gravest character, and sudden and complete obstruction of the basilar or of both vertebral arteries is almost invariably fatal. A slowly-developing occlusion of one or more of the large vessels in this region also terminates in death within a short period. Life may be prolonged and partial recovery take place in cases of relatively limited obstruction which happen to affect the least dangerous parts of the medulla, or when a considerable collateral circulation is established.

§ 516. *Treatment.*—Stimulants and tonics are plainly indicated when one of the bulbar arteries are obstructed; but unfortunately the diagnosis is so uncertain in many cases that it is difficult to follow out any definite course of treatment. At a later period electricity may be applied with the greatest hope of success.

(II.) HYPERÆMIA AND HÆMORRHAGE OF THE SPINAL CORD AND MEDULLA OBLONGATA.

1. *Hyperæmia of the Spinal Cord and its Membranes.*

§ 517. It is impossible to separate hyperæmia of the spinal cord and of the spinal membranes, either clinically or anatomically, hence the two must be considered together. By hyperæmia of the cord and its membranes, therefore, is understood an increased supply of blood in the structures contained within the vertebral canal.

§ 518. *Etiology.*—Hyperæmia of the cord is produced by excessive functional activity, such as occurs in severe exertion

or violent sexual excitement. Congestion is also present in the early stages of inflammation of the cord, so that all the causes of myelitis are likewise causes of hyperæmia of the cord. Hyperæmia of the spinal cord may also be caused by various toxic agents, such as strychnia, carbonic oxide, and alcoholic excess. Exposure to cold is probably the most common cause of spinal hyperæmia, but it may be produced by the suppression of accustomed discharges, and by concussion and traumatic injuries of the vertebral column, while it is a frequent sequel of the specific fevers and malarial infection.

Passive hyperæmia is generally caused by the conditions which favour general venous congestion or stasis, such as diseases of the heart and lungs, or diseases like tetanus and eclampsia.

§ 519. *Symptoms.*—The most prominent symptoms of spinal hyperæmia are pains in the loins and along the spine of a dull, oppressive character, which are not increased on pressure. The patient complains of tingling, formication, and tearing pains in the lower extremities, and a slight degree of hyperæsthesia of the skin, a girdle sensation, and a moderate increase of reflex activity may be present. The motor symptoms consist of transitory jerking of the muscles and trembling of the limbs, and Rosenthal says that the electrical excitability is increased.

Depressive symptoms may likewise appear at an early period of the disease. A sensation of numbness is felt in the lower extremities and there may be also a slight degree of anæsthesia.

The patient complains of fatigue on slight exertion, the limbs feel heavy and feeble, but it is probable that complete paraplegia never occurs in simple hyperæmia.

Paresis of the bladder is rare, but has been occasionally met with, and Hammond has observed erection of the penis.

The symptoms are almost always bilateral and are limited to the lower half of the body, or at least they begin in the lower extremities. Occasionally the affection may extend to the upper extremities, and in those cases the respiration is said to have been disturbed.

Brown-Séquard says that the symptoms are aggravated by lying on the back, with the head and legs raised, while they

are relieved by lying on the face or by standing or walking, and the patients are also said to feel worse in the morning while in bed. When serous effusion has taken place, it collects in the lower part of the spinal canal on the patient assuming the erect posture, and consequently the patients are then better in the horizontal position. There is no fever, and the pulse may be either quick or slow if the hyperæmia extend to the spinal centres of cardiac innervation. The general health suffers to a greater or lesser degree.

It is very difficult to distinguish between active and passive hyperæmia, but if the irritative phenomena predominate, it may be presumed that the case is one of active hyperæmia; and if depressive symptoms predominate, that it is one of passive hyperæmia.

§ 520. *Course.*—The development is at times sudden, and then the disease runs through an acute course, and is soon terminated. At other times the course of the affection is slow; it gradually increases in severity, and then continues for days, weeks, or months, with varying degrees of intensity. The disease generally ends in recovery, but relapses are not rare. Recovery is often favoured by critical hæmorrhages. It is probable that simple hyperæmia never ends fatally.

§ 521. *Diagnosis.*—The diagnosis of hyperæmia of the structures within the spinal canal is based on the slight and transitory nature of the sensory and motor disturbances, and upon the frequent and rapid changes in the symptoms, the absence of increase of temperature, the short and favourable course of the symptoms, and the success of treatment calculated to relieve congestion.

Concussion of the cord may be distinguished from hyperæmia by the history of an injury.

In spinal meningitis the prominent symptoms are spasm of the back and neck, pain on moving the limbs, and high fever—symptoms which are absent in simple hyperæmia.

Acute myelitis may be recognised by the presence of fever, severe paralytic symptoms, contractures, paralysis of the bladder, and bed-sores.

Spinal apoplexy begins suddenly, the paralytic symptoms are severe, and more or less lasting.

Spinal anæmia is distinguished from hyperæmia by the fact that lying on the back gives relief to the symptoms of the former, while they aggravate those of the latter.

§ 522. *Prognosis.*—The prognosis is on the whole favourable; but the affection, if unchecked, is liable to cause hæmorrhage or inflammation of the cord, and then the prognosis becomes serious.

§ 523. *Morbid Anatomy.*—After death the arteries contract and empty their contents into the veins, so that even when active hyperæmia has existed during life it may occasion no characteristic appearances after death; while, on the other hand, vessels may become much congested in consequence of a prolonged death struggle, or of hypostatic congestion after death. In active hyperæmia the grey substance often appears of a rose or scarlet colour, and the white substance of a reddish tinge, the blood-vessels are tortuous and congested, and on microscopic examination the smaller arteries and capillaries are seen to be enlarged and unusually distended with blood. In severe cases punctiform extravasations and ecchymoses may be seen dotted over the membranes, and in the substance of the cord. The spinal fluid is usually increased in quantity, and is of a muddy or reddish colour (Erb).

In passive hyperæmia the extra-meningeal plexuses of veins are especially distended with blood; the individual veins are enlarged and tortuous, and the whole cord presents a dark blue colour. The spinal fluid is almost always increased, and ecchymoses may also be present.

In chronic and frequently-repeated hyperæmia the pia mater and arachnoid are thickened and opaque, and highly pigmented.

§ 524. *Treatment.*—The most favourable cases of active hyperæmia are those which arise from the suppression of a habitual discharge or from exposure to cold. The patient should be directed to lie on the side or face, with the extremities as low as possible. Venesection may be practised in robust

persons when the symptoms are violent, but as a rule it is preferable to draw blood by leeching. The leeches may be applied to the region of the spinal column, anus, vagina, or cervic uteri, according to the nature of the case.

I have seen an aggravated case of spinal hyperæmia recover in a few days under the care of my colleague, Dr. Simpson. The treatment consisted in the application of Chapman's ice-bags to the spine, while the patient was ordered to lie with his face directed downwards. The application of cold affusions and douches, cold packing, and sea baths may take the place of the ice-bag.

A saline purgative may be of use by unloading the vessels and lowering arterial tension. Ergotin and belladonna are the favourite internal remedies, but it is very doubtful how far they are of use.

The diet should be plain and nourishing, but unstimulating, and everything which might increase the hyperæmia, such as sexual and alcoholic indulgence, must be avoided.

2. *Hæmorrhage into the Substance of the Spinal Cord.*

Hæmatomyelia—Spinal Apoplexy.

§ 525. *Definition.*—Spinal apoplexy consists of hæmorrhage into the substance of the cord.

§ 526. *Etiology.*—Contrary to what occurs in cerebral disease, spinal apoplexy is more common in youth and middle age than in old age. Men are more subject to the affection than women. Chronic affections of the cord are often brought to a sudden close by hæmorrhage, owing doubtless to coincident changes occurring in the walls of the vessels. Amongst the exciting causes are traumatic injuries, active congestion of the cord, and anything that produces a want of equilibrium between the pressure within and without the blood-vessels. It is doubtful how far excessive cardiac action in the absence of degeneration of the vessels can give rise to hæmorrhage. Hæmorrhage may occur in connection with tumours, or any morbid process which occasions softening of the substance of the cord.

§ 527. *Symptoms.*—The attack generally begins suddenly

with fulminant symptoms. The patient complains of violent pains, and becomes suddenly paraplegic, but without loss of consciousness. The disease may be preceded by premonitory symptoms, consisting either of those indicative of spinal congestion or of the symptoms which precede acute central myelitis, and these may last from a few hours or several days. But even in the cases in which the affection is preceded by premonitory symptoms, the onset of the characteristic symptoms is always sudden, and complete paraplegia develops in the course of a few minutes, or at most an hour. During the development of the paralysis the patient complains of violent pain, either localised or extending over the entire spinal column, but usually disappearing when the paralysis has become complete. When the cervical region is implicated, the paraplegia extends to the upper extremities, the respiratory muscles are affected, and the patient breathes laboriously by the aid of the diaphragm.

The paralysed muscles are flaccid, and more or less complete anæsthesia of every form of cutaneous sensibility having the same distribution as the motor paralysis is present. Paralysis of the rectum and bladder occurs; at first there is retention of urine and afterwards various degrees of incontinence.

Vaso-motor disturbances are generally present. Levier found the temperature of the paralysed extremities, as compared with that of the axilla, increased from 0·2° to 2°0 C.

The reflex actions vary much according to the seat of the lesion. When the grey matter is infiltrated down to its lowest point, they are completely abolished. When the seat of the hæmorrhage is higher up, reflex actions disappear for a short time, owing to the shock; but they may afterwards reappear in an exaggerated form. In most cases, however, the reflex actions disappear after a time. Priapism is mentioned as a symptom in a few cases. The paralysed muscles undergo atrophy, they lose their faradic excitability, and manifest the reaction of degeneration. At a later period, when secondary changes occur in the cord, a few of the muscles may become rigid and contracted. The symptoms of secondary myelitis may be superadded and give rise to violent pains, twitching movements and jerkings of the extremities, and the formation of contractures.

Throughout the whole course of the disease symptoms of motor irritation are almost entirely absent. During the first moments of the hæmorrhage slight muscular twitching and partial spasms may occur, but these phenomena soon give place to paralysis. Spasmodic symptoms may also occur at a later period, but they are caused by secondary degeneration. Tingling and other paræsthesiæ may occasionally be felt in the paralysed parts, but, as a rule, these symptoms are wholly absent, and the patients do not feel their limbs.

After a few days or weeks, according to the severity of the case, gangrenous bed-sores appear on the sacrum, trochanters, heels, and other places exposed to pressure. Paralysis of the bladder leads to alkalescence of the urine, cystitis, pyelitis, and their sequelæ, and the patient dies in a state of great marasmus.

The symptoms, of course, vary greatly according to the cause, extent, and situation of the hæmorrhage. In small hæmorrhages the symptoms are so destitute of any distinguishing features as to render the diagnosis a matter of great uncertainty. When the hæmorrhage is limited to the anterior cornua, the symptoms produced will be mainly those of local paralysis; while if it be limited to the posterior cornua, the symptoms will be extremely indefinite.

If the *lumbar region* of the cord be affected, the symptoms of paralysis and anæsthesia are restricted to the lower extremities, bladder, and rectum; reflex actions are absent, and rapid atrophy of the muscles and bed-sores occur at an early period of the disease.

If the *dorsal region* be affected, the paralysis extends higher up. The expiratory muscles and those which compress the abdomen are paralysed, but reflex actions may be retained for a long time, and atrophy of the muscles is slow.

If the *cervical region* be implicated, all the four extremities are affected, a portion of the inspiratory muscles are paralysed, oculo-pupillary symptoms may be present, and the implication of the reflex processes and nutrition depend on the downward progress of the lesion. If the hæmorrhage occur above the origin of the phrenic nerve, rapid death by asphyxia is inevitable. In a few cases the hæmorrhage has been found limited to one half of the cord.

§ 528. *Course, Duration, and Termination.*—The course depends on the cause of the disease and the extent and locality of the hæmorrhage. In severe cases of diffuse central bleeding a fatal termination occurs soon through paralysis of respiration, or death results from acute bed-sores, pyæmia, and septicæmia. If the hæmorrhage be small the case may be very protracted, but death ultimately results from bed-sores, cystitis, fever, marasmus, and other complications.

The lesion in the cord sometimes cicatrises, and partial recovery occurs even after cystitis and bed-sores have made their appearance; but in these cases some muscles or group of muscles usually remain paralysed and atrophied. Complete recovery is only possible when the clot is small.

The duration of the disease varies greatly. Rapid cases terminate in a few minutes, hours, or days; while in less severe ones, weeks, months, or even years may elapse before death occurs.

§ 529. *Morbid Anatomy.*—The bleeding is generally limited to the grey substance, and may involve the cornua or the entire grey substance, and may extend to various distances longitudinally. Two kinds of extravasation may occur: the hæmorrhagic or apoplectic clot, and the hæmorrhagic infiltration or softening.

The hæmorrhagic or apoplectic clot varies in size from that of a pea to that of a hazel-nut, and its longitudinal is generally longer than its transverse diameter. The clot is often seen through the pia mater as a bluish nodule, while the pia is sometimes ruptured, so that blood makes its way into the subarachnoidal space. The clot is surrounded by ragged walls formed by disintegrated nerve tissue. The hæmorrhage may pass for a considerable distance between the bundles of white fibres, and a large portion of the grey substance may be destroyed, giving rise to what is called a "tubular hæmorrhage." The portions of the cord most usually affected are the cervical and upper dorsal regions.

The clot after a time undergoes a series of further changes. It either slowly dries up to a crumbly, caseous mass of a dark colour, containing crystals of hæmatoidin, or undergoes a pro-

cess of softening, with subsequent absorption, so that at last a capsule of connective tissue is left, filled only with a serous fluid. When the extravasation is small it may be absorbed, so as to leave only a small cicatrix of connective tissue.

Secondary disease of the cord is very often found in the neighbourhood of the clot. This generally consists of softening, which extends to a variable distance both upwards and downwards. The grey matter often undergoes hæmorrhagic softening, and is sometimes converted into a softened mass of a reddish black or chocolate colour, while white softening may be observed in the neighbourhood of the clot. In old-standing cases secondary ascending and descending sclerosis occurs.

Hæmorrhagic infiltration or softening occurs in the grey substance exclusively. It is limited to one or more of the grey horns, or extends over the whole of the grey matter, but rarely spreads to the white substance. It may extend longitudinally a few centimetres only, or the whole length of the cord. The grey substance is changed into a reddish-brown mass dotted with dark red points. The microscope shows elements like those in the clot, but with the addition of granular corpuscles and degenerative changes in the nerve fibres and ganglion cells.

The usual evidences of acute central myelitis may be observed far beyond the limits of the hæmorrhagic infiltration.

§ 530. *Diagnosis.*—The diagnosis is chiefly based on the sudden and very rapid invasion of paraplegia without much irritation, and the immediate severity of the symptoms. It is distinguished from *cerebral apoplexy* by the retention of consciousness, the absence of all symptoms of paralysis of cerebral nerves, the paraplegic form assumed by the paralysis, and by the presence of paralysis of the sphincters.

In *meningeal hæmorrhage* there are active symptoms of irritation, hyperæsthesia and pain, violent spasms, while paralysis is less prominent, the disturbances of sensibility are slight, and the course of the attack is rapid and comparatively favourable.

Acute central myelitis is very similar in its symptoms to spinal apoplexy. In myelitis the paraplegia requires hours or days for development. It begins with symptoms of irritation, such as pain and slight spasm, the vertebral column is sensitive

to pressure, fever may be present, and anæsthesia and paræsthesiæ are prominent symptoms, while partial paralysis and weakness of the bladder precedes the occurrence of severe paraplegia. The ascending progress of central myelitis may be contrasted with the stationary nature of the symptoms in hæmorrhage.

Poliomyelitis anterior acuta in adults is often like hæmatomyelia. It may be distinguished by the presence of fever at the commencement, the absence of sensory disturbances, and of palsy of the bladder and bed-sores.

The ischæmic paraplegia caused by embolism of the aorta can alone be mistaken for hæmorrhage, and this accident may be recognised by absence of the femoral pulse and other attendant symptoms.

§ 531. *Prognosis.*—The prognosis is always grave. Large central hæmorrhages and those which are seated at a high level are necessarily fatal. The prognosis becomes more hopeful if the first few days and weeks pass without bringing severe complications, but complete recovery is rarely to be expected. Small circumscribed hæmorrhages are less dangerous, but it is rare that the diagnosis of such cases amounts to anything like certainty.

§ 532. *Treatment.*—Prophylactic treatment should be adopted, such as removing retained or suppressed menses, or alleviating heart disease and congestion of the cord. When the symptoms are related to a central myelitis, a very active antiphlogistic treatment should be adopted. Chapman's ice-bag should be applied to the back, but after the acute stage is over a more stimulating treatment may be adopted. For the hæmorrhage itself little can be done beyond relieving the congestion of the cord by the employment of local bleeding, free application of ice, and maintenance of the horizontal position upon the side or abdomen, with the internal use of digitalis or ergot, purgatives, and application of warmth to the extremities.

Trophic disturbances, cystitis, and bed-sores must be subjected to the usual treatment. If the first weeks pass without

serious results, iodide of potassium may be administered to promote absorption. Warm spring and brine baths, or a moderate hydropathic treatment, and the constant current may all be occasionally of use.

3. *Hyperæmia and Hæmorrhage of the Medulla Oblongata.*

§ 533. Hyperæmia strictly limited to the medulla must be an extremely rare occurrence, and in the majority of cases it is nothing more than part of a general hyperæmia of the brain or spinal cord.

§ 534. *Hæmorrhage* of the medulla is more closely related to the vascular diseases of the brain than to those of the cord, and the frequency of the occurrence of hæmorrhage is greater in the cerebral than in the spinal end of the medulla. Rupture of a vessel is on the whole a rare occurrence in the pons and medulla oblongata. The pathology of hæmorrhage of the medulla is the same generally as that of hæmorrhage of the cord. Disease of the vessels, such as miliary aneurisms, atheroma, fatty degeneration, and capillary dilatation resulting from processes of softening, is the most important condition which leads to the production of hæmorrhage. Cases of atheroma and aneurism of the basilar artery are often accompanied by hæmorrhage from the smaller branches in the medulla and pons. Bright's disease is a very important cause of hæmorrhage into the medulla. Caries of the cervical vertebræ, purulent basilar meningitis, and tumours in or around the medulla predispose to hæmorrhages by impairing the nutrition of the walls of the vessels.

Hæmorrhage is also produced traumatically through injuries to the skull and back of the neck. Westphal produced capillary hæmorrhage in the medulla of the guinea-pig by light blows of a hammer on the head. In extensive cerebral hæmorrhage, which breaks through into the ventricles, the fourth ventricle often becomes filled with blood through the aqueduct of Sylvius.

§ 535. *Symptoms.*—Very little is known of the symptoms of active hyperæmia in the medulla, but it is probable that some of the symptoms of general cerebral hyperæmia are due to

congestion of the medulla. These symptoms are dyspnœa, slow pulse, vomiting, general convulsions, and certain defects of speech. Certain initial symptoms of acute bulbar disease, such as pains in the head and back of the neck, spasms in the face and tongue, and formication in the region supplied by the fifth, are probably caused by congestion of the medulla oblongata.

Hæmorrhage into the medulla, even of limited extent, is always exceedingly dangerous. It commences generally with the most alarming symptoms, and not unfrequently causes instant death. In these cases the patients fall down with a cry or in epileptiform convulsions, and die instantaneously. Large effusions of blood into the hemispheres and basal ganglia sometimes reach the fourth ventricle; they irritate and oppress the medulla, quickly producing death, preceded by vomiting, convulsions, coma, and general paralysis.

Slight hæmorrhage into the medulla generally produces symptoms of an alarming and very threatening nature, and these are more grave the nearer the effusion is to the respiratory centre, for when the latter is affected instant death ensues. In cases which survive a longer time the patients utter a loud cry, or are attacked by buzzing in the ears, dizziness, sudden headache, vomiting, or convulsive spasm of the body, followed by coma. Epileptiform convulsions have been observed amongst the initial symptoms of hæmorrhage into the medulla and pons.

The motor paralysis varies greatly in extent, sometimes attacking the lower extremities only, sometimes only the upper, and at other times causing hemiplegia. In most cases all four extremities are either completely or partially paralysed. Some of the bulbar nerves are always more or less affected. The hypoglossal, facial, spinal accessory, and trigeminus are usually more or less completely paralysed, and sometimes the nerves of the orbit also. When there is a hemiplegia, the paralysis of the extremities occupies the side of the body opposite to the extravasation, while paralysis of the bulbar nerves occurs on the same side, giving rise to a characteristic *hemiplegia alternans*.

Paralysis of sensation when present follows the same rule as that of motion, but is not usually so well developed. When coma is present, it is impossible to ascertain anything

with respect to the condition of sensation. When the affection is unilateral, the sensory disturbances are also crossed; but owing to the peculiar course of the sensory fibres in the medulla, we can hardly expect a sharply-defined anæsthesia.

Respiratory disturbances are the most important and characteristic symptoms of the affection. If fatal asphyxia does not ensue at once, the respiration is impaired, becoming irregular, stertorous, often intermittent, and accompanied with great dyspnœa. The Cheyne-Stokes respiration is frequently observed; the breathing then becomes more and more embarrased till death from asphyxia results. Alterations in the action of the heart are generally less prominent, but the pulse is usually frequent, irregular, and intermittent.

Vaso-motor changes have not been often described, but in the period immediately succeeding the hæmorrhage, unilateral or bilateral rise of temperature of the skin has been noticed. A considerable rise of temperature occurs during the death agony, as occurs in other forms of apoplexy.

Disturbances of speech and deglutition and unilateral or bilateral paralysis of the soft palate result from the participation of the bulbar nerves in the paralysis. Deafness and buzzing of the ears are also frequently observed from implication of the auditory nerves. Frequently recurring vomiting and a continuous troublesome hiccough have been observed as symptoms. Potain found *polyuria* present in one case. Mader and Desnos found albumen in the urine in a case where the kidneys proved to be normal at the autopsy.

In cases where life is prolonged the electric reactions remain normal in the extremities, but there may be loss of faradic contractility and the reaction of degeneration in the muscles supplied by the paralysed cranial motor nerves.

§ 536. *Course.*—The disease is either fatal at once from paralysis of the respiratory centres, or death does not result for a few hours or days, but there is general paralysis and profound unconsciousness; or life may be maintained for a considerable time. In the latter case the patient gradually recovers his consciousness, some of the paralytic and other symptoms disappear, and nothing remains but hemiplegia or

partial paraplegia, and more or less difficulty in articulation and deglutition. In such cases contractures are very liable to ensue, just as occurs when the pyramidal tract is interrupted in any other part of its course. Very little is known of the symptoms of small capillary hæmorrhages in the medulla, but they are probably similar to those produced by emboli of the nutrient arteries of the bulb.

§ 537. *Morbid Anatomy.*—Hyperæmia of the medulla is characterised by the same anatomical appearances as cerebral hyperæmia, and requires no further description. A similar remark applies to hæmorrhage of the medulla. A clot goes through the same changes in the medulla as in the brain, and unless it is rapidly fatal it ends in the formation of a contracted scar or of a small cyst. Secondary degenerations of the pyramidal tracts are generally developed. Extravasations are usually of small size, except when the pons is simultaneously affected, and then they may be large; they are roundish, resembling an olive or bean, but frequently irregular. Near the median line we meet with small triangular spots of hæmorrhage, with the apex pointing forward, corresponding to the territory of a median bulbar artery.

§ 538. *Diagnosis.*—It is probably impossible to make a special diagnosis of hyperæmia of the medulla oblongata. Loss of consciousness, epileptiform convulsions, and sudden death are sufficiently characteristic symptoms of severe cases of hæmorrhage into the substance of the medulla. In cases of less severity the onset may be attended by general epileptiform convulsions, vomiting, hiccough, more or less threatening respiratory disorders, dysphagia, disorders of speech, paralysis of the tongue and soft palate, of the inferior branches of the facial and of the abducens oculi, the presence of albumen and sugar in the urine, a final rise of temperature, the extension of paralysis to all four extremities, the unequal degree of paralysis in the extremities of one side and the face and tongue on the other side, and the abolition of all reflexes in the regions supplied by the paralysed bulbar nerves. It may be concluded that the lesion is limited to the anterior half of the floor of the ventricle

when we see paralysis of the abducens, facial, and trigeminus, along with aural disorders and sugar and albumen in the urine. Hæmorrhage in the posterior portion of the rhomboid sinus produces paralysis of the hypoglossus, facial, and trigeminus, and of the spinal accessory and vagus, accompanied by grave respiratory disorders and usually by paralysed extremities, and it is a symptom of some importance when these latter alternate with paralysis of the tongue. Alternate paralysis of an upper and lower extremity probably indicates that the lesion is situated in the centre of the decussation of the pyramids.

§ 539. *Prognosis.*—The prognosis is very unfavourable, and when the hæmorrhage is of large dimensions the lesion is invariably fatal. There is only hope in cases of very limited hæmorrhage, or when the localisation is very favourable, especially when it is far removed from the respiratory centres. The patient's condition may then improve gradually and partial recovery take place.

§ 540. *Treatment.*—The rules of treatment are the same for hyperæmia and hæmorrhage as for the same processes in other parts of the brain. Venesection, combined with active stimulants, is the most suitable treatment in severe cases when respiration is threatened; the latter must be injected per rectum, as the patient cannot swallow.

In chronic cases, when paralysis continues and when speech and deglutition are impaired, a suitable application of electricity is indicated.

CHAPTER VII.

IV.—FUNCTIONAL AND SECONDARY DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

(I.) SPINAL IRRITATION.

§ 541. *Definition.*—This disease generally occurs in the female sex, and is characterised by great irritability of the sensory functions, along with motor weakness.

§ 542. *Etiology.*—The female sex predisposes to the disease, although it occasionally occurs in men. The greater number of cases are met with between the fifteenth and thirtieth years, and more frequently in neuropathic constitutions.

Everything which weakens the nervous system may act as an exciting cause, as emotional disturbances, excessive bodily exertion, severe marches, sexual excesses, bad food, exhausting diseases, intoxication with alcohol or opium, traumatic agencies, and exposure to cold.

§ 543. *Symptoms.*—The disease generally begins with ill-defined pains in the back, especially between the shoulder-blades, the patient also complains of excentric pains, increased nervous irritability, and general loss of power, these symptoms gradually increasing in severity until the disease is fully developed. In some cases the development occurs quickly, it may be in a few days.

When the affection is fully developed the patient complains of a general feeling of illness, and displays increased mental irritability. One of the most prominent symptoms is pain in the back, situated in various spots, most frequently between

the shoulder-blades, in the back of the neck, and less frequently in the loins. The pain is aggravated by exertion, and the vertebral column is exceedingly sensitive to pressure, the slightest tap over some of the spinous processes calling forth expressions of pain. Certain spots are found to be very sensitive when a hot sponge or the cathode of a galvanic battery is passed along the spine. The patient complains of neuralgiform pains in various parts of the body. These pains are felt in the upper extremities, occiput or face, lower extremities, pelvic region, bladder, genitals, or viscera. Paræsthesiæ, such as tingling, formication, and feelings of heat or of cold, are frequently associated with the neuralgiform pains. Actual anæsthesia is seldom observed. The patient feels weary and exhausted on slight exertion, and walking soon becomes impossible owing to the intolerable pain caused by it. Most patients soon come to remain on their backs, and even movements of the upper extremities may be avoided owing to the pain occasioned. Real paralysis does not exist, although in a few cases a certain amount of paresis may be present.

Spasmodic symptoms in the form of fibrillary twitchings, spasms of some muscles, choreic movements, and hiccough are observed. Even permanent contractures and epileptic attacks are said to arise from spinal irritation.

Vaso-motor disturbances are frequent. Most patients suffer from coldness of the hands and feet, which are often cyanotic, and the patients easily turn red or pale, owing to undue irritability of the vaso-motor nerves.

Functional disturbances of the viscera are generally present. The most common symptoms are eructations, nausea, vomiting, palpitations, asthmatic breathing, spasmodic cough, vesical spasms with increased desire to urinate, and abundant discharge of pale urine, but actual paralysis of the bladder or rectum does not occur. Increased mental irritability and depression, along with sleeplessness, are almost constant symptoms. There are also noises in the ears, dizziness, and inability to read continuously owing to the occurrence of muscæ volitantes, and other disturbances of vision.

§ 544. *Varieties of Spinal Irritation.*—The disease may be

divided into three classes, according as the symptoms point to the upper, middle, or lower parts of the cord.

(1) When the *cervical* portion is affected, the pain and sensitiveness are localised in the cervical vertebræ, and the prominent symptoms are then referred to the head. These symptoms are giddiness, sleeplessness, disturbances of the special senses, pain in the occiput, and pains in the areas of distribution of the nerves of the brachial plexus. In addition to these nausea, vomiting, palpitation, and hiccough, and impairment of power in the upper extremities may be complained of.

(2) If the *dorsal* portion of the cord be affected, the symptoms are local tenderness of the dorsal portion of the vertebral column, intercostal neuralgia, gastralgia, nausea, dyspepsia, and motor and sensory disturbances in the lower extremities.

(3) When the *lumbar* portion of the cord is affected, the symptoms are neuralgia in the lower extremities and pelvic organs, spasm or paresis of the bladder, cold feet, and weakness of the legs.

§ 545. *Course, Duration, and Terminations.*—The course of the disease is usually fluctuating, and relapses occur without apparent cause. Some cases run a comparatively acute course, but the duration generally extends over a period of months or years, and some patients suffer from occasional attacks all their lives, although most of them ultimately recover.

Nothing is known with regard to the morbid anatomy of spinal irritation. It is probably a functional disturbance of the cord, accompanied by alternating conditions of hyperæmia and anæmia.

§ 546. *Diagnosis.*—Spinal irritation is very difficult to distinguish from hyperæmia of the cord. In severe hyperæmia distinct paralysis is rarely absent, and the duration of the disease is not as long as that of spinal irritation. Hammond says that strychnine injected subcutaneously does good in spinal irritation, and harm in hyperæmia. Spinal irritation resembles in some respects spinal meningitis, but in the latter there are stiffness and painful tension of the muscles of the back, and fever.

The first stage of meningeal tumours is very similar to spinal irritation, but in the former only deep pressure on the spinous processes is painful, and there is no circumscribed hyperæsthesia in the vertebral region.

It is impossible to diagnosticate spinal irritation from *hysteria* in many cases, and indeed the two affections have been regarded as identical.

§ 547. *Prognosis.*—The disease is always chronic and may last for months or years, but the prognosis is generally favourable, and life is never in danger, although a great deal of suffering is produced.

§ 548. *Treatment.*—The treatment of spinal irritation offers difficulties from the great mental irritability and changeableness of the patient.

The first endeavour must be to remove the cause of the diseases when this is possible. The next endeavour must be to improve the general nutrition, and to direct special treatment to the spinal cord. A tonic regimen must be adopted, a full and stimulating diet, as well as moderately free use of wine or even in some cases brandy or whisky. Active and passive exercise in the open air must be taken, but fatigue should be avoided, and the patient should frequently rest in the recumbent posture. The air of mountains and forests is useful, as well as a moderate hydropathic treatment.

The most useful remedies in the treatment of the affection are quinine, iron, zinc, and strychnine. The ascending stabile constant current passed through the vertebral column, including the painful portions between the poles, may be of service. Each sitting should be short, and the strength of the current moderate. The negative pole acting directly on the painful vertebræ has often done good. Many patients of this class are benefited by general faradisation and central galvanisation.

Counter-irritants applied directly over the painful portion of the spine often effect wonders. Various symptoms, such as neuralgiform pains, require treatment as they arise.

(II.) FUNCTIONAL WEAKNESS OF THE SPINAL CORD.

Neurasthenia Spinalis.

§ 549. *Definition.*—Neurasthenia spinalis is observed in persons who are subject to the general symptoms grouped under

the popular name of "nervousness," but in it the functions of the cord are affected in a special degree.

§ 550. *Etiology.*—The affection generally occurs in neuropathic families, and the male is more liable to be attacked than the female sex. Youth and middle age suffer most from the disease, and it is more common in the upper than in the lower classes. The exciting causes are excessive mental or bodily exertion, the depressing emotions, and sexual excess.

§ 551. *Symptoms.*—Patients complain chiefly of great weakness of the lower extremities, accompanied by an intense feeling of fatigue on slight exertion. A dull feeling of weariness is, indeed, often felt by the patient in the lower extremities in the morning before rising. After prolonged exertion this feeling may be accompanied by occasional tremors of the legs, and a remarkable stiffness and pain of the muscles of the lower extremities, similar to that produced in a healthy man by prolonged marching. Symptoms of rapid exhaustion and fatigue may be observed in the arms also, but never reach the same intensity as in the legs.

The sensory disturbances consist of pain in the back, which is aggravated by the movements of the muscles. The pain is not intense, and varies greatly in its time of occurrence and position. It is increased or brought on by slight exposure to cold, and by venereal and other excesses.

A diffused sensation of burning in the skin of the back is often observed, especially between the shoulder-blades, which is usually accompanied by sensitiveness of some of the spinous processes, as in spinal irritation. Neuralgiform pains may be present in the extremities; they are never of long duration, but often recur after unusual exertion. The patient also complains of numbness and formication, especially in the lower extremities, of cold hands and feet, and occasionally there is a burning feeling in the feet.

The sexual functions are generally more or less interfered with, there is diminished power of erection and premature ejaculation, and the act of coition is followed by remarkable prostration and restlessness of the limbs.

There may be a little dribbling of urine, but the functions of the bladder are usually normal. The patient is much troubled with sleeplessness, and feels particularly prostrate in the morning, he complains of a sense of constriction of the head, is self-conscious and timid, and manifests a strong tendency to shed tears. Vertigo is usually absent, and the special senses and higher mental faculties remain unaffected.

Dyspepsia, along with constipation, flatulence, and palpitation, is frequently present. The patients are generally hypochondriacal, and live in constant dread of tabes dorsalis, or some serious affection of the cord. The general nutrition is generally impaired, the patient loses flesh, acquires a sallow look, and becomes anæmic. There is always great sensitiveness to cold and changes of weather.

The objective symptoms are almost entirely negative. The closest examination reveals no trace of motor disturbances or want of co-ordination. The sensory disturbances are equally slight. There is no great sensitiveness of the spinous processes, the reflex functions of the skin and tendons are normal, there is no muscular atrophy, and no change in the electrical reactions of the muscles.

§ 552. *Course, Duration, and Termination.*—The disease may occasionally begin rapidly, but, as a rule, it develops gradually and increases in severity for weeks or months, and then remains more or less stationary. Slight fluctuations in the intensity of the symptoms are common. Under proper treatment the disease begins to improve, but months or years may pass before complete recovery occurs, and relapses are common. Intercurrent febrile affections often appear to influence the affection favourably. In some cases the patient is compelled to relinquish business on account of the affection.

§ 553. *Morbid Physiology.*—The simultaneous occurrence of sensory and motor disturbances of the legs, and the affections of the bladder and sexual organs, show that the disease is of spinal origin, while its favourable course, and the absence of the usual objective symptoms indicative of organic disease of the cord, show that it is a functional affection. It is probable that a

certain amount of anæmia of the cord exists combined with an irritable condition of the nervous tissue itself, leading to a ready discharge of nervous force and subsequent exhaustion. It may also be assumed that repair of the exhausted tissues does not take place promptly and rapidly as in health.

§ 554. *Diagnosis.*—The diagnosis will be based on the great disproportion between the acute subjective complaints of the patient and the almost negative result of objective examination. The diagnosis becomes clearer when in addition there exists general nervous weakness and sleeplessness, and the causes are present which induce the disease.

This affection might be mistaken for the early stage of tabes dorsalis, but in the latter the presence of the lancinating pains and other disturbances of sensibility, the girdle sensation, and especially the ataxic symptoms ought to render the diagnosis easy.

Nervous weakness of the cord may be distinguished from *active hyperæmia* by the absence in the former of pain, cutaneous hyperæsthesia, and symptoms of motor irritation. It may be distinguished from passive hyperæmia by the absence of paretic symptoms and by the feeling of heaviness in the legs.

It may be distinguished from *incipient myelitis* by the absence of paræsthesiæ and anæsthesia, of paresis or paralysis of the limbs and of pronounced weakness of the bladder.

From spinal irritation it may be distinguished by the fact that in the former the sensory disturbances, as dorsal pains, neuralgias, and sensitiveness of the spines of the vertebræ, are the most prominent symptoms, while fatigue on exertion and sexual weakness are the main symptoms of the latter.

§ 555. *Prognosis.*—The patient generally recovers after a time, when the causes of the affection are removed and a suitable treatment adopted. Relapses are, however, of frequent occurrence when the patient remains exposed to the exciting causes of the disease.

§ 556. *Treatment.*—Particular attention must first be directed to remove the exciting causes of the affection. Great attention

ust be paid to the regimen and diet of the patient. His ork should be light and agreeable, and he should retire to st at an early hour. His food must be nourishing and easily gestible. Alcoholic beverages may be allowed in moderation, nd open-air exercise, short of fatigue, should be enjoined. exual excess must be carefully avoided, although coition need ot be entirely forbidden.

With regard to the special treatment, a moderate hydropathic reatment has been found useful. Change of air to a mountainous district is also exceedingly useful in promoting recovery, Switzerland and the Tyrol being very suitable places.

An ascending stabile galvanic current, of moderate intensity, should be applied to the vertebral column. Iron, quinine, and strychnine are the most useful internal remedies. Chalybeate baths are useful for anæmic persons, but patients who are sensitive to cold should at first be sent to the thermal *brine* baths. Sea baths are useful in the after-treatment.

Such symptoms as sleeplessness, pain, spermatorrhœa, impotence, and digestive disorders must be treated in the usual way.

(III.) REFLEX AND SECONDARY PARAPLEGIA.

§ 557. It has long been known that paralysis of the lower extremities is frequently associated with genito-urinary diseases. These affections were at one time grouped together under the name of *urinary paraplegiæ*. Brown-Séquard, however, showed that essentially the same symptoms might be set up by irritative diseases of the intestines, and other organs, and on the supposition that the paralytic phenomena were caused by a reflex spasm of the spinal vessels he named the affection *reflex paraplegia*. The paraplegia which is associated with diseases of the urinary organs and other viscera appears to consist of several varieties. The following may be distinguished: (1) Secondary myelitis, caused by an ascending neuritis of the nerves of the diseased organ; (2) Functional paralysis, caused by some mechanism not yet accurately determined, but which in the meantime may be called *reflex paraplegia;* and (3) Paralysis, caused by direct propagation of inflammation from

the nerves of the urinary passages to the lumbar and sacral plexuses :—

(1) *Secondary Myelitis.*—The diseases which usually cause secondary myelitis are gonorrhœa, stricture of the urethra, chronic cystitis, prostatic abscess, pyelo-nephritis associated with calculus, and nephritis. As a rule, spinal paralysis occurs only in chronic affections of the urinary passages.

The symptoms are usually those of a subacute transverse myelitis, situated at the superior part of the lumbar enlargement. They are, briefly, formication and numbness in the lower extremities, girdle sensation, and later anæsthesia or analgesia. Paraplegia is soon established, with excess of the reflex actions, but these become diminished and ultimately lost as the lumbar enlargement is involved, cystitis and bed-sores then form, and soon cause death. Inflammatory action may at times extend upwards and involve the upper extremities in the paralysis. It has been proved experimentally that inflammation of the sciatic nerve may cause myelitis (Tiesler), and several cases are recorded in which injury of it has been followed by myelitis in man. A case is recorded by Cornil in which tumour of the cauda equina produced a myelitis of the central grey substance of the cord, along with sclerosis of the posterior columns, and similar cases have been recorded by Simon, Lange, and Leyden ; a case of the kind has come under my own observation. In the case already mentioned, under the care of Dr. Morgan, a severe injury to the sciatic nerve was followed by the symptoms of subacute central myelitis. After death a microscopical examination showed perineuritis of the injured sciatic nerve, central myelitis, reaching up the whole length of the cord, along with grey degeneration of the posterior columns in the lumbar and dorsal regions, but limited to the columns of Goll in the cervical region, the portion which adjoins the posterior commissure being healthy too in the lumbar region.

In a case related by Duménil, a neuritis of the sciatic nerve was followed by paraplegia, and at a later period by paralysis of one of the upper extremities. The paralysed muscles became atrophied with diminution of their faradic contractility. At the autopsy the grey matter was found diseased, while the white substance was unaffected. Charcot describes a case in which lesion of one of the nerves of the forearm was first followed by inflammation of the peripheral portion of the nerve, atrophy of the muscles of the hand, and pemphigoid eruptions, while at a later period the arm of the opposite side was affected with atrophy and anæsthesia.

(2) *Reflex Paraplegia.*—In this form of the disease the paralysis never extends to the upper extremities, while the lower extremities are only paretic, and never completely paralysed. There is also complete absence of pains in the loins, girdle pains, dysæsthesiæ, anæsthesia, muscular tension, and contractures, paralysis of the bladder, bed-sores, and other trophic disturbances. The paralytic symptoms are variable in

their intensity, and may improve rapidly if there be an amendment of the peripheral lesion which is the cause of the affection.

Brown-Séquard observed that ligature of the hilus of the kidney in animals produced spasm of the vessels of the spinal cord, and he argued that the paralysis which is caused by diseases of the urinary organs is occasioned by anæmia of the spinal cord. Charcot, on the other hand, believes that the peripheral irritation produces an inhibitory effect on the spinal cord. In chronic Bright's disease the fibroid changes, which the spinal, like the other vessels of the body undergo, must cause anæmia of the cord, which may, in exceptional cases, reach such a degree as to cause some amount of paralysis.

(3) Peripheral paralysis from extension of neuritis from the nerves of the urinary organs is rare. Kussmaul reports a case in which inflammation of the urinary passages had given rise to a neuritis, which extended to the nerves of the sacral and lumbar plexuses. During life, the patient complained of shooting pains along the course of the sciatic nerves, while there was paresis of the lower extremities. Pelvic abscesses may cause inflammation of the sacral plexus, and thus occasion paralysis and anæsthesia of one or both the lower extremities (Adams).

(IV.) SALTATORY SPASM.

§ 558. Bamberger first described in 1859, under the name of saltatory spasm, two cases in which, as soon as the patients set their feet on the floor, the lower extremities became the subject of such strong clonic convulsions that the patients were thrown repeatedly into the air. Similar cases have been reported by P. Guttmann, and A. Frey has recently communicated a case and examined the subject in detail.

The common characteristic of all the published cases of the affection is that there is a great increase of reflex excitability in certain nerve tracts, so that on the sole of the foot being placed on the floor a singular spasm occurs, which has the effect of throwing the patient repeatedly into the air. These spasmodic contractions continue as long as the patient maintains the erect posture, and they cause the patient to hop and jump on the floor, and render him quite unable to stand still for an instant. When the patient sits or lies down the movements disappear, but can be instantly made to reappear by tickling, or pressing on the soles of the feet.

In saltatory spasm the reflex mechanism of the cord is alone affected, and there is complete absence of paralysis.

In some of the reported cases other spasmodic manifestations are mentioned, but the spasm which causes the patient to hop on assuming the erect posture is by far the most characteristic feature of the disease.

It was demonstrated in Frey's case that the reflex action did not begin in the skin, and he regards the symptom as being due to tension and stretching of the muscles, and it is not by any means improbable that the spasms are really due to increase of the reflex excitability of the tendons. Bamberger's first case appears to show that the reflex action may originate in the skin. His second case was associated with hysteria, and therefore allied to so-called chorea major. In Frey's case paresis, contractures, and atrophy were present, and consequently the saltatory spasm was to be regarded as a symptom of chronic myelitis. The distribution of the spasms varies greatly in individual cases. They are at times limited to the legs, while at other times they extend to the muscles of the back, face, neck, and pupils, but the arms always remain unaffected. Mental influences have been found to aggravate the spasm in some cases, and to arrest it in others. The diagnosis of the affection is easy on account of the very characteristic hopping movements as soon as the feet touch the floor.

§ 559. *Treatment.*—In Bamberger's first case the administration of morphia appeared to have a beneficial effect, but it is doubtful how far treatment has been attended by good results. The agents most worthy of trial are bromide of potassium, Calabar bean, ergotine, conia, and atropia.

(V.) TONIC SPASMS IN MUSCLES CAPABLE OF VOLUNTARY MOVEMENT.

§ 560. In this affection, if it can be called a separate affection, the voluntary muscles become the subjects of increased tension and tonic spasms the moment any attempt is made to move them. Such cases manifest a marked hereditary tendency. The most remarkable cases of this kind are recorded by Dr. Thomsen, who had himself been subject to the disease since childhood. The disease appeared in his children, brothers and sisters, and could be traced through four generations of his

ancestors. The disease begins in early life, and Dr. Thomsen was able to recognise it in his children even in the cradle.

The disturbance of movement consists in a peculiar stiffness and rigidity of the muscles on voluntary movement, and this may increase to a regular tonic cramp, so that intended movements are entirely prevented and the patients fall to the ground. Voluntary contraction of the muscles occurs very slowly; but when once it has begun it persists long and terminates only very gradually, so that patients cannot at once let go articles they have firmly seized. When, however, after a powerful voluntary exertion the muscles are got to act, the movements are effected with increasing freedom and ease, so as to be little distinguished from healthy movements. Emotional disturbances, increased attention directed to the movements, and cold, all act unfavourably on the condition. The muscular system in these patients is well developed; they are capable of performing a large amount of labour, and their general health and mental functions are unaffected.

§ 561. *Morbid Physiology.*—Thomsen thought that the affection was mainly of psychical origin, but there is not sufficient evidence in support of this opinion. The view ultimately adopted by Seeligmuller, that it arises from a congenital or inherited affection of the pyramidal tracts of the cord, appears more likely to prove correct. The question, however, can only be decided by further observations.

§ 562. *Treatment.*—No treatment has hitherto been of any service.

(VI.) INTERMITTENT SPINAL PARALYSIS.

§ 563. One of the most remarkable of the manifestations of malarial infection is the occurrence of intermittent attacks of paraplegia.

In the recorded cases (Hertz, Romberg, Hartwig) paraplegia became rapidly developed and advanced steadily to complete motor paralysis. The paralysis of the lower extremities may or may not be accompanied by anæsthesia and paralysis of the

sphincters. The paraplegia usually disappears in the course of a few hours, and gives place to an almost complete intermission, accompanied by the appearance of a critical sweat. This process is repeated, in a more or less regular manner, in the quotidian, tertian, or quartan type, and the affection is either cured or favourably influenced by quinine.

§ 564. *Morbid Physiology.*—All we know about the pathology of this affection is that it is in all probability due to the malarial poison acting on the spinal cord, but of its mode of action we know nothing.

§ 565. *Diagnosis.*—The intermittent character of the affection renders the diagnosis easy, and the treatment is the same as that which is applicable to all forms of intermittent fever.

(VII.) TOXIC SPINAL PARALYSIS.

§ 566. Opium, belladonna, arsenic, phosphorus, lead, mercury, carbonic oxide, sulphide of carbon, tobacco, camphor, ergot, alcohol, absinthe, mushrooms, copaiba, and many other toxic agents induce various forms of motor paralysis, such as paraplegia, paralysis of groups of muscles, or of single extremities, and general paralysis.

Permanent paralysis is only caused as a rule by these agents when the organism is exposed for a long period to their influence, although occasionally paralysis may result from a temporary poisoning.

Absolutely nothing is known with regard to the nature and locality of the lesion caused by the majority of these agents. Landouzy has recently collected all the various forms of paralysis which occur in the course of or subsequent to infective and other acute diseases, but inasmuch as many of these are not of spinal origin, it will be well to defer their consideration at present. This subject will be subsequently treated in greater detail.

(VIII.) HYSTERICAL PARAPLEGIA.

This form of paralysis will be described at greater length hereafter, and is mentioned in this place only with the view

of reminding the reader to be carefully on his guard lest hysterical paralysis be mistaken for more serious disease. In hysterical paraplegia, the lower extremities are generally maintained in a condition of rigid extension, while the feet are in the position of extreme talipes equino-varus. As a rule, however, there is no muscular atrophy, the electric reactions are normal, and the limbs become quite relaxed when chloroform is administered.

CHAPTER VIII.

V.—TRAUMATIC DISEASES, TUMOURS, AND ABNORMALITIES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

(I.) WOUNDS OF THE SPINAL CORD AND MEDULLA OBLONGATA.

THE affections comprised in this section are acute traumatic lesion of the substance of the cord and medulla oblongata.

§ 567. *Etiology.*—In fractures and luxations of the spinal column the injured vertebræ may be so displaced as to cause compression and crushing of the cord.

Gunshot wounds often injure the spinal cord either by the entrance of the bullet into the spinal canal or by fracture of the vertebræ. Stabs and cuts of the spinal cord are rare; but sharp instruments have been known to enter the cord, the point of the instrument having entered the canal either by dividing the vertebral arches or by passing through the intervertebral spaces.

Injuries of the medulla oblongata may be produced by a sharp instrument piercing between the occiput and altas, by bullets, splinters of bone, blows on the back of the neck without fracture, and on the top of the head by *contre coup*. Fractures and dislocations of the first two cervical vertebræ are also important causes of wounds of the medulla oblongata. Dislocation of the first vertebra, or rupture of the odontoid ligament, is accompanied by a backward displacement of the odontoid process, which presses against the anterior surface of the medulla, and causes instant death.

§ 568. *Symptoms.*

1. Wounds of the Spinal Cord.

The symptoms may be subdivided into those which are caused by (*a*) comparatively slight injuries of the cord, such as simple incised and punctured wounds; and (*b*) those which arise from the more serious lesions, such as compression, crushing, and tearing of the cord.

(*a*) The symptoms which indicate that an injury by cutting or stabbing in the neighbourhood of the spine has penetrated the cord will at first be those caused by loss of conduction to and from the brain in the portions situated below the seat of the injury. At the moment the injury is received there is usually motor paralysis of various extent in the form of paraplegia, hemi-paraplegia, or general paralysis. If the cord be completely divided, there is complete anæsthesia of the paralysed parts; but if only one-half of the cord be divided, the sensory paralysis is situated on the side opposite to the injury and to the motor paralysis. The anæsthesia is sometimes partial, and if the lesion be very restricted, hyperæsthesia in the form of a girdle is present. If the injury be of any considerable extent, paralysis of the bladder and rectum occurs, and there is also vaso-motor paralysis with increased temperature and redness of the regions affected by the motor paralysis. The reflex actions are usually abolished at first owing to the shock; but if the lesion be situated in the dorsal or cervical regions, they may after a time be exaggerated.

Girdle pains, caused by irritation of the posterior roots at the seat of injury, are usually present. After a time the symptoms of secondary traumatic myelitis complicate those caused by the primary lesion. The symptoms of irritation now appear, such as girdle pains, active pains in the paralysed parts, cutaneous hyperæsthesia of variable extent, and twitchings and spasms of single muscles and groups of muscles.

When the inflammatory action spreads through the whole thickness of the cord, the paralysis extends in the transverse direction, so that even when the wound has only injured a small portion of the transverse diameter of the cord, complete paraplegia, para-anæsthesia, and paralysis of the vaso-motor paths,

bladder, and rectum may occur. When the lesion is situated high up, disturbances of respiration become prominent, and various oculo-pupillary phenomena and vaso-motor disturbances of the head and face may be present. At a later period bed-sores, cystitis, pyæmia, and septicæmia supervene with all their deleterious consequences.

(*b*) The symptoms which indicate that the cord is crushed or torn in severe injuries of the spine are complete paralysis and anæsthesia of the portion of the body below the seat of injury. The reflex actions are abolished, there are retention of urine, involuntary evacuations with constipation and meteorism, painful erections, and elevation of the temperature of the body below the lesion. The local symptoms of injury to the spine and of displacement of the vertebræ are of course present. The symptoms of acute traumatic myelitis appear in a few days, consisting of bed-sores and pyæmia, with their usual consequences.

If the lumbar region be crushed, there is rapid atrophy of the muscles of the legs, with loss of electrical contractility, and cystitis. When the cervical region is injured, the temperature may rise to an excessive height (43°—44° C.) (109°—111° F.). In some cases, when the dorsal region has been injured, the temperature has been abnormally low for some days before death (Nieder). The severer cases are rapidly fatal by paralysis of respiration, while death is caused in other cases by acute bed-sores and pyæmia. In partial crushing of the cord the symptoms run a milder course.

§ 569. *Varieties.*—The symptoms vary according to the level at which the lesion is seated.

If the cord be injured at the level of the first or second cervical vertebra, death usually occurs at once; and when the lesion is situated above the origin of the phrenic nerves, respiration is only maintained by the forced action of the auxiliary muscles of inspiration, and the case terminates fatally in a brief space of time.

If the lesion be situated in the cervical region below the origin of the phrenic nerves, the arms are partially and the legs completely paralysed, anæsthesia being also partial in the former and complete in the latter; inspiration is performed by the diaphragm, expiratory acts are feeble, painful erections are often present, and life may be prolonged for some time.

If the lesion be situated in the dorsal region, the arms remain unaffected, the muscles of the trunk and lower extremities are paralysed below the seat of the lesion, the reflex actions soon become exaggerated, painful erections are rare, the bladder and rectum may after a time become paralysed, and bed-sores supervene with their usual consequences, or the myelitis assumes an ascending course, and the patient dies from asphyxia.

If the lesion be situated in the lumbar region, the arms and the greater portion of the trunk are unaffected, the legs, bladder, and rectum are totally paralysed, reflex actions of all kinds are abolished, there are no erections, and the muscles of the lower extremities undergo rapid atrophy, with extinction of their electrical reaction. The symptoms arising from lesion of the cauda equina are somewhat similar, but in it the region supplied by the lumbar plexus is unaffected.

2. Wounds of the Medulla Oblongata.

§ 570. If the injury to the medulla be a severe acute one, the patient collapses as if struck by lightning, and dies instantaneously. Sometimes he gives utterance to a piercing cry before falling, or death may be accompanied by a few transitory convulsions. All this results from sudden paralysis of the respiratory centre and complete interruption of all the conducting paths between the spinal cord and the brain.

When the wound is less severe, or when the medulla is only partially lacerated, the affection may last for some time, but these cases too, as a rule, terminate suddenly. It is probable that patients sometimes survive very small injuries to the medulla, but this is difficult to prove. Sudden death may perhaps sometimes occur from simple concussion of the medulla in the entire absence of any serious lesion.

§ 571. *Morbid Anatomy.*

Simple incised or punctured wounds produce injuries of various size and depth. The edges of the wound project at first beyond the pia and the wound is closed with coagulated blood. In a few days the edges are still further protruded, while the neighbouring parts of the cord are more or less softened, and the membranes are reddened and inflamed and covered with fibro-purulent exudations. In animals at least, and probably also in man, if life be preserved, the edges of the wound heal, and a cicatrix of connective tissue is formed.

Crushing produces softening and disintegration of the cord,

along with congestion and hæmorrhage into the membranes. The crushed spot is usually flat and thin, and the medullary substance is changed into a dark red or chocolate coloured mass composed of blood and the débris of nerve-substance. The adjoining parts become congested and subsequently undergo inflammatory softening. The microscope shows granular corpuscles, detritus of myeline, decomposed blood corpuscles, pigment and blood crystals, and remnants of ganglion cells, along with inflammatory swelling and disintegration of nerve fibres and axis cylinders. In a few weeks the cord for a considerable distance, both above and below the injury, undergoes softening, and this is specially apt to occur in the lower part of the cord. Ascending and descending secondary degeneration occur when life is prolonged, and in several cases fatal within a few days from the date of injury I have found decided evidences of a central myelitis up to the medulla oblongata.

If the patient live, the destroyed nerve tissue becomes absorbed, and a kind of cicatrix is formed, which may enclose cystic cavities containing clear fluid. Regeneration of nerve substance is not known to occur in man.

Complete severance of the cord occasionally occurs, and then the pia is also torn, and the two ends of the cord are separated by a considerable space. The spinal dura mater may remain uninjured. Inflammatory softening occurs as after crushing, and extends more or less upwards and downwards.

The anatomical changes found in the medulla when it is wounded, lacerated, or crushed resemble the acute injuries of the spinal cord.

§ 572. *Course, Duration, and Termination.*—In simple incised wounds of the cord a comparative cure may be effected, and life retained for many years. Physiological experiment shows that animals may be kept alive for a long period even after complete division of the cord, and it is quite possible that lesions of moderate severity may undergo repair. As a rule, however, the secondary myelitis set up continues to increase, the paralysis becomes more complete, bed-sores with all their attendant evils make their appearance, and the patient dies after protracted suffering. The severe forms of injury to the

spinal cord are always fatal. Death may take place a few hours or days after the injury from shock or paralysis of respiration, but in some cases life may be protracted for many months.

Acute severe injury to the medulla oblongata causes instant death, and slight injuries are exceedingly dangerous, because the inflammatory action set up by them generally leads rapidly to a fatal termination.

§ 573. *Diagnosis.*—In the case of a wound of the pia mater the occurrence of meningeal hæmorrhage might give rise to the idea of injury of the cord. In meningeal hæmorrhage, however, the symptoms of irritation are very prominent at the outset, while those of paralysis are less severe than in injury of the cord.

Hæmatomyelia induces a certain amount of crushing of the cord, and gives rise to similar symptoms, but in it there is usually no history of external injury, and when there is, the diagnosis between the two affections is not of much consequence.

Cases of severe *concussion* of the cord may usually be recognised by the absence of a clear demarcation of the anæsthesia and paralysis, and by the absence of bed-sores, and other trophic changes. If dislocations of the vertebræ are found to exist, crushing of the cord is more probable.

§ 574. *Prognosis.*—In all the severer forms of injury the prognosis is exceedingly unfavourable, but in cases of partial injury and simple incised wounds a certain amount of recovery may take place. Sudden injury to the medulla oblongata is almost uniformly fatal.

§ 575. *Treatment.*—The associated traumatic myelitis must be treated according to the principles applicable to other forms of acute inflammation of the cord.

(II.) SLOW COMPRESSION OF THE SPINAL CORD AND MEDULLA OBLONGATA.

§ 576. In all the lesions comprised under this section an external force slowly and gradually compresses the cord or

medulla oblongata in a limited longitudinal extent, giving rise to characteristic groups of symptoms.

§ 577. *Etiology.*—Any circumstance which gradually narrows the spinal canal and leads to a slowly increasing compression of the cord may become a cause of myelitis by compression. Such compression may be caused by meningeal, perimeningeal, and intramedullary tumours, inflammatory and hæmorrhagic processes, and parasites. Diseases of the vertebral column, especially caries of the vertebræ, constitute the most important causes of compression of the cord, and may produce pressure on the cord in several ways. The wasting and sinking of the bodies of the vertebræ produce kyphosis, which may narrow the vertebral canal to such an extent as to compress the cord.

In caries of the vertebræ, however, compression of the cord is generally produced by the extension of the inflammatory process in the bone to the spinal membranes. The irritation caused by the diseased vertebræ and especially by accumulations of pus produce a pachymeningitis, so that the outer layers of the dura are changed into a thickened mass of young fibro-plastic tissue, which either surrounds the dura like a ring or presses it from one side. The nerve roots are also involved in the morbid process, and become more or less thickened, swollen, and inflamed. The cause of pressure may be deposits of caseous pus, displaced fragments of bone, or protruding intervertebral cartilages.

Carcinoma of the vertebræ, whether primary or secondary, causes compression of the cord when it grows into the vertebral canal. The form of compression myelitis known as *paraplegia dolorosa* with most acute pains is then developed.

Amongst other diseases of the vertebræ which occasionally cause compression of the cord may be mentioned exostoses, syphilitic new formations, dry arthritis of the vertebræ, and thickening of the odontoid process of the axis.

External tumours of all kinds, such as carcinomata, sarcomata, aneurisms, and echinococci, growing against the vertebral column and entering the vertebral canal, occasion compression of the cord. When the gradual compression gives rise to transverse myelitis, another characteristic group of

symptoms appears, which constitutes the second stage of the affection. The irritation caused by tumours may produce myelitis in the absence of any compression. If the compression be not soon relieved, secondary ascending and descending degenerations of the cord supervene, and then another group of characteristic symptoms develop, constituting the third stage of the disease.

Slow compression of the medulla oblongata is caused by tumours growing from the bones, periosteum, meninges, or in the surrounding parts of the brain and growing in the direction of the medulla, or by tumours growing in the substance of the pons or medulla itself, and aneursims of the arterial trunks. Caries of the occipital bone or of the first two cervical vertebræ with consequent exudation, abscess, or dislocation of the odontoid process may also produce gradual compression of the medulla oblongata.

The medulla is often compressed, too, by abnormalities in the shape and size of the bones, such as contraction of the foramen magnum and enlargement of the odontoid process. Arthritis deformans in the articulations between the occiput and altas would appear to be a very rare cause of compression of the medulla.

§ 578. *Symptoms.*

1. Slow Compression of the Spinal Cord.

The symptoms of compression myelitis may be divided into (*a*) extrinsic and (*b*) intrinsic symptoms.

(*a*) *The extrinsic or prodromal symptoms* are, in addition to the signs of Pott's curvature or tumour, phenomena of irritation of the roots of the spinal nerves. The first symptoms generally consist of severe girdle pains, hyperæsthesia of the skin corresponding to the distribution of the pain, and eccentric neuralgiform pains generally fixed to one particular spot. Other symptoms generally present are severe pain in the back, local stiffness, and tenderness of the spinous processes. The neuralgic pains are often accompanied by herpetic or bulbous eruptions of the skin. Phenomena of motor irritation are superadded to the sensory disturbances in the distribution of

the nerves whose roots were first implicated. These consist of twitchings, spasms, and contracture of the muscles supplied by the affected nerves. The irritative motor symptoms are soon followed by weakness and paralysis, which is limited to single muscles or groups of muscles, and often accompanied by atrophy and loss of electrical excitability. In the paralysis caused by pressure on the roots of the nerves reflex actions are abolished. The initial stage may last months or years, and always precedes for some time that of compression of the cord. The irritative symptoms are absent in cases of intra-medullary tumours.

(*b*) *The Intrinsic Symptoms.*—The second stage of the disease is generally ushered in by paralysis of more or less rapid development, usually in the form of paraplegia. The paralysis may at times begin as hemiplegia, and afterwards develop into paraplegia. The paralysis may be preceded for a short time by paræsthesiæ in the lower half of the body, such as tingling, furriness, sensations of burning or coldness, and girdle sensations. The order in which the sensory and motor symptoms appear depends on the direction in which the pressure is made. The paralysed muscles are at first flaccid and offer no resistance to passive movements, but the cutaneous reflex actions are increased in the lower extremities, except when the lumbar enlargement is subjected to pressure.

Paralysis of the rectum and bladder occurs sooner or later when there is a considerable degree of compression, but it is usually a late symptom when the lesion is situated in the dorsal or cervical regions. As the disease progresses the muscles become gradually tense and rigid, and are affected with twitchings or transient tonic spasms. Contractures appear, which are at first temporary, but soon become permanent. The lower extremities continue in a permanent position of extension, which may subsequently give place to flexion.

The cutaneous and tendinous reflex actions are now greatly exaggerated, and slight dorsal flexion of one foot may produce active clonic movements, or convulsive tremors in both the lower extremities. The reflex actions originating in other parts are also increased. The introduction of a catheter or the evacuation of the bladder or rectum may cause active jerkings of the limbs,

and irritation of the inner side of the thigh has been known to produce erections of the penis.

The sensory disturbances are not usually so well marked as the motor, and complete anæsthesia of the paralysed parts is rare in vertebral caries. Cancer of the vertebral column, growing into the spinal canal, is, however, accompanied by pains of intense severity (Charcot). These pains consist of a severe girdle pain and pain radiating along the distribution of certain nerves, as the crural and sciatic nerves when the lumbar vertebræ are affected. The skin to which the affected nerves are distributed is intensely hyperæsthetic, so that the slightest touch is painful. The pains are constantly present but are liable to paroxysmal exacerbations of intense severity, which are difficult to allay even by large doses of narcotics. Patches of anæsthesia may be observed in the skin to which the affected nerves are distributed, while the pain still continues unabated *(anæsthesia dolorosa)*. The symptoms of compression of the cord are after a time superadded to these sensory disturbances, and then the condition has been called *paraplegia dolorosa*. On local examination of the vertebral column an excurvation of the spine may be observed, and the spinous processes in this region may be exceedingly tender to pressure or percussion. Inasmuch as cancer of the vertebral column is always secondary, the presence of a cancerous tumour in some other part of the body, or of the cancerous cachexia, greatly aids the diagnosis.

Trophic disturbances are not prominent in compression myelitis. When the lumbar or cervical enlargement is affected, or when secondary inflammation of the grey substance extends upwards or downwards to these parts, the muscles of the corresponding extremities undergo rapid atrophy, attended by loss of faradic contractility and the reaction of degeneration. In some cases a few of the muscles become atrophied, while others undergo contracture. An eruption of herpes sometimes encircles one-half of the body on a level with the lesion. In severe cases, and in the terminal period of ordinary cases, bed-sores and cystitis supervene, with their usual deleterious consequences.

The subsequent course of the disease is not uniform. The

less severe cases continue for a long time without change, but afterwards improvement may gradually take place. The anæsthesia first diminishes, the functions of the bladder are better regulated, and after a time motor power gradually returns. In severe cases the symptoms grow worse, the paraplegia becomes complete, the bladder and rectum are paralysed, cystitis, bedsores, and pyæmia supervene, and soon cause death.

§ 579. *Varieties.*—The symptoms differ considerably according to the situation of the lesion.

(*a*) *Compression of the Cervical Region of the Spinal Cord.*—When the *upper part of the cervical region* is affected the disease often begins by pain in the occiput, stiffness of the whole neck, obliquity of the head, and inability to nod or to rotate the head. The paralysis often begins in the upper extremities, while the lower are wholly or comparatively unaffected; but at a later period the extremities become paralysed, the reflex actions being exaggerated in all the extremities. Paralytic myosis or spastic mydriasis may be present on one or on both sides. Other symptoms which have been observed are repeated vomiting, difficulty of swallowing, incessant hiccough, retardation of the pulse, which may beat only 48 to 20 times in the minute, fainting fits with temporary arrest of the heart's action, and occasionally epileptic attacks.

If the *cervical enlargement* be affected, the initial symptoms of pain, anæsthesia, spasm, paralysis, and atrophy are localised in the upper extremities, and the symptoms appear in the lower extremities at a later period. Oculo-pupillary symptoms, disturbances of respiration, and retarded pulse may also occur. Reflex action may be abolished in the upper extremities.

(*b*) *Compression of the Dorsal Region of the Cord.*—The dorsal region is the most frequent seat of compression. The symptoms are girdle pains, intercostal neuralgia at different levels of the trunk, paraplegia up to the corresponding level, reflex actions in the lower extremities retained or increased, and the nutrition of the muscles and their electrical excitability normal.

(*c*) *Compression of the Lumbar Region of the Cord.*—If the lumbar region be affected, the paralysis is confined to the lower extremities, bladder, and rectum. The initial symptoms are localised in the lower extremities, where reflex actions are abolished, and the muscles are permanently relaxed and atrophied and exhibit the reaction of degeneration. If one lateral half of the cord be compressed, the characteristic symptoms of Brown Séquard's unilateral lesion appear.

2. Slow Compression of the Medulla Oblongata.

The initial symptoms are caused by irritation and subsequent paralysis of the roots of the nerves of the medulla and pons. Those of irritation first show themselves, consisting of neuralgiform pains in the region of the trigeminus, either on one or both sides, and buzzing in the ears. The motor irritative symptoms consist of twitchings of the facial muscles, transitory cramps in the tongue and lips, and occasionally clonic or tonic contractions in the extremities. When the medulla oblongata is seriously compressed, epileptiform convulsions, vomiting, dizziness, and hiccough are produced. The second stage is ushered in by paralysis of the sensory and motor nerves. There may be anæsthesia in the region of the trigeminus, often accompanied by intense pain and neuroparalytic ophthalmia. There may be loss of taste, or deafness on one or on both sides, and one or more of the cranial motor nerves may be paralysed, while the paralysed muscles undergo atrophy, lose their faradic contractility, and manifest the reaction of degeneration. After a time a true bulbar paralysis appears, the extremities become paralysed, and disorders of respiration supervene. The symptoms begin sometimes so suddenly as to simulate embolus or thrombosis. These acute symptoms are caused by a rapidly developing bulbar myelitis, or by œdema, thrombosis, or hæmorrhage. The optic nerves are not affected unless the tumour be of large size, and probably then only when it is accompanied by effusion into the ventricles of the brain.

§ 580. *Course, Duration, and Termination.*—The course of the disease depends on the nature of the primary lesion. Meningeal and intra-medullary tumours, as well as carcinoma or other malignant tumours of the vertebræ, are always fatal. In most cases of vertebral caries, on the other hand, the course is comparatively favourable. Many cases, however, progress slowly with remissions and exacerbations to a fatal termination. In other cases the recovery is incomplete, partial paralysis, contractures, muscular atrophy, and anæsthesia remain, and relapses are frequent.

§ 581. *Morbid Anatomy.*—The meninges are often hyper-

æmic, opaque, and adherent to the neighbouring parts, or covered with deposits of various thickness. The nerve roots may be closely united with the tumour or exudation. At first they are swollen and hyperæmic, and their fibres are in a state of fatty degeneration. At a later period the roots are atrophied, pale grey, degenerated, and nearly reduced to connective tissue.

The substance of the spinal cord is rendered more or less flat and thin at the point compressed, and it may be reduced to the size of a small quill. The compression is sometimes greater anteriorly, sometimes greater posteriorly, at other times from one or other side, so that the cord assumes a distorted and irregular appearance. The compressed spot varies in length, and is usually softened, although it may be sclerosed in long-standing cases. In chronic cases the usual ascending and descending changes occur above and below the level of the lesion.

A microscopical examination of the spinal cord reveals the appearances which usually characterise a chronic interstitial myelitis. In addition to the characteristic phenomena of ascending and descending sclerosis, a myelitis of the central grey substance may often be discovered for a considerable distance above and below the seat of lesion. In favourable cases restoration and almost complete recovery may take place, and consequently the nerve elements must be to some extent restored at the point of compression. Charcot and Michaud examined a case, fatal from other causes, in which recovery from compression myelitis had occurred. The transverse section of the cord at the seat of compression was much smaller than the other portions of the cord, and looked grey and degenerated. Microscopical examination showed that there was an excess of connective tissue at the seat of compression, through which a considerable number of normal though slender nerve fibres passed. The grey substance was much reduced in size, but some healthy ganglion cells were observed in it. It is probable that the axis cylinders of all the fibres were not destroyed, and that they had assumed a new medullary sheath on the pressure being removed.

The medulla oblongata may be flattened, either on one or

both sides, turned on its axis, and distorted in various ways. The tissue of the medulla is anæmic and softened, while extravasations of blood are often observed. The roots of the cranial nerves may be compressed and flattened, and the nerves then undergo degenerative atrophy. The pyramidal tracts of the lateral columns and the columns of Türck may undergo descending degeneration throughout the entire length of the spinal cord.

§ 582. *Diagnosis.*—The initial symptoms caused by compression of the roots of the nerves are of importance in the diagnosis of the affection, and in ordinary cases confirmation of the diagnosis will be obtained from the external appearances presented by the primary disease. In Pott's curvature the gradual formation of angular kyphosis, and the history of the case generally, afford indications of the nature of the affection which are unmistakable.

In carcinoma of the vertebræ the girdle sensation and other eccentric pains are of the most agonising severity; they occur in nocturnal paroxysms, and great hyperæsthesia usually exists in the painful region. If primary cancer can be found in another organ, or there be a general cachexia, the diagnosis will be less difficult.

The recognition of the rarer causes of compression of the cord, such as exostoses, syphilitic new formations, and aneurisms, is made from the general symptoms of the respective diseases.

Slow compression of the medulla oblongata may be suspected when symptoms of irritation in the regions of distribution of some of the bulbar nerves are followed by those of sensory or motor paralysis, and when electrical examination shows that the motor paralysis is of peripheral origin. The diagnosis is still further confirmed when the patient suffers from giddiness, violent headache, severe vomiting, epileptoid convulsions, and when twitching and subsequent paralysis and contractures occur in the extremities, more especially when the distribution of these in the latter is unsymmetrical. Cases of compression of the anterior pyramids of the medulla may closely resemble spastic spinal paralysis. The points of distinction between the two affections are that the paralysis begins suddenly in compression,

the upper extremities are usually affected before the lower, and bulbar paralysis sooner or later supervenes; while in spastic paralysis the commencement of the paralysis is slow and gradual, the lower extremities are usually affected before the upper, and bulbar symptoms probably never appear in primary lateral sclerosis.

§ 583. *Prognosis.*—In most cases of slow compression of the cord the prognosis is unfavourable. Cases due to the pressure of syphilitic formations, peri-meningeal exudations, and vertebral caries often recover. In young, well-nourished persons, who are not scrofulous, recovery with slight deformity of the vertebral column generally takes place. In many cases, however, recovery is imperfect, and a certain amount of paralysis of the lower extremities with contractures remains.

The prognosis of slow compression of the medulla oblongata is always unfavourable.

§ 584. *Treatment.*—In severe cases the treatment must be altogether palliative, and directed to the relief of pain and other discomforts.

The most promising cases for treatment are those of Pott's disease. In them rest in bed for months is necessary, in order to maintain the spine in a condition of repose. Various kinds of apparatus have been used for the support and protection of the spine. The best apparatus consists of the plaster of Paris bandage, introduced by Dr. Sayre, but I must refer the reader to surgical works for a full description of the method of application.

The internal treatment should be that adapted to scrofulous patients generally, consisting of fresh air, cod-liver oil, cream, iron, and quinine. The hot iron has been recommended by Charcot and others to be applied every two weeks on each side of the curvature.

In slow compression of the medulla oblongata, treatment is of very little avail, unless the case be one of caries, or syphilitic tumour.

(III.) HEMIPLEGIA ET HEMIPARAPLEGIA SPINALIS.

Unilateral Lesion of the Spinal Cord.

Brown-Séquard's Spinal Paralysis.

§ 585. *Definition.*—The symptoms which are grouped together under the name of unilateral spinal paralysis are mainly characterised by unilateral motor paralysis and hyperæsthesia on the side of the lesion, anæsthesia on the opposite side, and local symptoms caused by implication of the roots of the nerves on a level with the primary lesion.

§ 586. *Etiology.*—The symptoms depend not upon the nature of the lesion, but upon its localisation in one lateral half of the spinal cord, so that it is unnecessary to give a detailed account of all the causes of the affection. The penetration of the vertebral canal by pointed instruments constitutes the most frequent cause. Compression of the cord from meningeal tumours, fractures or dislocation of the vertebræ, meningeal hæmorrhage, intra-medullary tumours, hæmorrhage into the substance of the cord, and circumscribed sclerosis may also give rise to the symptoms of unilateral spinal paralysis.

§ 587. *Symptoms.* — The symptoms of unilateral spinal paralysis may be developed insidiously and gradually, or quite suddenly, the mode of invasion depending of course on the nature of the lesion. The most prominent feature of the affection is a motor paralysis, which is unilateral, and which may only involve one leg (hemiparaplegia), or, if the lesion be situated high up, may also implicate the arm of the same side (spinal hemiplegia). The muscles on the paralysed side usually undergo early and rapid atrophy, and their faradic excitability is diminished. The side opposite to the seat of the lesion is either free from paralysis or is only affected to a slight degree.

Evidences of *vaso-motor paralysis* are generally found on the side of the lesion, especially if the affection has been rapidly developed. The temperature of the paralysed limbs is usually raised to the extent in 1·8° F. or more, although it may be lower than natural when the disease has existed for some time.

Muscular sensibility and muscular sense are usually diminished on the affected side, but all forms of cutaneous sensibility, instead of being diminished, are greatly increased. Impressions of touch, temperature, and pain are felt with great acuteness, and there is an increased power of localising tactile sensations. At times, however, the hyperæsthesia is limited to a few only of the forms of cutaneous sensibility. The hyperæsthetic region of skin is usually bounded by an anæsthetic zone, which corresponds with the height and longitudinal extent of the lesion in the spinal cord; and a narrow hyperæsthetic zone which extends to the opposite side may sometimes be detected above the anæsthetic belt. The state of reflex action on the paralysed side varies. It has been found increased by Paoluzzi and Riegel, and diminished by Brown-Séquard, Bazire, and others. Not many observations have hitherto been made with respect to the state of the reflex irritability of the tendons, but Erb found it increased in one case.

There either are no motor disturbances, or they are only of slight degree on the side opposite the lesion, and both the muscular sense and the electro-muscular sensibility are retained. There is more or less complete anæsthesia of the skin. Some forms of sensibility may at times be involved to a greater extent than others. The anæsthesia extends to the median line of the body, and it is often bounded above by a slightly hyperæsthetic region, corresponding to a similar zone on the opposite side. There are no vaso-motor disturbances on this side, and reflex action is usually normal, although it has occasionally been found increased (Brown-Séquard). The patient sometimes complains of a painful feeling of constriction on a level with the lesion, along with various painful sensations, such as burning, darting, and boring pains, which may at times be more prominent on the anæsthetic, at other times on the hyperæsthetic and paralysed side, and occasionally occurs on both sides. Acute traumatic cases are usually associated at first with retention or incontinence of urine, but after a time only a certain amount of weakness of the sphincters of the bladder and rectum remains. The sexual functions are at times unaffected, and at other times more or less weakened.

Acute bed-sore may appear on the anæsthetic, and inflam-

mation of the knee-joint on the paralysed side, while well-marked ataxia may be observed on the return of motor power in the paralysed leg (Joffroy and Solmon).

§ 588. *Varieties of Unilateral Paralysis.*

The symptoms differ considerably, according to the level at which the lesion is situated in the cord.

(1) In a unilateral lesion of the *lumbar enlargement* of the cord an anæsthetic zone may be found on the paralysed side, corresponding to the area of distribution of one or more of the lumbar nerves, in addition to the other characteristic unilateral symptoms. This area is not always in the form of a belt, and may be situated round the abdomen, in the region of the groin, or over the anterior surface of the thigh, so that from an imperfect examination one might be led to believe that the anæsthesia was diffused over the lower extremities.

(2) Unilateral lesions of the *dorsal portion* of the cord give rise to the most characteristic symptoms of the disease, as already described.

(3) In unilateral lesions of the *cervical portion* of the cord the grouping of the symptoms varies greatly, according to the level at which the lesion is situated and its longitudinal extent. It is manifest that the distribution of the motor and sensory disturbances will differ according as the upper or lower roots of the brachial plexus are involved in the lesion, and according as the cilio-spinal region is or is not implicated.

In the lower extremities and trunk the motor and sensory disturbances are the same as when the dorsal portion is implicated. In the upper extremities on the side of the lesion a certain number or all of the muscles are paralysed, there is hyperæsthesia of certain parts of the skin mingled with anæsthesia of other regions or for certain varieties of sensation. On the side opposite to the lesion there is no paralysis, but there is more or less complete anæsthesia over the whole skin below the lesion, or over special territories.

The neck and head on the side of the lesion manifest anæsthesia and hyperæsthesia of certain areas of the skin and paralysis of the vaso-motor and oculo-pupillary fibres, giving rise to increased temperature of that side of the head and body, heightened sensibility, narrowing of the palpebral fissure, and contraction of the pupil. On the side opposite the lesion there is usually anæsthesia of the neck, along with a narrow zone of hyperæsthesia, and a normal condition of the face and eye.

§ 589. *Morbid Physiology.*—It is almost needless to mention that unilateral section will divide the columns of Türck, the anterior root-zones, the pyramidal tracts and direct cerebellar fibres of the lateral columns, the posterior root-zones and the

columns of Goll, and the anterior and posterior horns of grey matter; and when the lesion is of considerable longitudinal extent a considerable number of the anterior and posterior nerve roots may be destroyed.

Division of the pyramidal tracts in the anterior and lateral columns will sever the muscles below the point of the lesion from the cortex of the brain, hence there will be complete loss of voluntary power below and on the same side as the lesion. When the lesion is permanent, the pyramidal tracts undergo secondary descending degeneration, and, after a time, increased muscular tension and contractures are superadded to the motor paralysis. Recent investigations have apparently proved that the vaso-motor tracts also run in the lateral columns, thus division of these would produce vaso-motor disturbances on the side of the lesion.

Fig. 182.

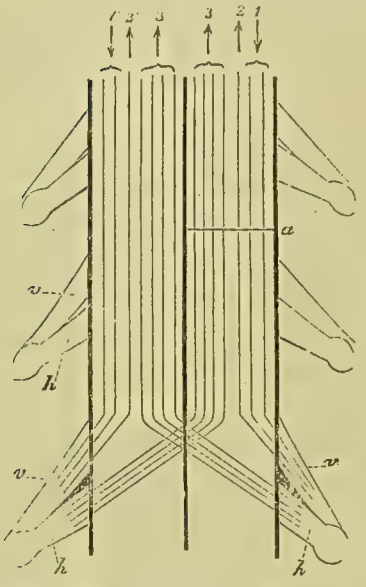


Fig. 182 (After Erb). *Diagram of the Course of the principal Conducting Paths within the Cord.*—1 and 1′, The motor and vaso-motor tracts, passing through the anterior root (*v*), and remaining on the same side of the cord; 2 and 2′, Tracts which conduct the muscular sensibility, also passing through the anterior roots, and remaining on the same side of the cord; 3 and 3′, The tracts which conduct sensory impressions of touch, temperature, pain, and tickling. These enter the cord through the posterior roots, and cross to the other side, and pursue their course upwards on that side. Section of the right half of the cord (*a*) must interrupt conduction through the motor, vaso-motor, and musculo-sensory tracts (1 and 2) on the right side, and the cutaneous sensory tracts on the left side (3′).

Division of the anterior root-zone and direct cerebellar tract is not known to give rise to any symptoms, probably because the result of injury to these parts is masked by the presence of motor paralysis. Similar remarks apply to injury of the columns of Goll and the posterior root-zones, but it is interesting to find that Joffroy and Solmon observed the occurrence of well-marked ataxy on the return of motor power in the paralysed extremity, caused doubtless by the injury of the posterior root-zone of that side.

Division of the grey matter of the one half of the cord

produces anæsthesia of one half of the body below the level of the lesion on the opposite side, showing that the sensory fibres cross over to the opposite side soon after their entrance into the cord. The fibres conducting impressions of touch, temperature, pain, and tickling decussate with those of the other side very near their point of entrance into the spinal cord, and run to the brain in the opposite side of the cord. Those concerned in the phenomena of muscular sense are supposed to enter the cord with the anterior roots, and, like the motor tracts, run through the cord on their own side of the body.

Brown-Séquard states that the conducting tracts of the various forms of cutaneous sensibility cross at different heights, those concerned in the sensation of temperature crossing somewhat earlier than the rest. He also thinks that they are separated from one another in their further course, each lying in certain definite segments of the cord, and that the sensitive tracts of the lower extremities lie behind those of the upper in the cervical portion of the cord. The centripetal fibres concerned in reflex action have a crossed course within the cord (Miescher).

It is evident, therefore, that section of one half of the spinal cord must cause anæsthesia of the opposite side for sensations of touch, pain, temperature, and tickling, and loss of the muscular sense, and motor paralysis on the same side.

FIG. 183.

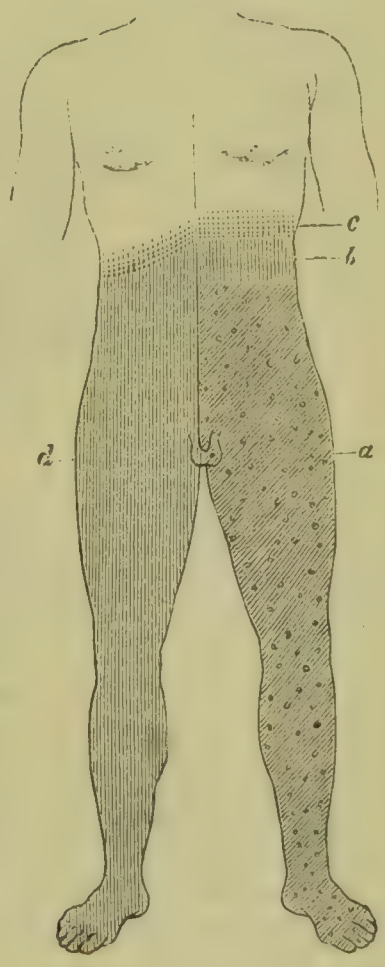


FIG. 183 (After Erb). *Diagram of the cutaneous symptoms in unilateral lesion of the dorsal portion of the spinal cord on the left side.*—The diagonal shading (*a*) signifies motor and vaso-motor paralysis; the vertical shading (*b*, *d*) signifies cutaneous anæsthesia; the dotted shading (*a*, *c*) indicates hyperæsthesia of the skin.

The phenomena which result from section of one half of the spinal cord are well illustrated by *Fig.* 183.

Anatomists have described a middle and a superior crossing of sensory fibres, both of which are supposed to take place in the medulla oblongata. The *sensory decussation* of the pyramids described by Meynert consists of fibres which issue from the nuclei of the cuneate and slender fasciculi. These fibres pursue an arcuate course around the central grey column, and become mixed with the crossing fibres of the lateral column. Flechsig, however, asserts that these fibres curve round the olivary body of the same side and enter into its substance. The most recent researches with regard to the sensory crossing have been undertaken by MM. Debove and Gombault. Their observations were made in a case of amyotrophic lateral sclerosis, in which the motor fibres of the anterior pyramids of the

FIG. 184.

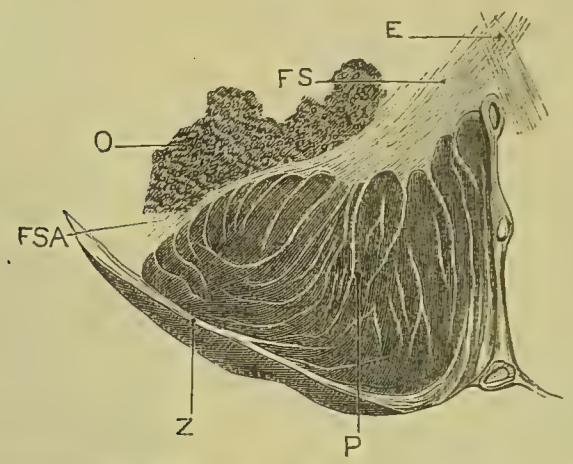


FIG. 184 (After Debove and Gombault). *Section of the Anterior Pyramid* (P) *of the Medulla Oblongata, on a level with the middle part of the crossing of the Sensory Fibres.*—FS, Sensory fibres; FSA, Posterior and external sensory fasciculus which does not penetrate into the substance of the pyramid; E, Crossing of the sensory fibres; O, Nucleus of the pyramid; Z, Stratum zonale.

medulla had undergone degeneration. The sensory fibres become subdivided into small fasciculi, which penetrate into the posterior and external portion of the anterior pyramids. The fibres then curve upwards and become inseparably mixed with the motor fibres. A little higher up some of these fibres become mixed with the fibres of the stratum zonale. These fibres are very well seen in some of my sections of the medulla oblongata from a nine months human embryo, but I have always regarded them as being derived from the external portion of the inner division of the inferior peduncle of the cerebellum, and I am by no means satisfied that their functions are sensory.

The relations of reflex action to unilateral section of the cord are not yet well ascertained, and the condition appears to vary in different animals. According to the experiments of Woroschiloff, it would appear that the fibres which check or control reflex action in a lower extremity

run their course principally in the same half of the cord, while those which call forth reflex action run chiefly in the opposite half. When the lesion extends longitudinally, so as to destroy several of the posterior root fibres before their crossing, there will be an anæsthetic zone on the paralysed side lying above the hyperæsthetic region, and corresponding in width to the number of fibres destroyed by the lesion. Implication of the cilio-spinal region causes vaso-motor disturbances in the face and side of the head, paralytic myosis, and narrowing of the palpebral fissure on the side of the lesion.

The narrow hyperæsthetic zone sometimes observed above the anæsthetic belt is explained by Brown-Séquard on the ground that the descending fibres of the posterior roots fall within the range of the lesion. He has, however, recently obtained a curious result by making a section of one half of the pons in animals immediately in front of the middle peduncle of the cerebellum, followed after a time by section of the other half. After section of the first half of the pons, there is hyperæsthesia of one half of the body, on the side of the lesion, and anæsthesia of the opposite half, the sensory disturbances being specially well marked in the lower extremities. In an animal which had undergone hemisection of the right side of the pons, and which was consequently hyperæsthetic on the right side, hemisection of the left side of the pons produced a reversal of the sensory phenomena, so that the left half of the body became hyperæsthetic and the right anæsthetic. This curious result shows that the phenomena of sensory conduction are by no means so definite or so well ascertained as those of motor conduction.

The existence of cutaneous hyperæsthesia on the side of the lesion is usually explained on the supposition that it is a phenomenon of irritation caused by the secondary inflammation surrounding the lesion; but after the result obtained by Brown-Séquard it will probably be better to suspend our judgment with regard to it in the meantime. It does not, however, appear to me that much light is thrown upon the point by postulating, like Brown-Séquard, the existence in the cerebro-spinal axis of special centres possessed of inhibitory and dynamogenic functions.

§ 590. *Course, Duration, and Termination.*—The course of the symptoms depends chiefly upon the nature of the lesion which has caused the affection and varies greatly in different cases. The lesion as a rule extends both longitudinally and transversely, and only remains stationary in rare cases. When the lesion extends transversely, paraplegia and the other symptoms of transverse myelitis result. As the inflammation subsides the symptoms of the unilateral lesion may recur, and may then continue unchanged for many years. Complete recovery has occasionally been observed in traumatic unilateral lesions, and

according to Brown-Séquard the motor disturbances disappear earlier and more completely than those of sensation. In tumour the disease generally extends transversely, giving rise to paraplegia.

§ 591. *Diagnosis.*—Hemiparaplegia, from lesion of *one side of the cauda equina,* may be distinguished from that caused by unilateral lesion of the cord by the circumstances that in the former the paralysis and anæsthesia are on the same side, and that, as a rule, only certain nerve territories of the lower extremities are affected.

The hemiplegic form of lateral sclerosis is readily recognised by the absence of all disturbances of sensation and of any disorder of the bladder or rectum. *Cerebral hemiplegia* may be distinguished from the spinal form by the fact that in the former there is either no disturbance of sensation or that it is to be found on the same side as the paralysis, and that there is unilateral paralysis of the face and tongue. The nature of the lesion is not readily ascertained in the absence of any distinct injury, and consequently the diagnosis must rest upon general pathological principles.

§ 592. *Prognosis.*—The prognosis depends entirely upon the nature of the lesion which causes the symptoms. The majority of traumatic cases end in recovery with proper care and treatment.

§ 593. *Treatment.*—The treatment is generally the same as that which is applicable to wounds of the spinal cord and to chronic myelitis.

(IV.) CONCUSSION OF THE SPINAL CORD.

(*Commotio Spinalis.*)

§ 594. *Definition.*—Concussion of the spinal cord includes cases in which traumatic injuries occasion severe functional disturbances without the production of recognisable changes.

§ 595. *Etiology.*—The most usual causes of spinal concussion are falls upon the feet, or buttocks, and blows over the

back. Another common cause of the affection is the shock to the whole body, occasioned by the sudden arrest of its motion when it is passing at a rapid rate through space, such as occurs in railway collisions. The persons who sit with their backs towards the direction from which the shock comes suffer most from spinal concussion in railway accidents.

The concussion may be limited to a portion of the cord when the vertebral column has been struck, but in cases of shock to the body the greater portion of the cord or the whole is affected. Violent mental excitement appears to produce symptoms of concussion of the cord, and lightning passing through the body causes a general shock in which the spinal cord participates.

§ 596. *Symptoms.*—The symptoms vary according to the nature of the injury, the degree of individual resistance, and various other circumstances. The most usual symptoms of the affection are feebleness of the extremities, amounting in some cases to decided paralysis, paræsthesiæ of various kinds, pain in the neck, loins, or along the spinal column, tenderness on pressure of some of the spinous processes, some degree of cutaneous hyperæsthesia, or more often anæsthesia, weakness of the bladder, and considerable emotional disturbance.

§ 597. *Varieties of Spinal Concussion.*—The following varieties may be distinguished :—

(1) *Severe form of Shock, giving rise to Aggravated Symptoms at once, and Death in a short time.*—The patient after the injury is found comparatively paralysed in all his extremities, with distinct anæsthesia, great prostration, and confusion of ideas or complete loss of consciousness and involuntary evacuations. The pulse is small, weak, and slow, the skin is cool and pale or slightly cyanotic, and there may be respiratory disturbances amounting sometimes to dyspnœa. In a few hours or days death occurs amid general prostration and paralysis of the respiration.

(2) *Slight Shock causing Severe Symptoms at the moment of injury, but soon ending in Recovery.*—The patient is found immediately after the accident more or less paralysed in the lower extremities and sometimes in the upper also, and complaining of severe and general pain in the body or in the lower half of it. Some degree of anæsthesia is usually present, but is not often complete, and the bladder is not always paralysed. Soon after the accident the reflex functions, especially the tendinous reflex actions, may be found exaggerated, and the electrical reaction in the

paretic parts may be increased or depressed. Improvement begins in a few days. The patient is able to stand and walk, at first slowly, feebly, and with tremor; the pains disappear, and recovery is complete in a few weeks.

(3) *Severe Symptoms at first, followed by Protracted Illness of some years' duration; Recovery in most cases.*—The patient complains of motor weakness soon after the accident, which gradually increases until the extremities are paralysed. He also complains of pains which are sometimes more or less diffused, but are often situated in the back of the neck, loins, or along the vertebral column. Various paræsthesiæ are complained of, but anæsthesia is not usually well marked. Retention of urine sometimes occurs. There may be vomiting and loss of consciousness at first, and patients often manifest a high degree of mental irritability for a long time. The extremities are cold and livid, and the vertebral column is tender on pressure and often excessively sensitive. Gradual improvement now occurs, but the patient complains of great weakness; there may be slight atrophy of some of the muscles, and complete recovery may not take place for several years, and the patient may remain irritable and sensitive long after all the paralytic symptoms have disappeared.

(4) *Very Slight Symptoms at the beginning, but after a longer or shorter time a Severe Progressive Spinal Disease develops; the Result is Doubtful.* These cases are usually caused by a railway collision, and the symptoms are generally insignificant immediately after the injury. The patient has a sensation of having been severely shaken, suffers from momentary weakness and slight confusion of mind, but soon recovers, picks himself up, and walks about. On the next day, or several days, weeks, or even months later, more threatening symptoms set in. Pain appears in the back and limbs, and gradually increases in intensity; the patient feels feeble, suffers from mental depression and sleeplessness, with a strong tendency to emotional weakness, and cannot attend to his business. He also often complains of noises in the ears, and there may be slight deafness, and on attempting to read, the letters become confused. The subsequent course of the disease varies greatly in individual cases, but the following are the most usual phenomena observed: The gait is uncertain, straddling, stiff, and dragging, indications of disturbed co-ordination are present, and the legs become progressively feebler. There is stiffness of the back and of the general attitude. The back is painful when moved, and some of the spinous processes are tender on pressure. Girdle sensations, paræsthesiæ of all sorts, anæsthesiæ in varying degrees and in different situations, or hyperæsthesiæ, may be present. Weakness of the bladder and diminution of sexual power are generally present. The expression of the countenance is changed, the complexion becomes pale and sallow, and the general nutrition impaired. Marked atrophy occurs in individual muscles and groups of muscles, and it may at times be extensively distributed. Disturbances of circulation manifest themselves in the form of cold extremities and bluish complexion. The patient is irri-

table and timid, suffers from a feeling of constriction of the head, sleeplessness, weakness of memory and intelligence, impaired power of work, and in fact his whole character has undergone change.

The symptoms point to a meningo-myelitis, associated with more or less considerable disturbances of the cerebral functions. The subsequent course of the affection varies. Periods of apparent improvement and comparative health alternate with those of downward progress, but on the whole a favourable termination is seldom witnessed. Cases, however, occur in which the disease ceases to progress, and in which considerable improvement may take place.

§ 598. *Morbid Anatomy.*—In cases which have terminated fatally at an early period small extravasations of blood have been found in the cord and its membranes, but it is probable that these are of secondary importance to the molecular disturbance of the whole substance of the cord produced by the shock. It is probable that chronic meningitis and myelitis may develop out of concussion, and then the usual appearances which distinguish these affections will be found after death.

§ 599. *Diagnosis.*—Cases of concussion in which severe symptoms develop immediately after the injury may be mistaken for crushing or contusion of the cord, hæmatomyelia, or hæmatorrhachis. The course of concussion, however, is much more rapid and favourable. It may be inferred, when a severe paraplegia comes to a favourable ending in a few days or weeks without bed-sores or other grave symptoms, that the case is one of concussion.

The initial symptoms are more severe in concussion than in hæmatorrhachis, and in the latter affection the preponderant symptoms are those of pain and spasm, and the paralysis is slight, while in the former the opposite conditions obtain.

Cases of concussion, in which the symptoms are slight at first and gradually increase in severity, are not essentially different from myelitis and meningo-myelitis with a slow beginning, and the diagnosis must depend in great measure on the connection of the affection with an injury.

Crushing of the cord and concussion are frequently combined, so that it is almost impossible to distinguish between the symptoms which are due to the one and those due to the other,

and the diagnosis can only be made after the disappearance of the symptoms of concussion.

§ 600. *Prognosis.*—In the severest form of concussion known as shock the prognosis is always grave, but the slighter cases of the kind generally recover. Compared with the severity of the symptoms the prognosis is good, and indeed the severe initial symptoms seem to be the very cases to warrant a favourable prognosis as compared with those the development of which is slow.

Even in cases where symptoms of meningitis or myelitis appear, the prognosis is not absolutely bad, but when, after one or two years of rational treatment, no further progress is made, recovery is hardly to be expected.

§ 601. *Treatment.*—The treatment must vary according to the form assumed by the affection.

In cases with severe initial symptoms the treatment must be first directed against the shock. The patient must be placed in the recumbent posture, and warmth applied to the body, and full doses of some stimulant, such as wine, coffee, tea, hot spirit and water, or drugs like aromatic spirit of ammonia ether, musk, and camphor, must be at once administered.

Symptoms of reaction must be treated by absolute rest in a suitable position. If the patient cannot bear lying on his face or side, Erichsen recommends him to lie on his back on a couch tilted at its foot. The usual remedies for myelitis must now be employed.

(V.) TUMOURS OF THE SPINAL CORD AND MEDULLA OBLONGATA.

§ 602. Tumours are rarely found in the substance of the spinal cord, but are more frequent in the medulla oblongata.

§ 603. *Varieties of Tumours found in the Spinal Cord and Medulla Oblongata.*—The following are the more usual tumours found in the substance of the cord and in the medulla oblongata :—

1. *Gliomata.*—The tumour is generally of a rounded or more or less

elongated shape, but at other times it may extend the whole length of the spinal cord, as in a case recorded by myself.

2. *Myxo-Gliomata.*—This form of tumour is only a variety of Glioma.

3. *Glio-Sarcomata*, *Myxo-Sarcomata*, *Sarcomata*, and *Carcinomata* are only rarely found in the substance of the cord and the medulla oblongata.

4. *Fibromata* have been observed in the medulla oblongata, either growing from the ependyma or in the medullary substance.

5. *Solitary Tubercle.*—This is one of the most frequent tumours of the spinal cord. It may appear at any period of life, but is relatively more frequent in youth than in old age. The favourite sites of solitary tubercles are the cervical and lumbar enlargements, especially the latter. They may be situated either in the grey or white substance, and vary in size from that of a hemp-seed to a hazel-nut. Solitary tubercle may attain to the size of a walnut in the medulla oblongata.

6. *Gummata or Syphilomata.*—These tumours are on the whole rarely found in the cord and medulla oblongata.

7. The *cystic* dilatations, which have been called hydromyelus or syringomyelia, may be reckoned amongst the tumours of the spinal cord when they compress the nervous substance; they are often associated with new growths, such as gliomata, myxo-gliomata, and myxo-sarcomata.

§ 604. *Etiology.*—The causes of tumours of the spinal cord and medulla oblongata are very obscure, but it is probable that injuries, such as blows or jars of the spinal column, may act as exciting causes.

§ 605. *Symptoms.*

(1) *Symptoms of Tumours of the Spinal Cord.*—The symptoms caused by the growth of a tumour within the substance of the cord are very variable, but are generally the same as those of *compression myelitis.* They are paraplegia, anæsthesia, increased reflex action, paralysis of the bladder and rectum, muscular atrophy, and bed-sores. The development of paraplegia may be preceded by shooting pains in the limbs, girdle pains, and various paræsthesiæ. Indefinite initial symptoms may persist for a long time, and then paralysis develop suddenly, occasioned by an acute attack of transverse myelitis or hæmorrhage into the substance or neighbourhood of the tumour. The development of paralysis proceeds at other times more slowly. One limb, probably an upper extremity, becomes first affected, the other extremities becoming gradually implicated until the paraplegia is complete.

In slowly-growing and dense tumours the nerve fibres appear to be thrust aside without being ruptured, and extensive changes may sometimes be found after death in the cord while only very slight and indefinite symptoms existed during life. In a few cases the symptoms are caused by a diffused ascending myelitis, while in occasional instances the disappearance of the reflex excitability and the occurrence of extensive muscular atrophy indicate that the grey substance has been destroyed by a secondary descending myelitis or by a descending extension of the new growth.

Various other symptoms may appear in the course of the disease, according to the situation of the tumour and the direction in which it is growing. The chief groups of symptoms which may be thus caused are progressive muscular atrophy, ataxia, and spastic paralysis.

(2) *Symptoms of Tumours of the Medulla Oblongata.*—Tumours in the substance of the medulla oblongata may remain latent for a long time, and may either cause no recognisable symptoms during life, or not until a few hours or days before death, which results from asphyxia.

The most prominent of the initial symptoms are paroxysmal attacks of headache, situated in the occiput or nucha, dizziness, violent vomiting, tinnitus aurium, hiccough, and occasionally epileptoid attacks. These symptoms persist and grow worse, while others are soon superadded to them, the latter being caused by compression of some of the fibres of the bulbar nerves as they pass through the medulla, or by destruction of portions of the bulbar nuclei. The most usual symptoms caused by compression of the fibres of the nerve roots are distortion of the face, unilateral and bilateral paralysis of the tongue, internal squint, disorders of articulation, vocalisation, and deglutition, a nasal tone of voice, and disorders of circulation and respiration. There may also be more or less extensive paralysis of the extremities, consisting of unilateral or bilateral paresis, with or without contractures. Hemiplegia, alternating with paralysis of the facial, abducens, or hypoglossal nerves, forms a characteristic symptom. Reflex action is often increased in the extremities.

Disorders of motor co-ordination have sometimes been

observed, which may resemble those of locomotor ataxia, but more commonly a cerebellar reel (§ 86), probably caused by interference with the peduncles of the cerebellum.

The sensory are not so well marked as the motor disturbances. They generally consist of pain, and paræsthesiæ in the nucha, back, and extremities, and anæsthesia may occasionally be present in the trunk and extremities. When the tumour or the inflammatory action which surrounds it extends into the pons, there may be paralysis of the masticatory muscles, hyperæsthesia, neuralgic pains, and subsequently anæsthesia of one or both sides of the face, while one or both eyes may suffer from neuroparalytic ophthalmia. Deafness on one or both sides may be met with, and double optic neuritis, amblyopia or amaurosis are almost constant accompaniments of the presence of tumour within the cranial cavity.

Glycosuria and polyuria may be present, and the temperature of the body is generally persistently depressed.

Psychical disturbances, such as loss of intelligence and memory, have occasionally been observed at an early period, and are almost constantly present towards the termination of the disease. These symptoms are probably caused by the hydrocephalus internus which almost always accompanies tumours of the medulla. The effusion into the ventricles may be, to some extent, caused by the pressure of the tumour on the veins, especially the venæ galeni; but probably depends rather upon the hindrance offered by the tumour to the return of the cerebrospinal fluid into the sub-arachnoid space.

As the disease advances, the headache and paralytic symptoms grow worse, the vomiting and hiccough may become incessant, epileptic attacks occur more frequently, coma supervenes, and the patient dies from asphyxia.

§ 606. *Localisation of the Tumour in the Cord and Medulla Oblongata.*

1. *Tumours of the Spinal Cord.*—The level at which the cord is affected can generally be made out by the height to which the paralysis extends, and by the condition of the various cutaneous and deep reflexes. The extent to which the grey substance is diseased, either by the invasion of the tumour or by secondary myelitis, can be ascertained by the muscular atrophy and other trophic affections associated with the paralysis; while

implication of the posterior or lateral columns may be respectively recognised by the presence of ataxia or spastic paralysis.

2. *Tumours of the Medulla Oblongata.*—(*a*) Tumours in the *anterior pyramids* of the medulla produce unilateral or bilateral paralysis of the muscles of the extremities and trunk, accompanied by contractures and increased tendon reflexes. Paralysis of the bladder is sometimes superadded, but sensibility remains unimpaired. In the later stages of the disease the symptoms of bulbar paralysis are superadded.

(*b*) Tumours on the *floor of the fourth ventricle* may cause vomiting, hiccough, glycosuria and polyuria, slow pulse, and respiratory disorders. The symptoms which indicate paralysis of cranial nerves may be present, but paralysis of the extremities if present at all is a late symptom. Sensory disorders are rare, but some degree of ataxia or reeling may be present.

(*c*) Tumours of the *restiform body* may give rise to sensory disorders in the extremities, unilateral anæsthesia in the face, auditory troubles, and ataxic walk, but our knowledge of the symptoms caused by these tumours is not sufficiently accurate.

(*d*) Tumours of the *formatio reticularis* and *olivary bodies* cause no symptoms by which their presence can be recognised, except those common to intracranial growths. When, however, these tumours increase to such a size that they compress the bulbar nuclei, the fibres of the bulbar nerves in their passage through the medulla, or the anterior pyramids, the grouping of the symptoms may be such as to render it possible to make an accurate diagnosis of the localisation of the lesion. The following case, for the notes of which I am indebted to Mr. Hodgson, who was then one of the House Physicians of the Royal Infirmary, well exemplifies the grouping of the symptoms which may take place from a tumour commencing in the *formatio reticularis*:—

William B——, æt. eight years, was admitted February 17, 1880, into the Royal Infirmary, under the care of Dr. Ross. He is a well-nourished boy for his years, and his mental faculties do not appear to be in any way impaired. He complains of headache, and the occipital region is sensitive to touch. Both eyeballs are inverted, the external recti muscles being completely paralysed. The right pupil measures 5 and the left 3 mm., but both contract readily to light. There is slight facial paralysis on the right side extending to the eyelid of the same side, which cannot be closed. The soft palate is loose and pendulous, and the uvula occupies the middle line, until a reflex contraction of the palatal muscles is excited when it assumes a curved form, the point being directed to the right and the convexity to the left. There are no sensory disorders of the face or body, no loss of taste, deafness, paralysis of the tongue, or recognisable paralysis of the extremities. The reflex contractility of the right facial muscles is diminished, but they still contract to a faradic medium current. The galvanic contractility is also somewhat diminished, but there are no qualitative changes. When the child stands his head is inclined to the left, but it is difficult to know whether this attitude is due to

paresis of the right sterno-cleido-mastoid muscle, or to a voluntary effort to correct the false images caused by the double internal squint. When he walks his gait is staggering, and he manifests a constant tendency to fall towards the right side, yet there is no paralysis of the right lower extremity. The cervical glands at the angles of the jaws and along the sterno-mastoid muscles on both sides are slightly enlarged. The heart and lungs are healthy. The urine is acid, S.G. 1026, contains no albumen or sugar, and is normal in quantity. The reflex of the sole, and the cremasteric, abdominal, epigastric, gluteal, and interscapular reflexes are normal.

March 22. When the patient stands he reels from side to side, and manifests a greater tendency to fall to the left than the right, and the patellar-tendon reflex is more marked in the left than right leg. There is also decided paresis of both the lower and upper extremity on the left side. Ophthalmoscopic examination reveals double optic neuritis.

April 9. The left half of the body is distinctly paralysed, with muscular rigidity, the right arm is feeble, and the patient cannot stand. The face is now symmetrical, and there is decided loss of expression, while both eyes remain open during sleep, and the patient cannot close them. The power of articulation is interfered with, and the food has to be placed far back on the dorsum of the tongue in order to secure deglutition. The masticatory muscles are feeble, the jaw hangs loose, the mouth is half open, and saliva dribbles constantly from the mouth. The simple movements of the tongue can still be readily effected.

The mental faculties are becoming blunted, but so far as can be judged the senses of taste and hearing are unimpaired, while even the sense of sight is fairly good. Sensibility to pain and touch are impaired on both sides of the face and in the limbs.

May 11. Since last report the paralytic phenomena have become gradually worse. The left arm and leg are now completely paralysed, while the right limbs are very feeble. There is double facial paralysis, masticatory paralysis with contracture of the muscles so that the jaws are held close together, and increasing difficulty of articulation and deglutition. The right conjunctiva has been for some time red, and covered by tenacious secretion, and the cornea is now becoming cloudy. The patient has had an attack of severe vomiting about two weeks ago, but it has not recurred.

May 20. Since last report the symptoms have become gradually worse. All the limbs are paralysed, the right cornea is ulcerated, while the left conjunctiva is injected and covered by tenacious secretion. Inability to swallow was manifested this morning, and he died in the afternoon from arrest of respiration.

At the autopsy no changes worth noting are observed in the nervous system with the exception of the medulla oblongata and pons. On the fourth ventricle being exposed, a tumour is observed to project from its surface on a level with the striæ medullares. It is about the size of a

pigeon's egg, and the greater portion of it lies to the right of the median raphé, while it projects forwards into the substance of the medulla and pons. Two tumours, each about the size of a hazel-nut and occupying symmetrical positions on each side of the middle line, are observed in the upper part of the ventricle immediately under the valve of Vieussens. A few scattered miliary tubercles are found in the apices of the lungs. The tumours in the pons were tubercular.

Dr. Leech, who kindly transferred this case to my care, told me that the symptoms began by internal squint of the right eye, and staggering gait, with a tendency to fall to the right side, these symptoms being followed by right facial paralysis. The symptoms were so definite in this case that an accurate diagnosis could readily be made. The headache, gradual invasion of the symptoms, and the presence of double optic neuritis, showed that the case was one of intracranial tumour, while the swelled glands in the neck, and other circumstances, indicated its probable tubercular nature. The succession of the various groups of bulbar symptoms were so definite that there could be no possibility of mistaking the localisation of the main focus of disease. The early paralysis of the external rectus of the right side showed that the primary focus began on a level with the sixth nerve, and to the right of the median raphé. Suppose, then, that the tumour began to grow in the formatio reticularis on the right side (*Fig.* 185, *ar*) on a level with the sixth nerve (RVI), and

FIG. 185.

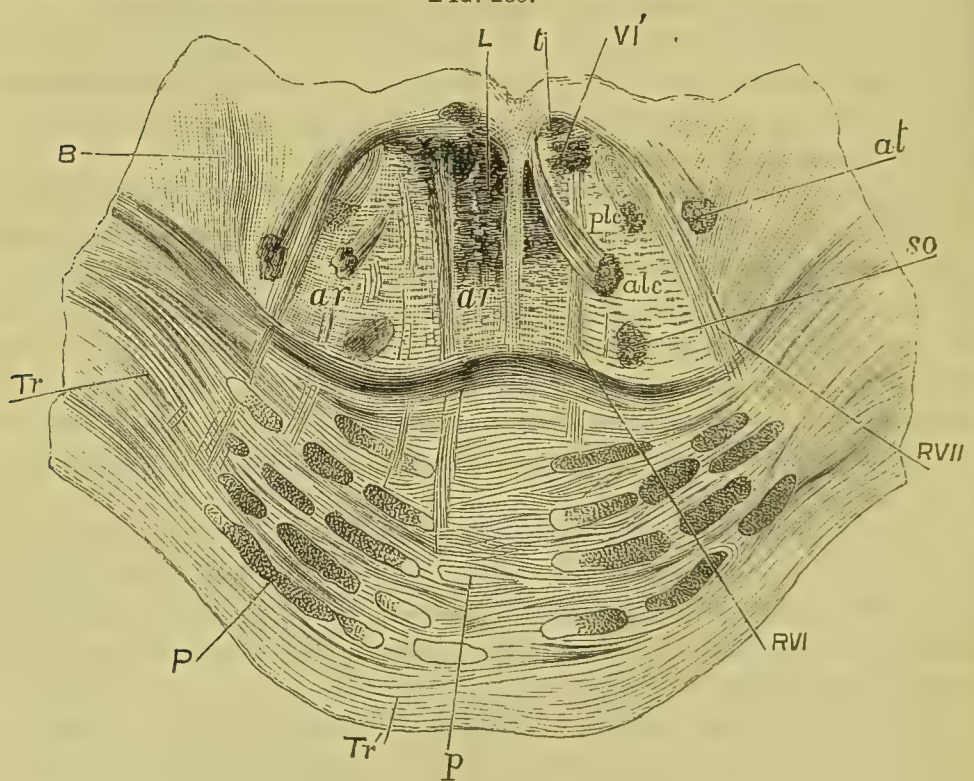


FIG. 185. *Transverse Section of the Pons on a Level with the Points of Origin of the Sixth and Seventh Nerves.* (See *Fig.* 126, p. 40.)

that it grew equally in all directions. It would first produce paralysis of the right external rectus, and as it grew forwards it would press on the transverse fibres of the pons, and produce reeling, with a tendency to fall to the right, or the symptoms which would have been caused by disease of the middle peduncle of the cerebellum. As the tumour enlarged to the right it would press on the fibres of the seventh nerve (RVII), and cause paralysis of the right muscles of the face, having the characters of a peripheral paralysis. As it grew towards the left it would press on the left sixth, and then on the left seventh nerve, causing successively internal squint of the left eye and left facial peripheral paralysis. As it grew still further forwards it would compress the fibres of the pyramidal tract, first on the right side, causing crossed paralysis of the left extremities, and then on the left tract (*Fig.* 185, *P*, *p*), causing paralysis of the extremities on the right side.

Again as the tumour extended downwards it would compress the ninth, tenth, twelfth, and probably also the eleventh nerves, and so give rise to the more purely bulbar symptoms present, such as the vomiting, and disorders of articulation and deglutition. As it extended upwards it would compress the motor and to some extent the sensory divisions of the fifth nerves, causing the masticatory paralysis, the facial sensory disturbances, and the neuro-paralytic ophthalmia. Not only was an accurate localisation of the lesion capable of being made in this case, but we were even able to anticipate to a large extent the later symptoms which supervened. When paralysis of the left rectus occurred, we were able to foretell that it would soon be followed by peripheral paralysis of the left side of the face. It was also possible to anticipate that left hemiplegia would supervene, which would end in general paralysis if the patient survived long enough, and that the upward and downward extensions of the tumour would give rise to motor and sensory disturbances in the region of the fifth on the one hand, and difficulties of articulation and deglutition on the other.

§ 607. *Course, Duration, and Terminations.*—The course of intramedullary tumour is usually very protracted, and the patient may live for many months after complete paraplegia is established. Intramedullary tumours are, with the exception of gummata, uniformly fatal. Death may be caused by cystitis, bed-sores, and their consequences, arrest of respiration when the myelitis takes an ascending course, or intercurrent disease.

The course of tumours of the medulla oblongata is probably uniformly fatal. The disease may, however, extend over a period of years, with considerable variations in the intensity of the symptoms, but, as a rule, it proceeds uninterruptedly to a fatal termination.

§ 608. *Diagnosis.*—It is not always possible to make a decided diagnosis; but the presence of a tumour may be suspected when the intensity of the paralytic symptoms fluctuate, or when symptoms of central myelitis or hæmatomyelia supervene upon those of a long-continued and insidious spinal affection. Evidence of scrofula, tuberculosis, or syphilis may greatly aid the diagnosis. The initial symptoms of irritation are generally of less intensity in intramedullary than meningeal tumour, and the former is more apt to give rise to an ascending myelitis than the latter.

Tumour of the *medulla oblongata* may be recognised with some certainty when the general symptoms of an intracranial growth are accompanied by signs of local irritation or paralysis of some of the bulbar nerves, and when in addition there are obstinate vomiting, hiccough, glycosuria, or polyuria. When ataxia or reeling is a prominent symptom of tumour of the medulla oblongata, or when tumours of the cerebellum implicate the medulla secondarily, it may be impossible to distinguish tumours in the two regions. If, however, the patient has been under observation from an early period of the disease, the mode of invasion and succession of the symptoms generally enables the diagnosis to be accurately made.

In *progressive bulbar paralysis* the regular symptoms of tumour of the medulla oblongata, such as headache, vomiting, hiccough, disorders of hearing, glycosuria, amblyopia, epileptiform convulsions, and double optic neuritis, are never present.

It may be impossible to distinguish tumours in the substance of the medulla from those which grow in the membranes or surrounding bones, but signs of irritation and paralysis in the region of distribution of the cranial nerves are probably less prominent in the former than in the latter.

§ 609. *Prognosis.*—Except in the case of syphilitic gummata, the prognosis is absolutely unfavourable.

§ 610. *Treatment.*—It is only when syphilis exists that treatment is of any avail, when large doses of iodide of potassium should be promptly administered. In other cases the treatment should be the same as that of acute and chronic myelitis.

CHAPTER IX.

DISEASES OF THE MEMBRANES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

(I.) VASCULAR DISEASES OF THE MEMBRANES.

1. *Hyperæmia of the Spinal Membranes.*

§ 611. Hyperæmia of the spinal membranes cannot be separated from hyperæmia of the spinal cord, and has already been sufficiently considered.

2. *Hæmatorrhachis—Meningeal Apoplexy.*

§ 612. Hæmatorrhachis implies any effusion of blood in, about, or between the spinal meninges.

§ 613. *Etiology.*—The disease is more frequently observed in men than women, but very little is known with respect to the predisposing causes of the affection.

The most usual exciting causes are injuries of the spinal column, such as fractures and contusions. Caries of the vertebræ has in some cases led to injuries of the vessels of the cord and to hæmorrhage from them. Excessive bodily exertion, the violent spasms of epilepsy, eclampsia, and tetanus, the sudden suppression of accustomed discharges, and all the circumstances which induce spinal hyperæmia may act as exciting causes of meningeal apoplexy.

Meningeal hæmorrhage may occur in scorbutus, purpura hæmorrhagica, smallpox, and typhoid fever and other acute infectious diseases. Aneurisms have been frequently known to rupture into the vertebral canal, and blood effused into the brain or cerebral membranes may sometimes pass down into the spinal canal.

§ 614. *Symptoms.*—The disease begins suddenly. The patient is attacked with violent pains and falls down, but generally without loss of consciousness. The development of the disease is occasionally slower and more gradual, and premonitory symptoms, such as lumbar pains and headache, may then be present. The severity of the symptoms may increase after a few hours or days.

The characteristic symptoms are those of irritation and subsequent paralysis. The phenomena of irritation consist of a violent pain, localised at a spot corresponding to the seat of the hæmorrhage and radiating in various directions along the distribution of the nerves, the roots of which are first implicated. Eccentric sensations, such as pain, formication, burning, and tingling, are associated with the local pain, and hyperæsthesia is also mentioned as being occasionally present.

The chief symptoms of motor irritation are spasmodic jerking of the muscles which may sometimes increase to general convulsions, trembling of the extremities, and tonic spasm and contracture of various groups of muscles. During this stage the vertebral column is stiff and painful, so that it is impossible for the patient to raise himself in bed or to assume the sitting posture.

Paralytic symptoms, however, soon supervene in the lower extremities, but seldom amount to complete paraplegia. There is also a more or less distinct anæsthesia of the lower extremities, associated with feelings of furriness and numbness, as well as with sensations of swelling and heaviness in the limbs and trunk. The distribution of the symptoms depends upon the seat of the lesion.

Reflex excitability is said to be depressed in some cases, but probably this only occurs in the regions supplied by the nerve roots directly affected. Weakness of the bladder and rectum is rarely mentioned, but may be present in severe cases. Fever is absent at first, but may occur during the irritative reaction which sets in on the second or third day.

§ 615. *Varieties of Meningeal Hæmorrhage.*

(1) When the *cervical* region is affected, the attack begins with pain in the arms and shoulders, stiffness in the neck, and pain in the occiput.

The anæsthesia and paralysis are most marked in the upper extremities, and oculo-pupillary symptoms, difficulty of breathing, and a retarded and weak pulse may be present.

(2) When the *dorsal* region is affected, there is pain in the back and abdomen, girdle-pain, stiffness in the dorsal part of the spine, paralysis of the legs and abdominal muscles, while the reflex excitability is retained in the lower extremities.

(3) When the *lumbar* region is affected, there are pain and stiffness in the loins, tearing pains in the lower extremities, perinæum, bladder, and genitals, well marked paralysis of the lower extremities with loss of reflex activity, and paralysis of bladder and rectum.

§ 616. *Course and Terminations*.—The symptoms remain stationary for some time, but improvement usually sets in sooner or later. The symptoms of inflammatory reaction are seldom prominent or soon disappear. The disease usually runs a favourable course when it is uncomplicated, and in a few weeks or months a tolerably good recovery may be anticipated. Death, however, is not a rare occurrence when the hæmorrhage occurs at a high level so as to affect the respiratory centres. If the extravasation be large, the severity of the compression may lead to complete paraplegia, cystitis, bed-sores, and their usual consequences.

§ 617. *Diagnosis*.—The diagnosis must be founded on the sudden occurrence of the illness, the peculiar combination of phenomena of irritation and paralysis, the absence of severe cerebral symptoms, the paraplegic character of the symptoms, the speedy improvement, and usually favourable termination. Meningeal hæmorrhage may be distinguished from concussion of the spinal cord by the absence of spasm and other symptoms of irritation in the latter. From spinal apoplexy the meningeal affection may be distinguished by the severity and suddenness of the paralysis and the high degree of anæsthesia in the former.

Both meningitis and myelitis develop more slowly than meningeal hæmorrhage, and both are accompanied by fever. In central spinal myelitis marked anæsthesia is never wanting, and paralysis is usually complete from the beginning.

§ 618. *Prognosis*.—The prognosis is always doubtful; but if the extent of the hæmorrhage be not great, and if it be not

caused by a permanent lesion, such as the growth of a tumour, improvement and complete recovery may take place. When the hæmorrhage is considerable, or when it is situated in the cervical region, or if the symptoms of myelitis supervene, the prognosis becomes unfavourable. When, on the other hand, the hæmorrhage is small, the reaction moderate, and the patient young, the prognosis is favourable.

§ 619. *Treatment.*—When the symptoms of meningeal hæmorrhage have occurred, absolute rest in the horizontal posture, with the patient lying on his side or face, should be maintained. The primary object is to prevent the bleeding from extending, the most usual remedy being application of ice to the vertebral column, and ergot may be given internally when symptoms of inflammatory reaction set in. Leyden recommends mercurial inunction and repeated small doses of calomel. When the period of reaction has passed, absorption may be promoted by the external and internal use of iodine, and the galvanic current.

(II.) INFLAMMATION OF THE SPINAL DURA MATER.

Pachymeningitis Spinalis. Peri-meningitis.

§ 620. Pachymeningitis may be subdivided into two varieties: (1) External pachymeningitis, when the morbid products are deposited in the loose cellular tissue between the dura mater and vertebræ; and (2) Internal pachymeningitis, when the inner surface of the dura mater is attacked.

(i.) Pachymeningitis Spinalis Externa. Peri-pachymeningitis.

External pachymeningitis consists of inflammation of the outer layers of the dura mater and the cellular tissue surrounding it.

§ 621. *Etiology.*—Various diseases in and about the vertebral column, such as vertebral caries and bed-sores, constitute the most frequent causes of the affection.

§ 622. *Symptoms.*—Pain in the back, which varies in its seat and extent according to the locality of the lesion, is one of

the most constant and important symptoms of the affection. Rigidity of the back, which renders it difficult and painful for the patient to sit up, spasm of various groups of muscles, eccentric pains in the form of a girdle or shooting into the extremities, formication and slight hyperæsthesia of the skin, are the usual symptoms complained of.

After a time symptoms of compression of the cord are gradually superadded. Both sensory and motor paralysis may occur slowly or suddenly, and muscular tension, increased reflex action, especially increased tendon reflex, paralysis of the sphincters, and bed-sores appear after a time. These symptoms are caused partly by local compression of the cord and partly by myelitis and secondary degenerations.

The symptoms may develop in an acute or chronic form. In the acute purulent forms the prominent symptoms are caused by irritation, while in the chronic fibrinous form the symptoms of compression and paralysis of the cord predominate.

§ 623. *Course.*—When pachymeningitis externa accompanies caries of the vertebræ the disease is frequently arrested and the paralytic symptoms disappear. In the severer cases its course may vary, but is usually protracted, and only after the lapse of many weeks is there a termination in recovery or death.

§ 624. *Morbid Anatomy.*—The essential nature of the affection consists of an inflammation of the outer layers of the dura mater and surrounding cellular tissue, with exudation of a purulent, plastic, or tuberculous nature. The exudation has been found as much as half an inch in thickness. The inner surface of the dura is also thickened and opaque, and frequently covered with a thin fibrinous deposit. The pia mater and arachnoid do not often participate in the affection, but they have at times been found adherent to the dura, opaque, and infiltrated with pus. The morbid changes are usually limited to a small portion of the cord, even when they extend over the greater part of the dura mater.

The cord itself is more or less compressed, flattened, anæmic, and often softened. Red softening and hyperæmia are found in the neighbourhood of the compressed portion, and in chronic

cases ascending and descending secondary degenerations are observed. The nerve roots which pass out at the seat of the pachymeningitis are compressed and atrophied, or inflamed and soft. The primary lesion which has produced the pachymeningitis will of course be found on post-mortem examination.

§ 625. *Diagnosis.*—The diagnosis is chiefly founded on the slowly-increasing symptoms of meningeal irritation and of compression of the cord. Valuable aid to diagnosis may be obtained by the discovery of abscess or the other morbid changes near the vertebral column.

§ 626. *The Prognosis* depends upon the nature of the primary lesion which has caused the pachymeningitis.

§ 627. *Treatment.*—The first aim of treatment is to remove the effects of the original lesion. The use of brine baths, iodide of potassium and iodide of iron, frictions with mercurial ointment, and various other remedies have been employed, and Charcot recommends the use of the cautery for obstinate cases.

(ii.) Pachymeningitis Interna (Hypertrophica et Hæmorrhagica).

§ 628. *Definition.*—Internal pachymeningitis is an inflammation chiefly of the inner surface of the dura mater, with deposition of morbid products between the dura mater and arachnoid. There are two forms of the disease: (1) Pachymeningitis interna hypertrophica, consisting of thickening of the dura mater; and (2) pachymeningitis interna hæmorrhagica, or hæmatoma of the spinal dura mater, consisting of the formation of successive pseudo-membranous layers, between which blood is effused.

§ 629. *Etiology.*—The causes usually assigned to the hypertrophic form are exposure to cold and damp, and excessive use of alcohol.

The hæmorrhagic form often accompanies hæmatoma of the cerebral dura mater, and is produced by the same causes. It is often associated with dementia paralytica and other mental disorders. It appears sometimes to result from alcoholic excess, and Leyden has described a traumatic form.

§ 630. *Symptoms.*—The *hypertrophic form* usually occurs in the cervical region, and has been described by Charcot under the name of *pachyméningite cervicale hypertrophique.* During the first stage of the disease, which lasts two or three months, the symptoms of sensory irritation predominate. The most usual of these symptoms are neuralgiform pains in the neck and head, which extend to the shoulders and arms, and a painful sensation in the upper part of the chest, as if the patient were bound by a tight cord. The muscles of the neck are in a state of spasmodic rigidity, the patient often complains of formication and numbness of the upper extremities, and cutaneous trophic disturbances, such as vesicular and herpetic eruptions, may make their appearance on the upper extremities.

The transition to the second stage is characterised by the gradual development of paralysis and muscular atrophy. At first there is simply paresis of the extremities, which after a time becomes developed into a more or less complete paralysis, with flaccidity of the affected muscles. In the upper extremities the paralysis often predominates in the muscles supplied by the ulnar and median nerves, while those supplied by the musculo-spiral nerve are comparatively spared. The consequence of this mode of distribution of the paralysis is that the hand is maintained in the position of exaggerated extension, the phalanges are flexed on the metacarpal bones and upon one another, so that the fingers are held like claws, and the thumb

FIG. 186.

FIG. 186 (After Charcot). *Attitude of the Hand in Pachymeningitis Cervicalis Hypertrophica, when the disease is situated on a level with the lower half of the cervical enlargement of the spinal cord.*

is extended (*Fig.* 186). This position of the hand is not, however, so much a sign of the disease as it is of its locality. When this deformity is present, the lesion is situated in the lower half of the cervical enlargement of the cord, and the distortion indicates that the roots of the ulnar and median nerves stand at a lower level in the cord than those of the musculo-spiral nerve. When the disease is situated in the upper cervical region and implicates the upper portion of the cervical enlargement, the resulting deformity differs greatly from that just described. The distortion of the hand present under those circumstances is shown in (*Fig.* 187), taken from a photograph of a remarkable case under the care of Dr. Leech. The arm is held close to the side, the forearm is extended on the arm and strongly pronated, the hand is flexed on the forearm, the fingers are in a line with or only slightly extended on the metacarpal bones, and the phalanges are extended upon one another, while the thumb is flexed into the palm (*Fig.* 187).

Fig. 187.

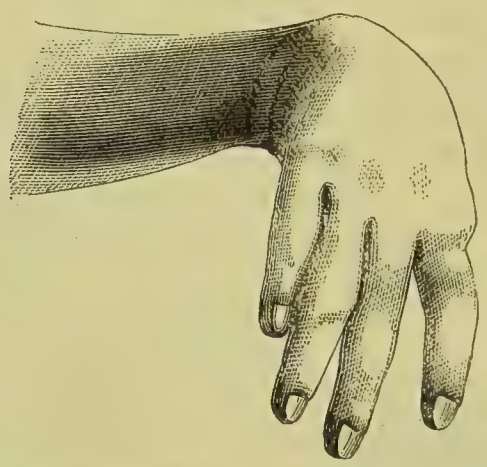


Fig. 187. *Attitude of the Hand in Pachymeningitis Cervicalis Hypertrophica, when the lesion is situated on a level with the upper half of the cervical enlargement.*

All the muscles of the forearm and hand were no doubt more or less paralysed in this case, but it will be seen that the muscles supplied by the musculo-spiral nerve were on the whole more affected than those supplied by the ulnar and median nerves.

The paralysis is accompanied by marked atrophy and loss of the faradic contractility of the affected muscles. After a time

muscular tension and contractures appear in the paralysed muscles, and circumscribed areas of anæsthesia may be observed in the skin of the upper extremities. At a later period the lower extremities become paralysed, and contractures with increase of the tendinous reflexes appear after a time in the muscles, similar to that which occurs in primary lateral sclerosis. The muscles of the lower extremities do not undergo atrophy like those of the upper extremities, or at least not until a late period of the disease. In severe cases complete paraplegia, with marked anæsthesia, paralysis of the bladder, and bed-sores arise, leading to a fatal termination.

The symptoms are at first due to compression of the cord by the dura mater contracting around it and to transverse myelitis at the spot compressed, and at a later period to descending degeneration of the pyramidal tracts. This form of the disease has a resemblance to progressive muscular atrophy, amyotrophic lateral sclerosis, and other diseases attended by atrophy. The most important points of distinction are the stage of pains, the partial anæsthesia, and the paraplegia without atrophy.

In *hæmorrhagic pachymeningitis interna* the symptoms are very obscure, and usually complicated with those of co-existing cerebral disease. They point to a slow meningitis, and consist of pains in the loins and back, tearing pains in the extremities, stiffness of the vertebral column, increasing muscular weakness which may gradually develop into complete paraplegia, contractures, various degrees of cutaneous hyperæsthesia and anæsthesia, and weakness of the bladder. If a patient with these symptoms be at the same time suffering from cerebral paralysis and chronic alcoholism, hæmorrhagic pachymeningitis may be suspected.

The diagnosis is not readily made in this form of the affection.

§ 631. *Morbid Anatomy.*—In the *hypertrophic form* the dura mater is much thickened and consists of a large number of concentric layers of cicatricial connective tissue. The pia mater and arachnoid are also thickened and adherent to the dura mater. The thickened membrane may compress the cord on one side, or from behind forwards, but usually embraces it like a ring. The compressed portion of the cord is pale and soft, and

generally presents all the characteristics of transverse myelitis. The nerve roots on a level with the lesion are compressed, and the muscles supplied by the nerves issuing from them are in a condition of degenerative atrophy.

Fig. 188.

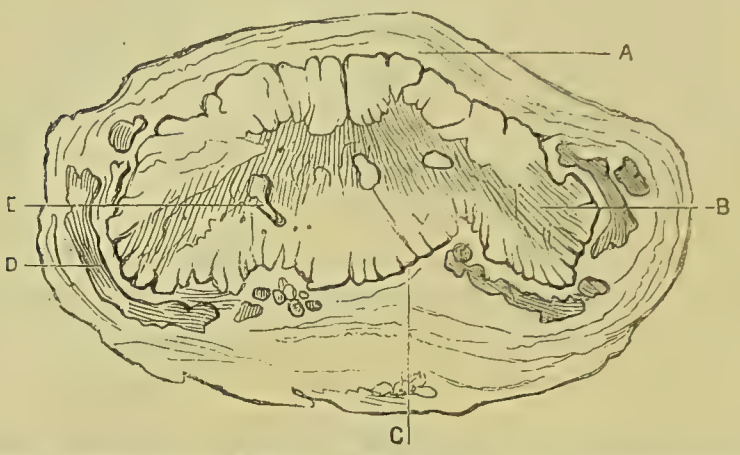


Fig. 188 (From Charcot and Joffroy). *Transverse Section of the Middle of the Cervical Enlargement, from a case of Hypertrophic Cervical Pachymeningitis.*—A, Hypertrophied dura mater; B, Roots of the nerves traversing the thickened membranes; C, Pia mater adherent to the dura mater; D, Lesion of chronic myelitis; E, Cystic formation in the grey substance.

In the *hæmorrhagic form* a portion of the dura mater is covered by a soft, rusty-brown exudation composed of fibrine and connective tissue, and studded with numerous hæmorrhagic extravasations. The exudation contains numerous blood-crystals, pigment, detritus of decomposed blood, and is only slightly adherent to the dura mater or arachnoid, so that it is readily stripped off.

§ 632. *Treatment.*—The treatment is the same as that of meningitis in general.

In the chronic form counter irritation, preparations of iodine, galvanism, and the use of baths or the cold water treatment deserve a trial.

(III.) INFLAMMATIONS OF THE PIA MATER AND SPINAL ARACHNOID.

Leptomeningitis Spinalis—Perimyelitis and Arachnitis.

§ 633. Spinal leptomeningitis presents many varieties, but for practical purposes it is sufficient to subdivide the disease into the acute and chronic forms.

(i.) Leptomeningitis Spinalis Acuta.

The acute variety begins suddenly with violent sensory disturbances, attended by marked fever.

§ 634. *Etiology.*—The predisposing causes of the affection are a scrofulous or tubercular constitution, insufficient food, damp dwellings, and sexual and other excesses. The disease attacks by preference children, young persons, and the male sex.

The most important of the exciting causes are exposure to cold, wounds and injuries of the vertebral column, and extension of inflammation from neighbouring structures. Tubercular basal meningitis is usually accompanied by a spinal affection of the same nature. Spinal meningitis may occur along with or during convalescence from pneumonia, acute articular rheumatism, and other febrile and infectious diseases. Epidemic cerebro-spinal meningitis will be subsequently described.

§ 635. *Symptoms.*—Acute spinal meningitis is generally complicated by a simultaneous affection of the cerebral pia mater, so that it is not always easy to separate the purely spinal from the cerebral symptoms.

The disease begins suddenly, but the outbreak of the characteristic phenomena may be preceded by premonitory symptoms, such as general heaviness and depression, slight chilliness, gastric disturbances, transitory pains in the head and back, restlessness, and sleeplessness.

The characteristic symptoms of the disease are ushered in by a rigor followed by pyrexia of irregular type, and if the pia mater of the brain be affected, vomiting and severe cerebral symptoms are also present. The patient now complains of an intense, deep-seated, boring pain in the loins, back, or nape of the neck, corresponding to the localisation of the lesion. The pain is increased by movements of the vertebral column and by pressure on the spinous processes. The pain is subject to remissions, followed by exacerbations of great severity, and radiates from the vertebral column round the trunk, shooting in all directions through the extremities.

The muscles of the vertebral column are in a state of spas-

modic rigidity. When the inflammatory process is limited, the rigidity may be limited to certain portions of the vertebral column corresponding to the situation of the lesion, but the spasm may extend over the whole length of the spine, so as to resemble a tetanic seizure.

The muscles of the extremities are also subject to painful tension and spasm. The limbs are then rigid and immovable or the subjects of painful twitchings.

Cutaneous and muscular hyperæsthesia is often present in the extremities and trunk in places corresponding to the areas of distribution of the nerves whose posterior roots are involved in the inflammation. Reflex activity is at first increased and subsequently diminished.

Functional disturbances of the bladder and rectum appear at an early period of the disease, probably owing to a spasm of the sphincters. When the cervical part of the cord is involved in the inflammation the muscles of respiration become rigid and painful, producing difficulty of breathing, which may increase to such an extent as to cause asphyxia.

When the cerebral pia mater is implicated the patient may suffer from vertigo, violent headache, delirium, unconsciousness, and coma. These may occur at an early or late period of the disease, and usually indicate a fatal termination.

As the disease progresses symptoms of sensory and motor paralysis supervene, although those indicative of irritation may for a time be variously combined with them. Cutaneous sensibility becomes diminished and complete anæsthesia may be established, while the extremities manifest various degrees of motor weakness up to complete paralysis. When the lesion is situated high up, paralysis of the respiratory mechanism may lead to a fatal termination, and the Cheyne-Stokes respiration has been repeatedly observed towards the fatal termination. The pupils may be contracted, dilated, and unequal. In fatal cases deep coma supervenes, accompanied by a rapid elevation of temperature.

At other times deceitful signs of temporary improvement appear, but paralysis and bed-sores supervene, and death follows after long suffering. Sometimes, however, there is real improvement, and slight cases may speedily recover, but in most instances

convalescence is slow and the symptoms of sensory and motor irritation only disappear after a long period.

Incurable defects are often left behind, such as anæsthesia of variable degree and extent, and paralysis of individual muscles, groups of muscles, or of entire extremities. The muscular paralysis may be associated with atrophy and contractures. The symptoms which indicate sclerosis of single columns of the cord, such as ataxia and spastic paralysis, may sometimes become permanently established.

If the inflammation extend to the medulla oblongata or to the base of the cranium, the characteristic symptoms, vomiting, headache, delirium, and paralysis of the ocular muscles, supervene.

§ 636. *Course, Duration, and Termination.*—In cerebro-spinal meningitis death occurs early, occasionally within a few hours, but more usually it is postponed for a few days. In less violent cases the duration may be two or three weeks, and the severity of the symptoms fluctuates greatly.

In other cases the acute symptoms subside and the disease assumes a chronic form which is usually associated with myelitis. Cystitis and bed-sores supervene, and the patient dies from exhaustion.

In mild cases the threatening symptoms may disappear rapidly and the patient speedily recover. But convalescence is as a rule protracted even when the patient ultimately makes a complete recovery, but in a large number of cases a certain amount of paralysis and anæsthesia remains.

§ 637. *Morbid Anatomy.*—The morbid changes found in acute spinal meningitis may be subdivided into three groups, according to the period of the disease:—(1) A stage of hyperæmia and commencing exudation; (2) a stage of serous, purulent, or fibrinous exudation; (3) a stage in which chronic changes are established.

(1) In the first stage the pia mater is congested, of a rosy or a dark red tint, and dotted with hæmorrhagic extravasations. The tissue is swollen, infiltrated with serum, and the cerebro-spinal fluid is slightly turbid. The arachnoid is also congested, and the hyperæmia extends to the cord and to the dura mater.

(2) In the second stage the spinal fluid becomes more and more turbid, and fibrinous flakes and plates are found in the subarachnoid tissue or adhering to the surface of the dura mater. The pia mater becomes more and more opaque, and the subarachnoid tissues are transformed into a gelatinous mass. The exudation becomes more and more purulent, and at last the whole pia mater and subarachnoid tissues are infiltrated with pus. The spinal fluid assumes a sero-purulent appearance and contains numerous flakes of fibrine.

Small miliary nodules may be found in some cases distributed along the course of the vessels of the pia mater, constituting tubercular spinal meningitis.

The distribution of the exudation varies greatly. It usually covers the greater part or the whole of the cord, but the posterior surface is affected in a greater extent than the anterior, and the changes are sometimes limited to a small portion of the cord. The exudation not unfrequently extends from the spinal canal into the brain, and the arachnoid is always involved in the inflammatory action. It is thickened, opaque, infiltrated with serum or pus, and often abounds with grey miliary tubercles, while the subarachnoid tissue is similarly affected.

The dura mater is often hyperæmic, opaque, and covered with thin fibro-purulent exudation. The nerve roots are almost always involved, they are enveloped in thick masses of exudation, and are often swollen and softened. The cord itself is pale and œdematous, or congested, and is usually softened either at limited spots or diffusely.

(3) In the third stage chronic changes supervene and become permanent, the most common of these being opacity and thickening of the spinal membranes, adhesions, accumulations of fluid in the arachnoidal space (hydrorrhachis), and sclerosis and atrophy, either diffused through the cord or affecting isolated portions or systems. In cases where absorption has taken place, there is, of course, no third stage.

§ 638. *Morbid Physiology.*—The pains in the back, eccentric pains in the extremities, hyperæsthesia, and muscular rigidity and twitchings are caused by irritation of the posterior and anterior roots of the nerves. The sensory and motor paralysis

which characterises the second stage of the disease is caused by the compression of the nervous tissues occasioned by the exudation. Implication of the anterior roots and anterior grey horns explains the muscular atrophy which is sometimes observed, while affection of the posterior grey horns in the lumbar region accounts for the paralysis of the bladder and rectum, cystitis, and bed-sores which are sometimes present, while extension of the morbid process to the upper part of the cervical region causes the disturbances of respiration which occur.

§ 639. *Diagnosis.*—The general evidences of the disease are fever, pain and rigidity of the back, stiffness of the neck, muscular spasms, cutaneous hyperæsthesia and paræsthesia, pains in and paralysis of the limbs, retention of urine, constipation, and dyspnœa.

When the membranes of the brain are implicated, the cerebral symptoms will of course constitute the most prominent feature of the disease.

It is not always possible to distinguish acute spinal meningitis from acute myelitis, inasmuch as the two affections are often combined. Stiffness in the back and neck, eccentric pains in the limbs, and hyperæsthesiæ are characteristic of acute spinal meningitis; while sensory and motor paralysis predominate in myelitis.

In tetanus cerebral symptoms are always absent, there is no hyperæsthesia of the skin, and reflex excitability is very greatly exaggerated. The presence of the risus sardonicus and the early occurrence and severity of the tetanic spasm in tetanus render the diagnosis between it and spinal meningitis as a rule easy.

The diagnosis of the tubercular form of the disease must rest chiefly on general considerations, such as the evidence of scrofula or tuberculosis of other organs. It is probably always associated with tubercular basilar meningitis; so that the presence of the cerebral symptoms characteristic of that affection may be of use in clearing up the diagnosis of the spinal disease.

§ 640. *Prognosis.*—The prognosis varies greatly in different cases. Hyperacute and tubercular meningitis, and that caused

by deep bed-sores, are always fatal. The prognosis is more favourable in rheumatic and traumatic cases. The prognosis is unfavourably influenced by the following circumstances: very young or advanced age, implication of the cervical portion of the cord, early symptoms of paralysis, signs of exhaustion, high fever, progressive rise of temperature and frequency of pulse, great difficulty of breathing, and severe cerebral symptoms.

§ 641. *Treatment.*—In the early stage of the affection energetic antiphlogistic treatment must be adopted. The main remedies of this class are blood-letting and the application of cold. Local blood-letting will generally suffice, and cold must be applied sedulously by means of Chapman's ice-bags. A smart purgative, such as the compound jalap powder or the aperient saline waters, may also be administered.

When the temperature has fallen slightly blisters may be applied along the spine, and in the milder cases weaker counter-irritants may be adopted. Mercury is very useful in the treatment, the inunction being the best method of application. From fifteen grains to a drachm of the ointment should be rubbed daily into the back and extremities. The patient should be placed in a quiet, airy, moderately-warmed room, and kept lying in the recumbent posture on his side and face, and should be prevented from using any exertion. Nourishment should be fluid, easily digestible at first, but care must be taken to allow a more generous diet at an early period of the disease.

Various symptoms require to be treated during the course of the affection. Sedatives are required in order to relieve pain, sleeplessness, and hyperæsthesia, and for this purpose large doses of opium and subcutaneous injections of morphia are best. Warm baths afford great relief, and moist packing of the whole body often soothes and induces sleep.

When the affection has assumed the chronic form, large doses of iodide of potassium should be administered. The sequelæ, such as paralysis, atrophy, anæsthesia, and weakness of the bladder, must be treated with baths and electricity.

(ii.) Leptomeningitis Spinalis Chronica.

§ 642. *Etiology.*—The disease frequently develops from the

acute form and then has the same causes. The acute form is more apt to degenerate into the chronic variety in debilitated and badly-nourished subjects, or in those who are addicted to alcoholic and other excesses.

§ 643. *Symptoms.*—The symptoms are, on the whole, the same as those of the acute variety, but they develop more gradually, and are slower in their progress. When the disease develops from the acute form the violent symptoms disappear, but the pain, stiffness, abnormal sensations, and paralysis remain for a longer time, and the disease passes on to become chronic.

In the majority of cases the disease begins in an insidious manner, the commencement being marked by abnormal sensations in the lower extremities, gradually increasing pain and stiffness of the back and neck. The pain in the back is described as a feeling of drawing and pressure in the loins, and is aggravated by movements of the vertebral column, though not increased by pressure on the spinous processes.

A girdle sensation, as well as shooting and boring pains, is felt in the trunk, on a level with the morbid lesion. The patient complains of tingling, formication, and a feeling of coldness in the limbs, which feel weak and heavy. These abnormal sensations may be accompanied by tearing or shooting pains, either limited to the region of some nerve trunk, or changing from one place to another.

The eccentric symptoms are limited to the region of distribution of the nerves whose roots originate in the part chiefly affected, and they are more marked as a rule in the lower extremities, although the upper are not unfrequently affected.

Symptoms of motor irritation are of subordinate importance in chronic meningitis, although some are almost always present. A certain degree of stiffness of the back and neck and involuntary drawing up or extension of the limbs are rarely absent.

The heaviness and weakness of the limbs gradually increase, but the paraplegia in spinal meningitis is seldom complete and fluctuates in its intensity from day to day. If there be a considerable effusion of spinal fluid the paralysis increases when the patient stands; but at other times the paralysis increases

when the patient is lying on his back, and then it is probable that the cord is liable to become passively congested.

Hyperæsthesia is a frequent symptom, but anæsthesia is rare. There is usually only slight blunting of the cutaneous sensibility, limited to the feet and lower part of the legs.

In severe cases the muscles may undergo atrophy with loss of electric excitability. Anæsthesia is developed, the reflex actions are abolished, bed-sores and cystitis appear, and the patient dies from pyæmia and marasmus.

§ 644. *Course, Duration, and Results.*—The disease is always slow, and extends over a period of months or years. Some cases recover, but the return to health is slow, and often interrupted by relapses. The sensory disturbances are the first to disappear, the motor persisting longer. In many cases recovery is only partial, and paralysis of some muscles or extremities, with or without atrophy, circumscribed anæsthesia, weakness of the bladder, and other symptoms remain permanently. In the great majority of cases chronic meningitis ends after a time fatally. The symptoms which precede and cause death are usually paraplegia, paralysis of the bladder, cystitis, bed-sores, and marasmus. In other cases the morbid process extends to the cervical region, giving rise to difficulty of breathing. At other times death is brought about by an acute attack of purulent meningitis supervening on the chronic form. Death may also be caused by many other complications and accidents.

§ 645. *Morbid Anatomy.*—The pia mater and arachnoid in chronic spinal leptomeningitis are congested, thickened, opaque, often pigmented, and closely adherent to the dura mater on the one side and the cord on the other. The spinal fluid is usually in excess; it is generally turbid, tinged with blood, or mixed with an abundant fibrinous exudation. Numerous thin and small calcareous plates may be observed on the arachnoid, especially in the lumbar region. The cord is usually implicated in the morbid changes. Transverse myelitis, or cortical, systematic, or disseminated sclerosis may be observed.

§ 646. *Diagnosis.*—The diagnosis of chronic spinal lepto-

meningitis presents no difficulty when the disease is fully developed and uncomplicated, but is difficult during the first obscure symptoms of the affection, and when it is complicated with chronic myelitis.

Pain and stiffness in the back, eccentric pains in the extremities, girdle pains, and other symptoms caused by irritation of the roots of the nerves, a slight degree of paralysis with fluctuations in its intensity, especially when the last varies according as the patient is in the prone or erect posture, hyperæsthesia or a slight degree of anæsthesia, normal or absent tendon reflexes, and painful muscular jerkings point strongly to spinal meningitis.

When severe paralysis and anæsthesia are present, the pains slight, and the tendinous reflexes exaggerated, the presence of myelitis may be inferred.

Spinal meningitis may be readily distinguished from locomotor ataxy, but it must be remembered that the two diseases are often combined.

§ 647. *Prognosis.*—The prognosis is always grave, although many apparently hopeless cases have been known to recover; as a rule, some permanent damage to the cord is generally left behind.

§ 648. *Treatment.*—The acute form should always be subjected to energetic treatment, with the view of avoiding the establishment of the chronic variety. Active antiphlogistic treatment does no good in the chronic form of the disease.

Counter-irritation along the vertebral column is one of our best means of treatment. Repeated large blisters to the back are the most effectual of this class, although the milder counter-irritants may be sufficient in some cases.

Iodide of potassium in moderate doses is the only reliable internal medicine. Mercury should not be administered unless the presence of syphilis be suspected. Ergot and belladonna are of no use. The patient should be kept warm, and warm baths of all sorts are efficacious.

(IV.) TUMOURS OF THE SPINAL MEMBRANE.

§ 649. The tumours which are found within the spinal canal usually develop from the spinal membranes. The majority of them spring from the dura mater, but some originate from the arachnoid and pia mater, and remain limited to these membranes. Morbid growths may also arise from the neighbouring structures and extend towards the canal so as to involve the membranes secondarily.

§ 650. *Etiology.*—The causes of meningeal tumours are obscure. Many cases have been observed where the first symptoms occur after a fall or blow on the back or spine; in many cases the commencement of the disease dates from childhood. Disease of the vertebræ, the tubercular and scrofulous diathesis, syphilis, and probably an inherited tendency to the formation of carcinomatous and other growths are the main predisposing causes.

§ 651. *Symptoms.*—The symptoms may be divided into two groups—(1) those caused by local irritation and compression of the nerve-roots and membranes first involved in the tumour; and (2) those caused by irritation and compression of the cord itself, and by consecutive myelitis.

The symptoms of the first group may precede those of the second by many years. They are very variable, as might be expected, when it is considered that they must largely depend upon the locality of the tumour, and the direction and rate of its growth.

Violent Pains of a lancinating, tearing, and boring character are complained of, and these may remain confined to a single point, or attack a nerve trunk. They either surround the trunk like a girdle at various levels, or invade the upper or lower extremities of one or both sides. They may also extend gradually or suddenly into neighbouring nerve districts. They are often increased by movement of the spinal column, and, like all neuralgic pains, are made worse by sudden changes of weather.

Paræsthesiæ, such as tingling, formication, numbness, either

in the form of a girdle or limited to certain regions of the extremities, are observed.

Twitchings and Spasms of individual muscles may appear when the motor roots are first subjected to the pressure of the tumour.

These symptoms are almost always accompanied by local pain, and stiffness of the spine in the neighbourhood of the tumour.

Symptoms of both sensory and motor paralysis appear sooner or later, but these are at first limited to the region of distribution of the nerves which take origin from the part of the cord affected. These symptoms consist of circumscribed anæsthesia, often associated with pain (*anæsthesia dolorosa*), and local paresis or paralysis of the corresponding muscular groups, followed by atrophy and the reaction of degeneration.

If the cervical portion be the seat of the affection, an upper extremity may first be seized by pains, paræsthesiæ, partial paralysis, and atrophy before the symptoms of compression of the cord appear. When the dorsal region is the seat of the tumour, the illness is introduced by intercostal neuralgias and herpes zoster. Neuralgias and trophic changes in the district of the lumbar or sacral plexuses indicate that the lumbar region is involved.

After a period of weeks, months, or years, according to the rate of growth of the tumour, the symptoms due to compression and myelitis appear either gradually or suddenly. When paraplegia occurs suddenly, or in a period of a few hours, it is generally caused by secondary myelitis. If the paraplegia result from the compression caused by the slow growth of the tumour, one lateral half of the cord may be subjected to pressure, or the compression may occur on the anterior or posterior surface of the cord. Motor phenomena predominate when the anterior surface is chiefly affected, and sensory phenomena when the posterior is principally involved. When the cord is completely compressed, or transverse myelitis occurs, the whole of the body below the seat of the tumour becomes more or less completely anæsthetic and paralysed, and the bladder and rectum are paralysed, while cystitis and bed-sores with their consequences supervene.

Symptoms of motor irritation often accompany those of paralysis. These usually consist of muscular twitching and transient spasms, and after a time secondary degeneration occurs, causing contracture of the extremities.

The reflex actions, both superficial and deep, are usually increased, but when the grey substance becomes secondarily diseased, or when the tumour is situated in the lumbar region, the reflexes are abolished.

Muscular atrophy may at first be limited to the area of distribution of the nerves, the anterior roots of which are compressed by the tumour; but after a time all the paralysed muscles undergo rapid atrophy, and their faradic contractility disappears. Death is generally caused by cystitis and bed-sores, but when the tumour is situated high up death may be caused by arrest of respiration. Death not unfrequently results from an intercurrent attack of pneumonia, or spinal meningitis.

§ 652. *Course, Duration, and Termination.*—The first stage of the disease is very insidious and may extend over many years. When once paraplegia makes its appearance the progress is usually more rapid; but even then years may pass before death occurs. The symptoms fluctuate greatly in severity, and the entire duration of the affection varies from eight months to four or five years; although cases are known which have extended over a period of fifteen years. The disease generally ends fatally, but in the case of syphilomata, scrofulous tumours, and inflammatory new formations, complete or partial recovery may take place.

§ 653. *Diagnosis.*—During the first stage of the disease it may be inferred that there is a circumscribed lesion of the cord, but it is not possible to make an accurate diagnosis of tumour. When symptoms of a slowly-developing compression of the cord are present, and when these have been preceded by symptoms of irritation or compression of the nerve roots, a tumour may be suspected.

The diagnosis of the *nature* of the tumour must be made by a careful examination of all the circumstances of the case.

When Pott's disease or marked scrofula exists, a peri-menin-

gitic exudation may be inferred; and when there is primary cancer of the vertebræ or of some other part, a carcinomatous tumour may be considered probable; while if there be other evidences of syphilis a gummatous tumour is to be expected. Echinococcus may be inferred to be present when the parasite has been found in other organs, and neuroma if neuromata are found on the peripheral nerves.

§ 654. *Morbid Anatomy.*

Fibroma and fibro-sarcoma are usually small, oval tumours, 3 to 5 cm. long and 2 to 4 cm. thick. They spring from the dura or pia mater, and may be situated either within or without the sac of the dura. They consist of connective tissue, with more or less abundant spindle or round cells.

Sarcoma occurs in every possible form, as hard and soft, fibrous or cellular, and often as a cystosarcoma. It generally originates from the inner membranes, and is usually of an elongated form, and frequently lobular.

Myxoma almost exclusively originates from the arachnoid or pia mater. It is a soft, juicy, lobulated tumour of moderate size, and pale colour.

Psammoma appears in the form of a small roundish or oval-shaped smooth or lobed tumour, and generally originates in the soft membranes. It is really a sarcomatous tumour with granular concretions of lime imbedded in its substance.

Lipoma has repeatedly been found in the vertebral canal, and may originate either in the fatty tissue outside the dura mater or from the soft membranes.

Enchondroma, of the size of a hazel-nut, and firmly adherent to the dura mater and connected vertebræ, has been found by Virchow.

Osteoma, in the form of so-called cartilaginous discs, is very common in the arachnoid, and diffuse ossification of the dura mater also occurs, but neither of these can be regarded as proper tumours.

Multiple Fibrous Melanoma has been found in the spinal canal by Virchow and Sander.

Neuromata have been found on the nerve roots, especially of

the cauda equina. They are generally false neuromata, and are either multiple, or occur singly.

Carcinoma springs very rarely from the spinal membranes. These tumours are almost always developed secondarily by extension from the vertebræ or neighbouring parts, or by metastasis from other organs.

Miliary tubercles are found in the soft membranes, and closely allied to these are tumours which originate from inflammatory, hæmorrhagic, and other processes in the spinal membranes or the neighbouring parts, such as peri-pachymeningitic exudations, circumscribed masses of a purulent or caseous nature, scrofulous exudations between the dura and vertebral column, and hæmatoma.

Syphilomata usually consist of gummata of the dura or pia mater.

Parasitic growths are rarely met with in the vertebral canal. Cysticercus cellulosæ has been found by Westphal, and echinococcus has been occasionally observed.

§ 655. *Prognosis.*—The prognosis is always very serious, and the more quickly the symptoms have developed the worse it is. The presence of carcinoma warrants the worst prognosis. In the inflammatory, syphilitic, scrofulous, and hæmorrhagic forms the prognosis is more favourable. If paraplegia be complete the prognosis is hopeless.

§ 656. *Treatment.*—The internal remedies from which most is to be expected are iodide of potassium and mercury.

The painful and other distressing phenomena must be relieved by suitable remedies.

(V.) DEFORMITIES OF THE SPINAL MEMBRANES.

§ 657. *Spina Bifida* consists of an abnormal accumulation of fluid within the cavity of the dura mater in connection with a greater or lesser deformity of the vertebral column. It presents itself as a sac-like dilatation and pouching of a more or less circumscribed portion of the cavity of the dura mater, which is generally associated with deficiency or absence of one or more vertebral arches. The sac protrudes like a hernia through the

cleft, and raises the skin in the form of a tumour of variable size. The seat of the tumour is generally in the sacral and lumbar regions, and more rarely in the dorsal or cervical portions of the cord. It is almost always situated in the middle line and seldom deviates to either side. As a rule there is only one tumour, but several are occasionally present. They vary in size from a hazel-nut to that of a child's head, and are usually round or elliptical in form, but at times may extend over a large portion of the spinal column. The tumour is either sessile or pedunculated, and sometimes subdivided into two or lobulated.

The skin over the tumour may be normal, or stretched thin, red, and ulcerated, and at times an umbilicated depression may be seen at some point on the surface of the tumour, caused by the insertion of the end of the cord in the interior of the sac.

The dura mater is either thickened or thin and stretched, and usually lies immediately beneath the skin. The arachnoid usually encloses the fluid, and if hydromyelia exist, the pia mater takes part in the formation of the sac. The neck of the sac is more or less narrow, and communicates with the spinal canal. Occasionally it may be closed by adhesions, so that the external sac forms a cystic tumour.

The condition of the spinal cord varies in different cases. As a rule the cord is normal and takes no part in the deformity except that its lower extremity may be adherent to the sac. The cord may be lengthened, and rendered thin and flat by its extremity being drawn out of the vertebral canal. In these cases the nerve roots return along the walls of the sac or through the fluid. When a hydrorrachis interna exists along with the spina bifida, the lower portion of the cord is more or less destroyed, atrophied, and the cavity of the sac communicates directly with the dilated and open central canal of the cord.

The contents of the sac consist generally of a light colourless and clear fluid, identical with the normal cerebro-spinal fluid, and its quantity may amount to from 3oz. to 2lb. or more.

§ 658. *Symptoms.*—The most marked symptom at birth consists of a tumour over the vertebral column, usually seated in the sacral or lumbar region, and occasionally at a higher

point of the spinal column. When the tumour is large the sac may burst during birth, and the child then generally dies from asphyxia a few hours or at most a day or two after birth. The tumour may remain stationary or increase in size. In rare cases it may develop for the first time after birth.

The tumour is tense, elastic, fluctuating, and when the skin is thin and stretched it may appear translucent. At times the skin may be ulcerated, so that the wall of the sac is constituted by the distended spinal membranes. The sac can be emptied by slow and gradual pressure except in those cases in which communication of the spinal canal has been cut off by closure of the sac; and if there be coincident hydrocephalus, pressure on the tumour causes swelling and protrusion of the fontanelles. The swelling also increases on assuming the erect posture, and during coughing and sneezing. The tumour is sometimes sensitive on pressure. It may remain stationary and give rise to no other symptoms, and the patient may arrive at maturity without developing any serious symptoms. As a rule, however, it increases in size and produces pressure on the lower part of the cord and the cauda equina, so that the children affected soon suffer from paraplegia, incontinence of urine and fæces, and bedsores, and the case soon terminates fatally. When there is coexistent hydrorrachis interna, paraplegia and paralysis of the sphincters are present from the beginning, and death speedily results.

Rupture of the sac may be caused in various ways, and is followed by purulent inflammation which usually extends from the sac into the spinal canal, and a purulent spinal meningitis results, which terminates fatally in a few days. When the perforation is very minute and the fluid flows out very slowly, the case has been known to terminate favourably and to lead to the cure of the disease. When the opening is large and the fluid is rapidly evacuated, death may follow very quickly preceded by general convulsions.

§ 659. *Diagnosis.*—The diagnosis is as a rule exceedingly easy, and presents no difficulty except when the tumour is small and when the orifice of communication is small or entirely closed. The characteristic signs of the disease may be gathered

from the symptoms already described. The sacral tumour, resulting from the displacement of the abdominal and pelvic viscera, can as a rule be readily distinguished from the tumour of spina bifida, and the malformations and defects which not rarely coexist with spina bifida, such as hydrocephalus, deformities of the lower extremities, anomalies of the genital apparatus, inversion of the bladder, with congenital fissure of the abdominal walls, can be readily recognised.

§ 660. *Prognosis.*—The prognosis is as a rule very unfavourable. The majority of the children die either from accidental opening of the sac, progressive growth of the tumour, or in consequence of operations undertaken to effect a radical cure. The larger the tumour and the higher its situation the more unfavourable is the prognosis. The prognosis is also bad when the orifice of communication is large, when hydromyelia and hydrocephalus are associated with it, and when the constitution of the child is feeble.

§ 661. *Morbid Physiology.*—The origin of spina bifida is still doubtful. Some regard it as being due to dropsy of the subarachnoidal space, or as a primary dropsy of the central canal, with disappearance of the cord and secondary widening and distention of the spinal membranes. If these changes take place before the vertebral arches are closed, a cleft of the vertebral column may be induced. Others think that the cleft of the vertebral column is the primary part of the process and the hydrorrachis is developed at a subsequent period.

§ 662. *Treatment.*—The surgical operations which have been attended with the best results are methodical compression of the tumour, simple and repeated puncture of the sac, and puncture with subsequent injection of iodine.

Simple puncture may be performed repeatedly, and it is best effected by means of the hypodermic syringe. The sac should not be completely emptied, and pressure should be afterwards lightly applied. For the details of the treatment of spina bifida surgical works must be consulted.

PART IV.—DISEASES OF THE ENCEPHALON.

CHAPTER I.

ANATOMICAL AND PHYSIOLOGICAL INTRODUCTION.

THE encephalon is invested with three membranes continuous with those of the spinal cord. They are named (1), dura mater; (2), arachnoid; and (3), pia mater.

§ 663. *Dura Mater.*—The cranial part of the dura mater is adherent to the inner table of the skull, especially along the lines of the sutures and the margins of the foramina. Strong flattened prolongations from the inner surface of the dura mater

FIG. 189.

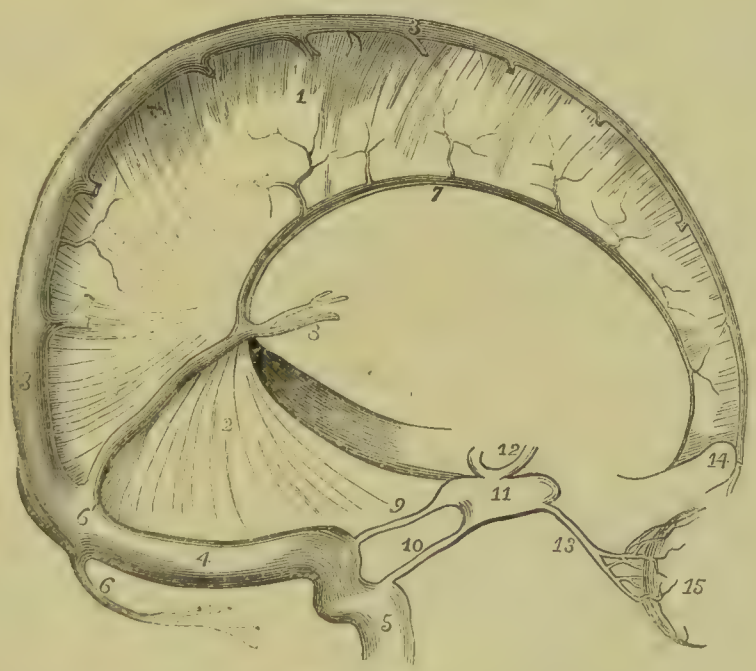


FIG. 189 (From Turner). *Dura Mater and Cranial Sinuses.*—1, Falx cerebri; 2, Tentorium; 3, 3, Superior longitudinal sinus; 4, Lateral sinus; 5, Internal jugular vein; 6, Occipital sinus; 7, 8, Veins of Galen; 9 and 10, Superior and inferior petrosal sinus; 11, Cavernous sinus; 12, Circular sinus, which connects the two circular sinuses together; 13, Ophthalmic vein, from 15, the eyeball; 14, Crista galli of ethmoid bone.

form partitions between divisions of the encephalon. These partitions are respectively named the *falx cerebri, tentorium cerebelli,* and *falx cerebelli.*

The cranial dura mater is mainly composed of two layers of fibrous tissue, which separate from each other along certain lines, so as to form tubular passages, named *sinuses;* these transmit the venous blood returning from the brain.

The sinuses usually pass from before backwards, and several join opposite the internal occipital protuberance at a spot which is called the *torcular Herophili.* The blood is drained from the *torcular* by the *lateral sinuses,* which terminate in the internal jugular veins. The minute structure of the membranes of the brain is the same as already described in the case of the spinal membranes.

§ 664. *The Arachnoid.*—The arachnoid is a delicate and transparent membrane, and between it and the dura mater is a space, containing a small quantity of limpid serum, which lubricates the smooth opposed surfaces of the two membranes. This space is regarded as equivalent to the cavity of a serous membrane, and is named the *arachnoid cavity* or *sub-dural space.* The arachnoid and pia are separated by a distinct space called the *sub-arachnoid space.* The space contains a limpid cerebro spinal fluid, varying in quantity from two drachms to two ounces.

Pacchionian Bodies. — Clusters of granular bodies are observed on each side of the longitudinal sinus imbedded in the dura mater, named Pacchionian bodies. These bodies spring from the arachnoid membrane, and sometimes attain a relatively large size.

§ 665. *Pia Mater.*—The pia mater closely invests the whole outer surface of the brain, and dips in the fissures between the convolutions, differing in this respect from the arachnoid, which passes from the summit of one convolution to that of another. A wide prolongation of this membrane passes into the interior of the cerebrum, named the *velum interpositum.* The pia mater is prolonged along the roots of the cranial and spinal nerves and filum terminale. It is the vascular membrane of

the brain, and the arteries which pass from it into the substance of the latter are invested by it with a loose funnel-shaped sheath, which opens into the sub-arachnoid space, and contains cerebro-spinal fluid (Key and Retzius). The ventricles of the brain are also supposed to be in free communication with the sub-arachnoid space.

THE BRAIN OR ENCEPHALON.

The part of the central nervous axis, which is contained within the cavity of the skull, is termed the *brain or encephalon.* The brain is conveniently divided into (1), the medulla oblongata; (2), the pons variolii; (3), the cerebellum; and (4), the cerebrum.

§ 666. *The Medulla Oblongata.*—The medulla oblongata is the expanded upper end of the spinal cord, and has already been described.

§ 667. *The Pons Variolii.*—The pons rests on the dorsum sellæ of the sphenoid bone, and is marked on its inferior aspect by a median longitudinal groove, in which the basilar artery lies; its posterior surface receives the pyramidal tracts and the upward continuation of the anterior root-zones, and the grey matter of the cord; its anterior surface gives origin to the two crura cerebri; each lateral surface is in relation to a hemisphere of the cerebellum; the superior surface forms in part the upper portion of the floor of the fourth ventricle, while the corpora quadrigemina rest upon its anterior half.

Structure of the Pons.—The pons consists of grey and white matter. The greater portion of the grey matter of the pons is an upward continuation of the grey matter of the spinal cord and medulla oblongata, which has been already described. In addition to the grey matter on the floor of the fourth ventricle, there is a considerable quantity interposed between the transverse fibres of the pons. The transverse fibres derived from each lateral lobe of the cerebellum appear to terminate in the interposed grey matter of the opposite half of the pons.

The white matter of the pons consists of longitudinal and transverse fibres. The longitudinal fibres are the upward continuations of the anterior pyramids of the medulla, the anterior root-zones of the cord, and probably also fibres ascending from the olivary body. The longitudinal fasciculi are also reinforced by fibres arising in the pons itself.

The transverse fibres go from one hemisphere of the cerebellum to that of the opposite side, although the fibres are probably interrupted in the pons by interposed grey matter. These fibres, therefore, constitute the commissural or connecting arrangement, by means of which the two hemispheres of the cerebellum become anatomically continuous with one another.

THE CEREBELLUM.

§ 668.—The cerebellum occupies the inferior occipital fossæ. It consists of two lateral hemispheres joined together by a median portion called the *vermiform process.* On the superior surface of the cerebellum this is a mere elevation, but on its inferior surface it forms a well-marked projection, named the *inferior vermiform process.* This process lies at the bottom of a deep fossa (*vallecula*), which is prolonged to the posterior border of the cerebellum, and forms there a deep notch, in which the falx cerebelli is lodged.

The Peduncles.—The cerebellum is connected below with the medulla oblongata by the two restiform bodies which form its *inferior* peduncles. The crossed connection of the fibres of the inferior peduncles of the cerebellum with the olivary bodies has already been described. The cerebellum is connected with the corpora quadrigemina and crura cerebri by two *superior* peduncles. The greater portion of the fibres of the superior peduncles decussate in the upper end of the pons and in the tegmenta, the fibres of one side becoming connected with the red nucleus of the tegmentum of the opposite side. The transverse fibres of the pons form the *middle* peduncles of the cerebellum.

Folia.—The surface of the cerebellum consists of numerous *laminæ* or *folia,* which are separated by fissures or sulci of different depths.

Fissures.—The great horizontal fissure begins behind the middle peduncles, passes horizontally backwards round the circumference of the cerebellum, dividing its tentorial and occipital surfaces. From this primary fissure numerous others proceed, and some that are constant in their position and deeper than the rest have been described as separating the cerebellum into lobes.

Lobes.—The tentorial surface is subdivided into two smaller

lobes, named *anterior superior* and *posterior superior*. On the occipital surface each hemisphere is subdivided from behind forwards into the *posterior inferior lobe*, the *slender lobe*, the *biventral lobe*, and the *flocculus*. Immediately internal to the biventral lobe is the *amygdala or tonsil*, which forms the lateral boundary of the anterior part of the vallecula. The inferior vermiform process is subdivided into a posterior part or *pyramid*, an elevation or *uvula* situated between the two tonsils, and an anterior pointed process or *nodule*. Stretching between the two flocculi, and attached midway to the sides of the nodule, is a thin, white, semi-lunar-shaped plate of nervous matter, called the *posterior medullary velum*, whilst the layer of grey matter stretching between the uvula and tonsil on each side is called the *furrowed band*.

§ 669. *Internal Structure.*—The cerebellum consists of both grey and white matter. The grey matter forms the exterior or cortex, while the white forms the interior of the organ. A vertical section through the cerebellum presents an arborescent appearance known by the name of *arbor vitæ*. Independent masses of grey matter are found in the interior of the cerebellum. If the hemisphere be cut through to the outer side of the median lobe, a nucleus of grey matter is observed similar in its arrangement to the olivary body, and named the *corpus dentatum*. It encloses

Fig. 190.

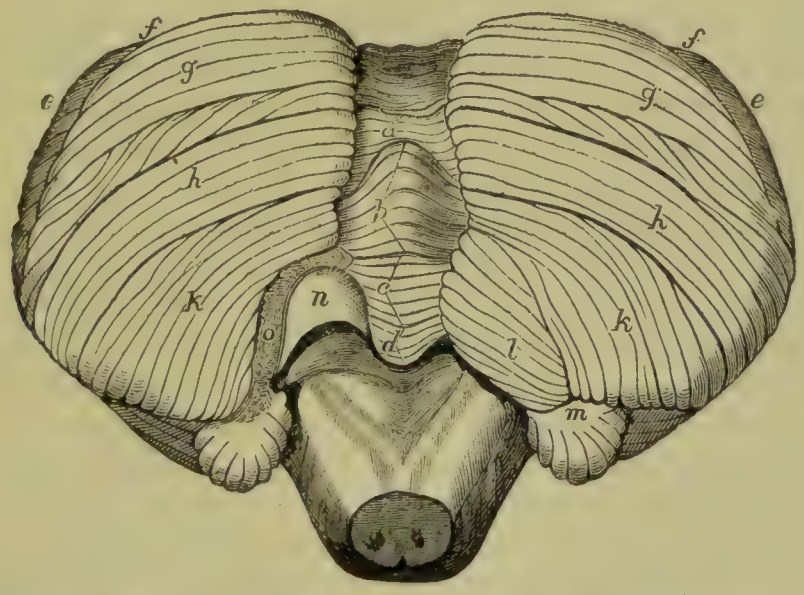


Fig. 190 (From Turner). *The Occipital Surface of the Cerebellum.*—*a*, Vallecula; *b*, Pyramid; *c*, Uvula; *d*, Nodule; *e*, *e*, Margin of tentorial surface; *f*, *f*, Great horizontal fissure; *g*, *g*, Posterior inferior lobes; *h*, *h*, Slender lobes; *k*, *k*, Biventral lobes; *l*, Tonsil; *m*, Flocculus; *n*, Posterior medullary velum; *o*, Cut surface from which the left tonsil has been detached.

white fibres which leave the interior of the corpus at its inner and lower sides. Stilling has described two grey masses situated in the anterior end of the inferior vermiform process which he named *roof nuclei.*

The *white matter* is for the most part continuous with the fibres of the peduncles of the cerebellum. The fibres of the inferior peduncles pass upwards to join the grey matter of the superior surface of the cerebellum, especially in the median lobe. They are also connected with the corpus dentatum and roof nuclei. Those of the superior peduncles descend from the corpora quadrigemina and reach the grey cortical matter, more especially that of the inferior surface of the cerebellum, and they are also connected with the corpus dentatum. The fibres of the middle peduncles terminate chiefly in the cortex of the lateral lobes. The cerebellum also contains fibres which connect different parts of its grey matter with one another, named *fibræ propriæ.* Stilling describes a median *fasciculus,* the fibres of which connect the superior and inferior vermiform processes. Other fibres cross the median plane to unite symmetrical regions of the lateral lobes. Meynert describes a cerebellar origin of the auditory and fifth nerves.

Minute Structure.—The grey matter of the cortex is divided into an *external grey* or *cellular* layer, and an *internal rust-coloured layer* of about equal thickness. The *external layer* consists of a delicate matrix containing cells and fibres. Most of the fibres have a direction at right angles to the surface, the majority of them being the processes of Purkinjé's cells, to be immediately described. Of the cells some are small, and appear to belong to the connective-tissue matrix, while others are larger, and probably connected with the processes of Purkinjé's cells. The inner part of the external layer contains fibres which run parallel with the surface.

The inner or granule layer consists of granule-like corpuscles, which lie in dense groups in a gelatinous matrix, containing a plexus of fine nerve fibres. Some are round, while others are angular and possess a protoplasmic envelope with processes, which are supposed to be connected with the plexus of fine nerve fibres amongst which they lie.

The cells of Purkinjé lie in a single layer, between the outer and inner layers of the cortex. Most of them are flask-shaped bodies, containing a spherical nucleus and nucleolus. The long axis of the cell is generally directed at right angles to the surface. From the external surface of the cells two processes are given off; these pass out towards the surface and divide repeatedly in their course. The finer subdivisions of these processes have been said to curve back towards the granule layer, where, according to Boll, they form a network of extreme minuteness, from which it is believed that nerve fibres arise. From the inner end of the cell another fibre is given off; it is unbranched, passes into the granule layer, and is supposed to be continuous with the axis cylinder of a nerve fibre.

The medullary centre consists of nerve fibres arranged in parallel or interlacing bundles. They form the central stem of the folia, whence

they radiate into the cortex. They disappear in the granule layer, and are commonly believed to be continuous with the inner processes of Purkinjé's cells.

THE CEREBRUM.

§ 670. The *cerebrum* constitutes the largest division of the encephalon, and lies above the level of the tentorium. It consists of a narrow constricted portion—the crura—of certain basal ganglia—the corpora quadrigemina, optici thalami, and corpora striata—and of an upper expanded portion—the cerebral hemispheres.

§ 671. *Exterior of the Cerebrum.*—The cerebrum is ovoid in shape and presents superiorly, anteriorly, and posteriorly a deep *median longitudinal fissure*, which subdivides it into two hemispheres. The two hemispheres are connected across the median plane by the *corpus callosum*. The outer surface of each hemisphere is convex, and adapted in shape to the concavity of the inner table of the cranial bones. Its inner surface is flat, and is separated from the opposite hemisphere by the *falx cerebri*. The under surface, where it rests on the tentorium, is concave, and is separated by that membrane from the cerebellum and pons. From the front of the pons two strong white bands, the *crura cerebri* or *cerebral peduncles*, pass forwards and upwards to enter the basal ganglia of their respective hemispheres. The *optic tracts* wind round each crus, and converge in front to form the optic commissure from which the two optic nerves arise. The crura cerebri, optic tracts, and optic commissure enclose a lozenge-shaped space which includes from behind forwards the *posterior perforated space*, the *corpora albicantia*, and the *tuber cinereum*, from which the *infundibulum* projects to join the *pituitary body*. Immediately in front of the optic commissure is a grey layer, the *lamina cinerea* or lamina terminalis of the third ventricle; and between the optic commissure and the inner end of each Sylvian fissure is a grey spot perforated by small arteries, the *anterior perforated space*.

The peripheral part of each hemisphere consists of grey matter, and exhibits a characteristic folded appearance, known as the *convolutions* or *gyri* of the cerebrum. The convolutions are separated from each other by *fissures* or *sulci*, some of

which are considered to subdivide the hemispheres into *lobes*, whilst others separate the convolutions of each lobe from each other.

FIG. 191.

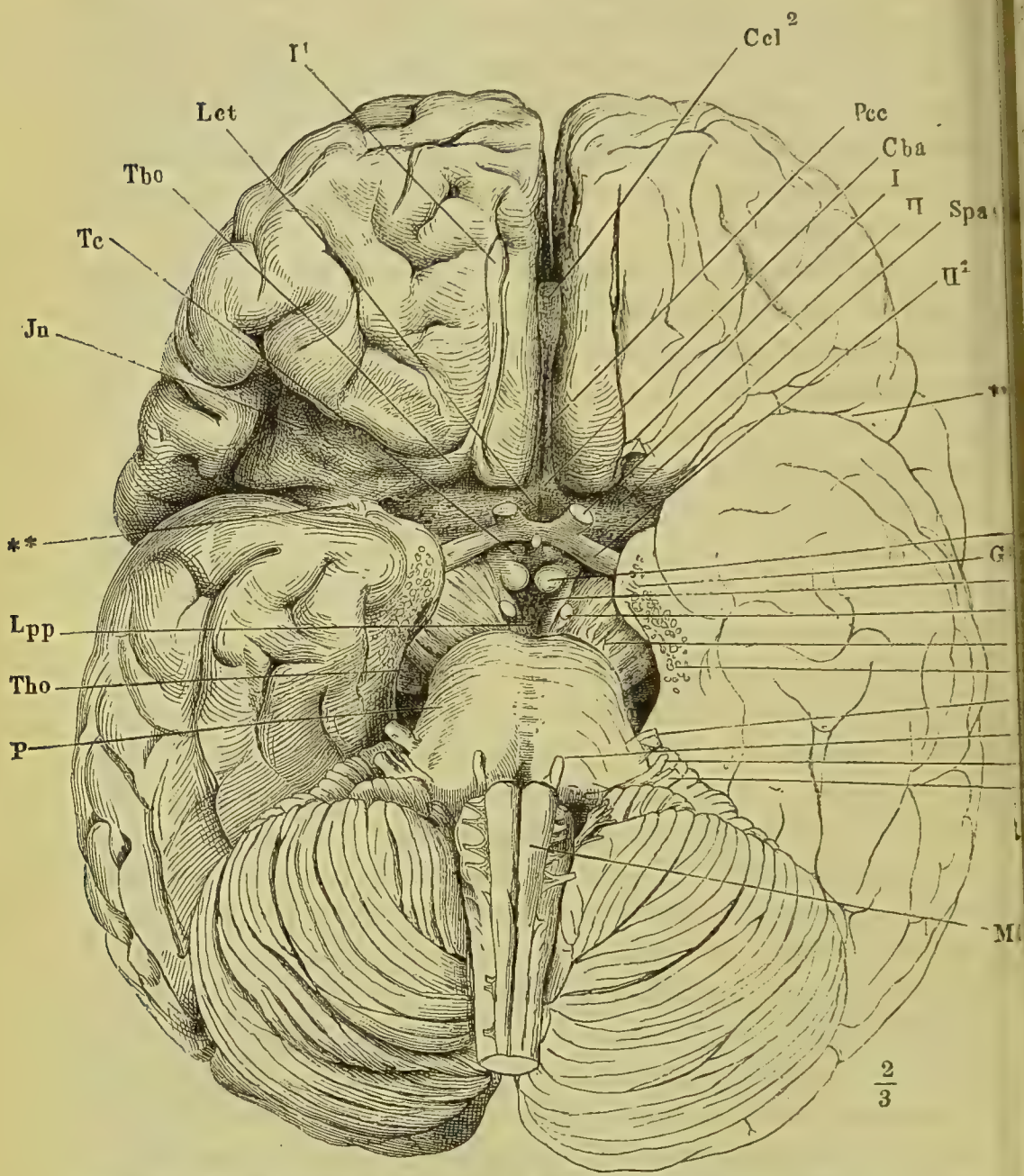


FIG. 191 (From Henle's Anatomie). *Base of the Brain.*—P, *Pons;* Tho, *Optic thalamus;* Lpp, *Posterior perforated space;* Jn, *Island of Reil.*

Tbo, Olfactory bulb.
Lct, Lamina cinerea. Tc, Tuber cinereum.
Ccl², Knee of the corpus callosum.
Pcc, Peduncles of the corpus callosum.
Cba, Commissure of the corpus callosum.
Spa, Anterior perforated space.
Cc, Corpora albicantia.
Gf, Gyrus fornicatus.
T, Tegmentum.
B, Crusta.
Sr, Substantia reticularis.
Mo, Medulla oblongata.

The Roman letters indicate the corresponding cranial nerves: I, Olfactory nerve; I¹, Olfactory bulb; II, Optic nerve; II¹, Optic tract; *, Sylvian fissure; **, the point of the temporo-sphenoidal lobe drawn back to show the continuity of this lobe with the posterior convolution of the Island of Reil.

§ 672. *Lobes of the Cerebrum.*—They are five in number, named respectively *frontal, parietal, occipital, temporo-sphenoidal,* and *central.* The divisions between these lobes are marked partly by certain conspicuous fissures, and partly by artificial lines.

§ 673. *The Primary Fissures.*—The *Sylvian fissure* is the first to appear in the development of the hemisphere. It passes obliquely along the outer surface of the hemisphere from before backwards, and upwards. In man it divides into two rami—the posterior or horizontal (*Fig.* 192, S′), and the ascending or anterior (*Fig.* 192, S″). The portion included between these two branches receives the name of the *operculum,* and forms the roof of the central lobe or Island of Reil. Below the fissure of Sylvius lies the temporo-sphenoidal lobe, and above and in front of it the parietal and frontal lobes. The frontal is separated from the parietal lobe by the *fissure of Rolando* (*Fig.* 192, *c*) or *Central Sulcus.* It extends from the longitudinal fissure obliquely, downwards and forwards, along the outer surface of the hemisphere towards the Sylvian fissure. The *parieto-occipital fissure* commences at the longitudinal fissure, about two inches from the posterior end of the hemisphere. It passes down the inner surface of the hemisphere, and also transversely outwards for a short distance on the outer surface, and separates the parietal and occipital lobes from each other.

§ 674. *Secondary Fissures and Convolutions.*—The *temporo-sphenoidal lobe* presents on the outer surface of the hemispheres three parallel convolutions, named the *superior* (*Fig.* 192, T1), *middle* (*Fig.* 192, T2), and *inferior temporo-sphenoidal* (*Fig.* 192, T3) *convolutions.*—The fissure which separates the superior and middle of these convolutions is called the *parallel fissure.*

The *occipital lobe* also consists of three parallel convolutions, named *superior* (*Fig.* 192, O1), *middle* (*Fig.* 192, O2), and *inferior* (*Fig.* 192, O3) *occipital convolutions.*

The *frontal lobe* consists of three convolutions arranged in parallel tiers from above downwards, and named *superior* (*Fig.* 192, F1), *middle* (*Fig.* 192, F2), and *inferior* (*Fig.* 192, F3) *frontal convolutions.* These are prolonged anteriorly to the

orbital surface of the frontal lobe, and terminate posteriorly in the convolution which forms the anterior boundary of the fissure of Rolando, named the *ascending frontal convolution* (*Fig.* 192, A).

The secondary fissures which separate the superior, middle, and inferior frontal convolutions from one another are the *supero-frontal* (*Fig.* 192, *f*1), and the *infero-frontal* (*Fig.*

FIG. 192.

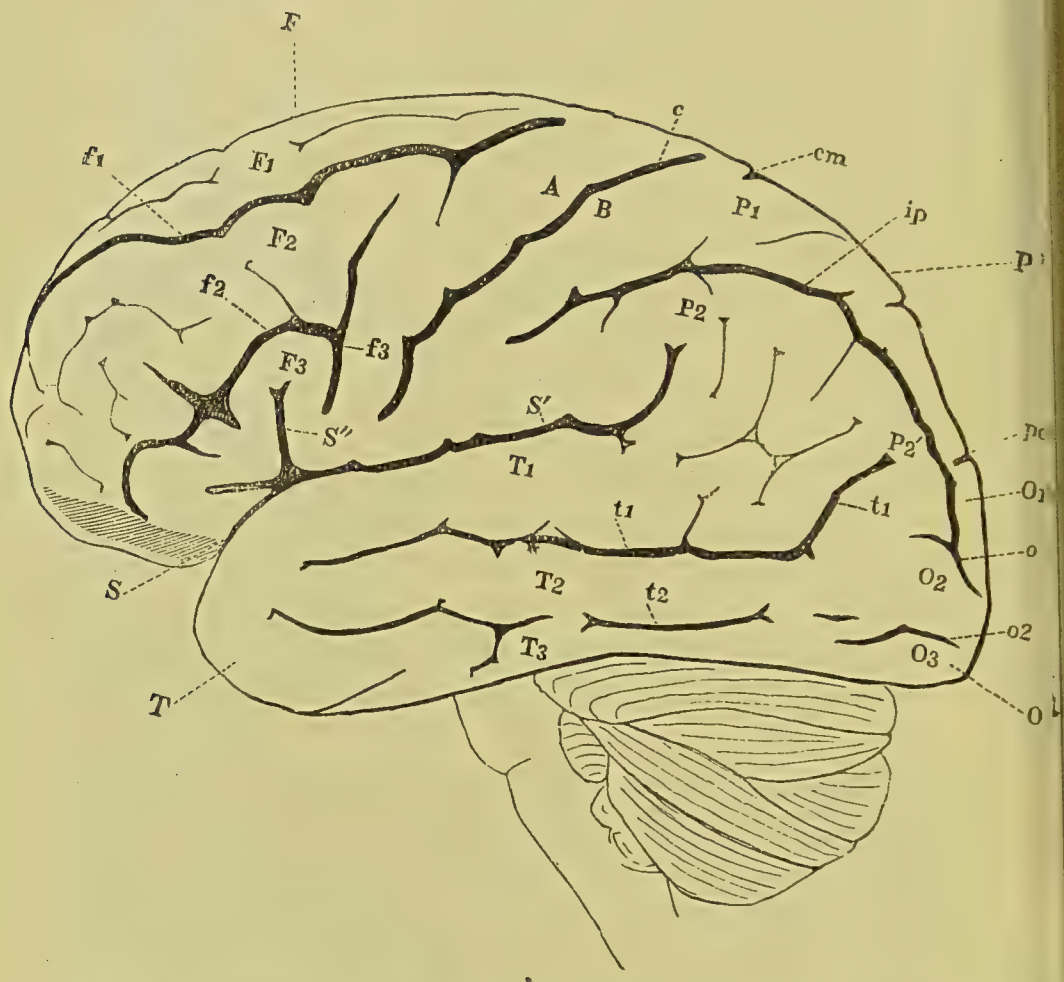


FIG. 192 (Ecker). *Lateral View of the Human Brain.*—F, Frontal lobe. P, Parietal lobe. O, Occipital lobe. T, Temporo-sphenoidal lobe. S, Fissure of Sylvius, S′ Horizontal, S″ Ascending ramus of the same. *c*, Sulcus centralis, or fissure of Rolando. A, Anterior central or ascending frontal convolution. B, Posterior central or ascending parietal convolution. F_1 Superior, F_2 Middle, and F_3 Inferior frontal convolutions. f_1 Superior and f_2 Inferior frontal sulcus; f_3 Sulcus præ-centralis. P_1 Superior parietal or postero-parietal lobule; P_2 Inferior parietal lobule, viz. P_2 Gyrus supra-marginalis, P_2' Gyrus angularis. *ip*, Sulcus intra-parietalis. *cm*, Termination of the calloso-marginal fissure. O_1 First, O_2 Second, O_3 Third occipital convolutions. *po*, Parieto-occipital fissure. *o*, Sulcus occipitalis transversus; o_2 Sulcus occipitalis longitudinalis inferior. T_1 First, T_2 Second, T_3 Third temporo-sphenoidal convolutions. t_1 First, t_2 Second temporo-sphenoidal fissures.

192, *f*2), while the continuity of the three frontal convolutions with the ascending frontal one is interrupted by the *antero-parietal sulcus,* or *Sulcus Præ-centralis* (*Fig.* 192, *f*3). The ascending ramus of the fissure of Sylvius (*Fig.* 192, S″) also interrupts the continuity of the inferior frontal with the ascending frontal convolution.

FIG. 193.

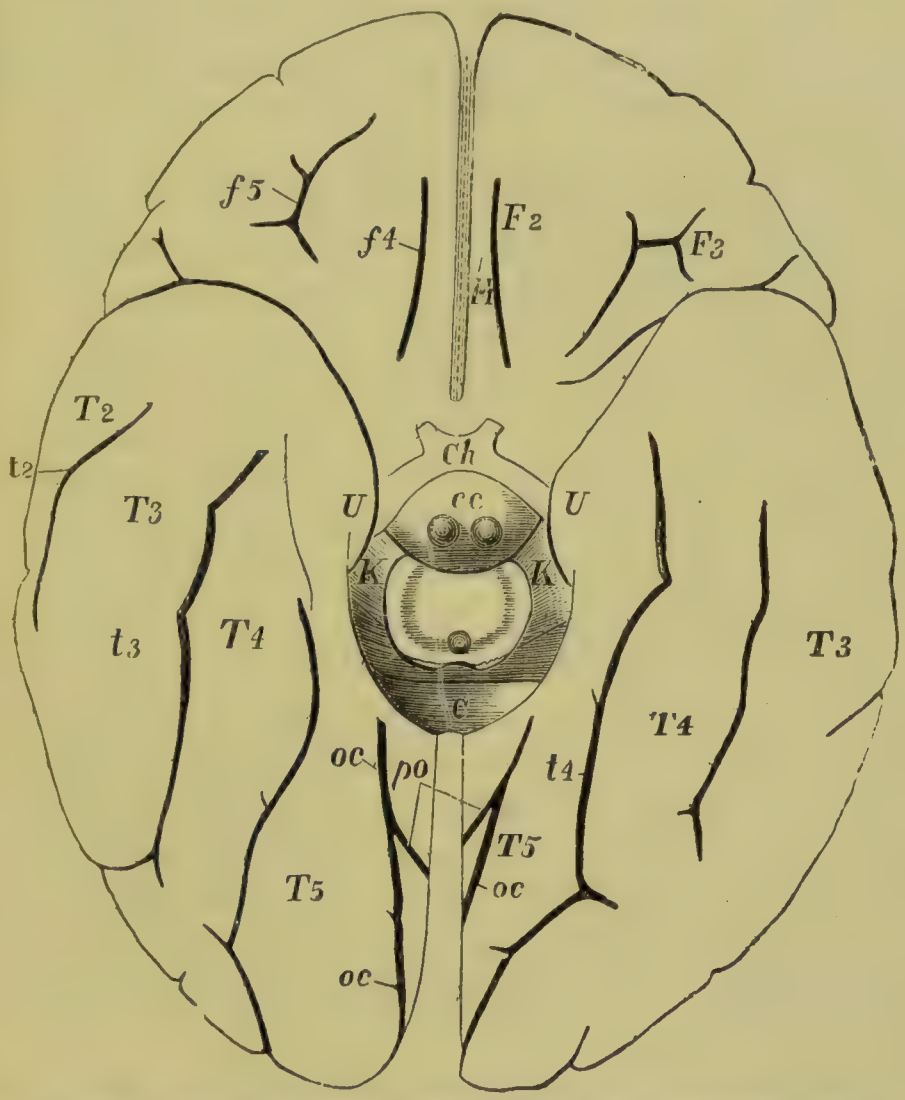


FIG. 193 (After Ecker and Duret). *View of the Brain from below.*

*F*1, Gyrus Rectus.
*F*2, Middle frontal convolution.
*F*3, Inferior frontal convolution.
*f*4, Sulcus olfactorius.
*f*5, Sulcus orbitalis.
*T*2, Second or middle temporo-sphenoidal convolution.
*T*3, Third or inferior temporo-sphenoidal convolution.
*T*4, Gyrus occipito-temporalis lateralis (lobulus fusiformis).
*T*5, Gyrus occipito-temporalis medialis (lobulus lingualis).
*t*4, Sulcus occipito-temporalis inferio
*t*3, Sulcus temporo-sphenoidalis inferior.
*t*2, Sulcus temporo-sphenoidalis medialis.
po, Parieto-occipital fissure.
oc, Calcarine fissure.
H, Gyrus hippocampi.
U, Gyrus uncinatus.
Ch, Optic chiasma.
cc, Corpora albicantia.
KK, Crura cerebri.
C, Corpus callosum.

The *orbital surface* or *orbital lobule* presents two fissures—the *olfactory sulcus*, which runs parallel with the longitudinal fissure and lodges the olfactory bulb, and the *orbital sulcus* (*Fig.* 193, *f5*), which lies in the centre of the lobule and is often triradiate. The *straight* convolution (*Fig.* 193, F1) lies between the longitudinal fissure and the olfactory sulcus, and is continuous at its anterior extremity with the superior frontal convolution. Three convolutions are sometimes described as lying around the orbital sulcus, named, according to their positions, the *internal*, the *anterior*, and the *posterior* orbital convolutions.

The *parietal lobe* presents several convolutions. The most anterior is the *ascending parietal* convolution (*Fig.* 192, B), which lies immediately behind the fissure of Rolando, and is bounded posteriorly by a sulcus termed the *intra-parietal sulcus* (*Fig.* 192, *ip*). The *postero-parietal convolution* or *superior parietal lobule* (*Fig.* 192, P1) springs from the upper end of the back of the ascending parietal convolution, and forms the boundary of the longitudinal fissure, extending as far back as the *parieto-occipital* fissure. The *supra-marginal convolution* (*Fig.* 192, P2) springs from the lower end of the ascending parietal convolution at its posterior aspect, and arches round the posterior extremity of the Sylvian fissure. The *angular gyrus* (*Fig.*192,P2′) is continuous with the supra-marginal convolution, and bends round the posterior extremity of the parallel fissure. The supra-marginal convolution and angular gyrus have together been described as the *inferior parietal lobule* (Ecker), or the *convolutions of the parietal eminence* (Turner). They occupy the hollow in the parietal bone which corresponds with the parietal eminence.

The occipital is connected with the parietal lobe by two *annectant* or *bridging gyri*. The *superior annectant gyrus* passes between the postero-parietal and the superior occipital convolutions, whilst the *second annectant gyrus* connects the middle occipital with the angular gyrus. Two annectant gyri also pass from the inferior occipital convolution to the lower convolutions of the temporo-sphenoidal lobe.

The *central lobe*, or *Island of Reil*, lies deeply within the fissure of Sylvius, being invisible except when the lips of the

fissure are separated. It consists of about six short, straight convolutions (gyri operti), which radiate outwards from the anterior perforated space. The anterior convolution is continuous with the adjacent posterior orbital convolution, while the posterior convolution joins the temporo-sphenoidal lobe. Externally, the island of Reil is separated by a deep sulcus from the contiguous convolutions of the operculum, and it covers the *lenticular nucleus* of the corpus striatum.

The small convolutions which lie behind the parieto-occipital fissure form the internal convolutions of the occipital lobe, named the *internal occipital lobule*, or *cuneus* (*Fig.* 194, Oz). Those which lie immediately in front of the internal part of the parieto-occipital lobule and between it and the curved posterior extremity of the calloso-marginal fissure are called

FIG. 194.

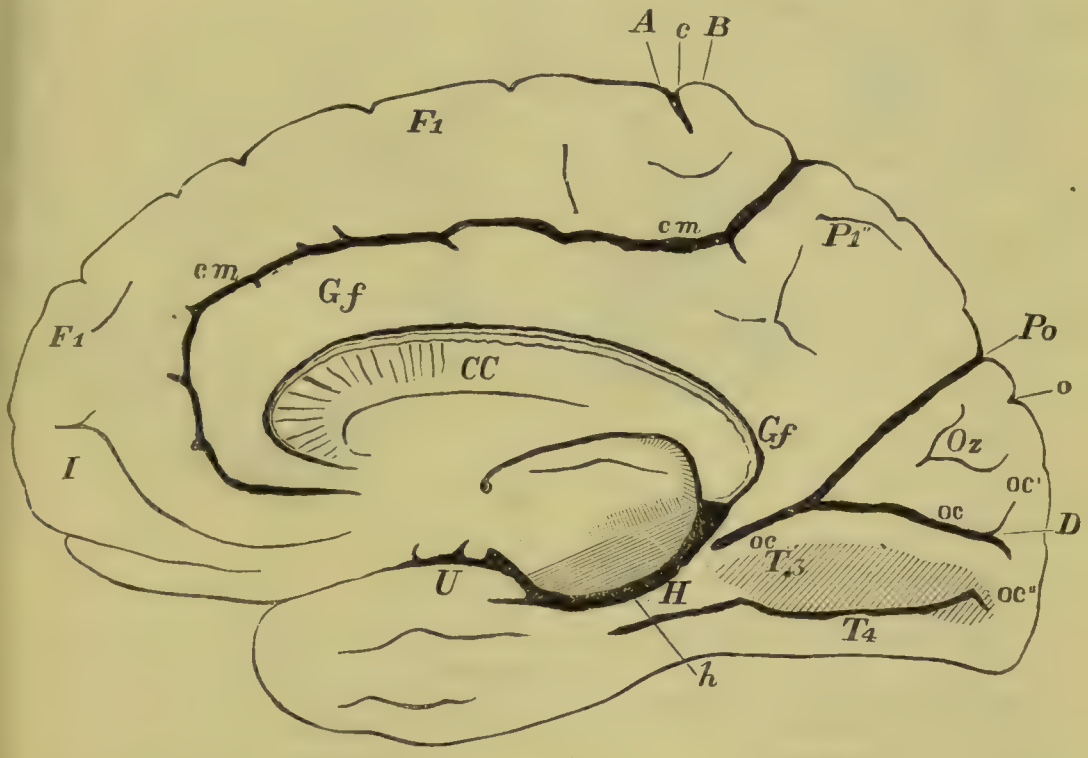


FIG. 194 (Ecker). *View of the Median Aspect of the Right Hemisphere of the Human Brain.*—CC, Corpus callosum, longitudinally divided. Gf, Gyrus fornicatus. H, Gyrus hippocampi. h, Sulcus hippocampi. U, Uncinate gyrus. cm, Sulcus calloso-marginalis. F_1, Median aspect of the first frontal convolution. c, Terminal portion of the sulcus centralis, or fissure of Rolando. A, Anterior; B, Posterior central convolution. P_1'', Præcuneus. Oz, Cuneus. Po, Parieto-occipital fissure. o, Sulcus occipitalis transversus. oc, Calcarine fissure. oc′, Superior; oc″, Inferior ramus of the same. D, Gyrus descendens. T_4, Gyrus occipito-temporalis lateralis (lobulus fusiformis). T_5, Gyrus occipito-temporalis medialis (lobulus lingualis).

the *præcuneus* or *quadrilateral lobule* (*Fig.* 194, $P_{1''}$). The *paracentral lobule* lies immediately in front of the præcuneus. It consists of the upper extremities of the ascending frontal and parietal convolutions, viewed from the internal surface of the hemisphere. It is cutomary to name the convolution which

FIG. 195.

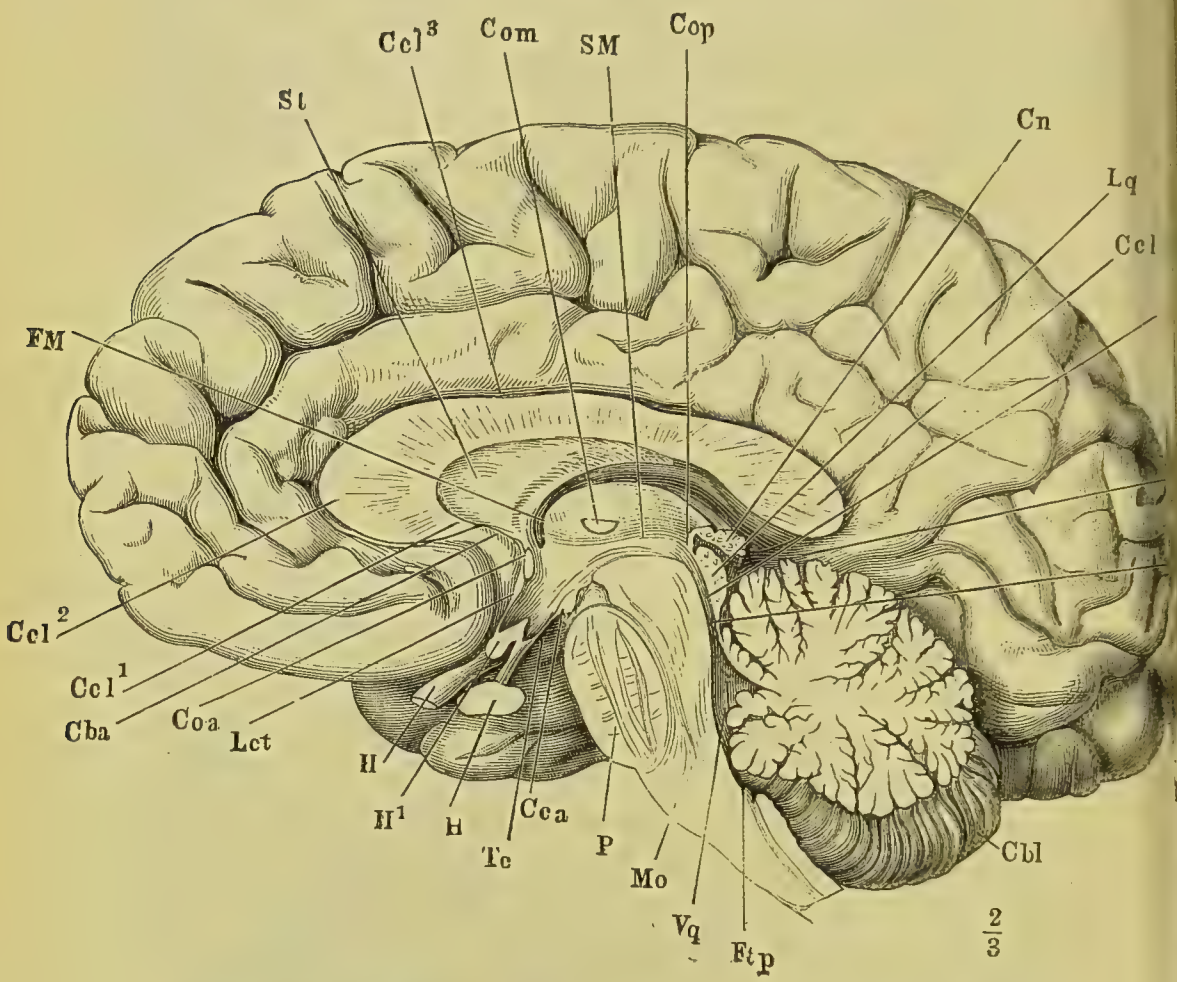


FIG. 195 (From Henle's Anatomie). *Internal View of the Hemisphere of the Cerebrum.*

Ftp, Posterior transverse fissure.
Vq, Fourth ventricle.
Mo, Medulla oblongata.
P, Pons.
Cca, Corpora caudicantia.
Tc, Tuber cinereum.
H, Pituitary body.
II^1, Optic tract.
II, Optic nerve.
Lct, Lamina cinerea.
Coa, Anterior commissure of the third ventricle.
Cba, Commissure of the corpus callosum.
Ccl^1, Rostrum.
Ccl^2, Knee of the callosum.
Ccl^3, Corpus callosum.
Ccl^4, Splenium of the corpus callosum.
Sl, Septum lucidum.
Com, Median commissure of the third ventricle.
SM, Sulcus Monroi.
Cop, Posterior commissure of the third ventricle.
Cn, Pineal gland.
Lq, Corpora quadrigemina.
A, Aqueduct of Sylvius.
Fta, Anterior transverse fissure.
Vma, Anterior medullary velum.
Cbl, Cerebellum.

extends forwards from the parieto-occipital fissure along the margin of the longitudinal fissure to the anterior extremity of the hemisphere, and which then turns back to the anterior perforated space, the *marginal convolution.*

The *internal* is not divided into lobes like the external surface, but the convolutions may be studied in connection with the corpus callosum and with certain fissures situated in this surface.

The *parieto-occipital* fissure (*Fig.* 194, Po) is continuous with the fissure of the same name on the external surface. It extends downwards and forwards, and blends with the calcarine fissure. The *calcarine fissure* (*Fig.* 194, oc) commences at the posterior extremity of the hemisphere, usually in a bifurcated manner, and extends forwards to terminate beneath the posterior extremity of the corpus callosum. It marks the position of the *calcar avis* or *hippocampus minor* in the posterior cornu of the lateral ventricle. The *calloso-marginal fissure* (*Fig.* 194, cm) commences beneath the anterior extremity of the corpus callosum, and passes forwards, upwards, backwards, round the corpus callosum, terminating behind the superior extremity of the ascending parietal convolution.

The convolution which immediately bounds the corpus callosum is termed the *gyrus fornicatus* (*Fig.* 194, Gf). It begins at the anterior perforated space, turns round the anterior end of the corpus callosum, extends parallel to its upper surface, and then turns round its posterior end. It is separated from the corpus callosum by the *callosal fissure,* and from the marginal convolution by the calloso-marginal fissure. The posterior end of the gyrus fornicatus curves downwards and then forwards under the name of *gyrus uncinatus,* or *gyrus hippocampi* (*Fig.* 194, H), to the tip of the inner surface of the temporo-sphenoidal lobe. The uncinate gyrus ends anteriorly in a crook-like extremity, or crochet, named the *uncus gyri fornicati,* or *subiculum cornu ammonis* (*Fig.* 194, U). The gyrus is separated anteriorly by a narrow-curved fissure, called *hippocampal* or *dentate fissure,* from a white band named the *tænia hippocampi.* This band possesses a free-curved border, round which the pia mater enters the lateral ventricle through the great transverse fissure of the

cerebrum. The grey matter of the gyrus hippocampi terminates at the bottom of the hippocampal fissure in a well-defined dentated border named the *fascia dentata.* The hippocampal fissure marks the position of an eminence in the descending cornu of the ventricle called the *hippocampus major.*

Running along the internal aspect of the occipital and temporo-sphenoidal lobes is a fissure termed the *collateral,* which marks the position of the collateral eminence in the lateral ventricle. It separates two convolutions from each other which connect the occipital and temporo-sphenoidal lobes with each other, and are therefore named the *occipito-temporal convolutions* (*Fig.* 194, T4, T5). The upper of these is termed the *gyrus occipito-temporalis medialis,* or *lingual lobule* (T5); while the lower is named the *gyrus occipito-temporalis lateralis,* or *lobulus fusiformis* (T4).

§ 675. RELATIONS OF THE CONVOLUTIONS TO THE SKULL.

The relations of the primary fissures and convolutions of the brain with relation to the skull have been investigated by Broca, Féré, Turner, and others. The following is an abstract of Turner's conclusions :—

Definite Landmarks on the Surface of the Skull.—The following structures and markings are easily recognised on the skull. The external occipital protuberance (*Fig.* 196, *o*), the parietal (P) and frontal (F) eminences, and the external angular process of the frontal bone (A), the coronal (*c*) and lambdoidal (*l*), squamous (*s*), squamoso-sphenoid (*ss*), and parieto-sphenoid sutures (*ps*), and the curved line of the temporal ridge (*t*).

Primary Areas of the Skull.—The coronal suture (*c*) forms the posterior boundary of the *frontal area.* A vertical line (*Fig.* 196, 2) drawn from the squamous suture (*s*) upwards through the parietal eminence (P) to the sagittal suture lies almost parallel to the coronal suture, and subdivides the parietal region into an *antero-parietal* (*Fig.* 196, SAP + IAP) and a *postero-parietal area* (*Fig.* 196, SPP + IPP). The *occipital region* lies between the lambdoidal suture (*l*) and the occipital protuberance (*o*), with the superior curved line extending from it (*Fig.* 196, *o*).

Secondary Areas of the Skull.—These four primary divisions of the skull may be subdivided into secondary areas. The temporal ridge (*Fig.* 196, *t*) starting from the external frontal process curves backwards across the frontal (A), antero-parietal, and post-parietal areas to the internal angle of the occipital bone, and subdivides each of these regions into an upper and

a lower area. The upper frontal area, which includes all the frontal regions above the temporal ridge, is again divided by a line drawn vertically upwards and backwards from above the orbit through the frontal eminence to the coronal suture (*Fig.* 196, *c*). This line divides the upper frontal area into a *supero-frontal* (SF) and a *mid-frontal area* (MF).

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

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

FIG. 196.

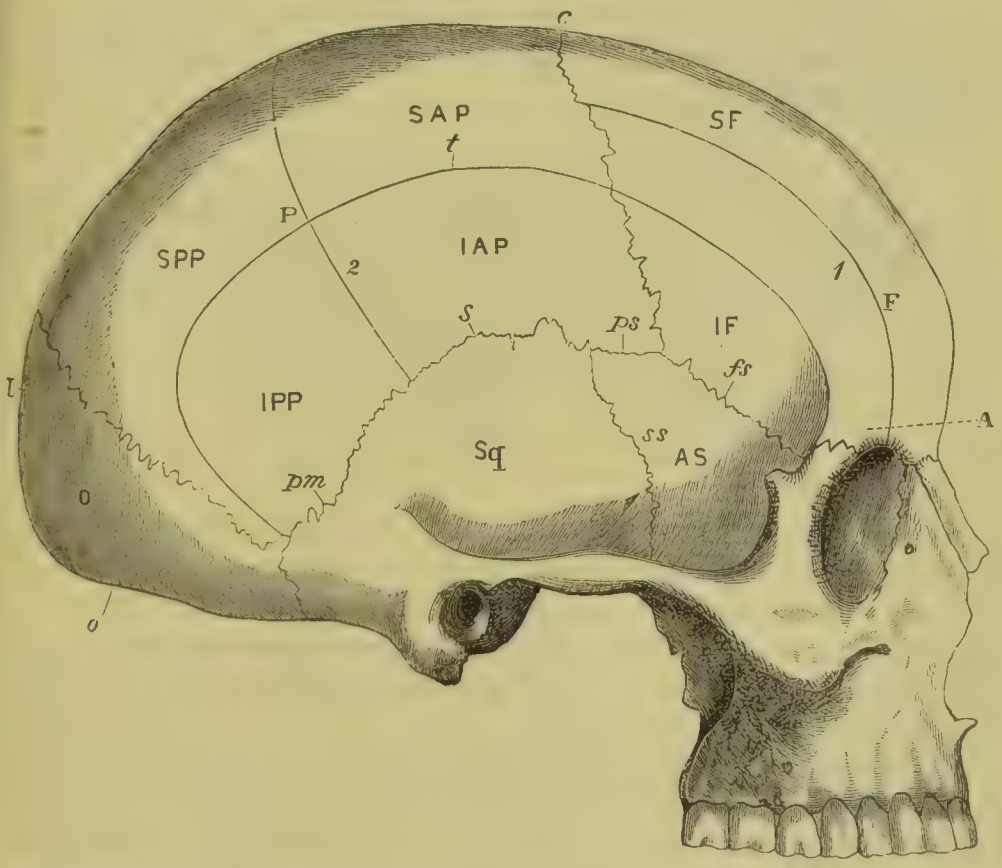


FIG. 196 (Ferrier). *Lateral View of the Human Skull.*—A, The external angular process of the frontal bone. F, The frontal eminence. P, The parietal eminence. *o*, The occipital protuberance. *c*, The coronal suture. *l*, The lambdoidal suture. *s*, The squamous suture. *t*, The temporal ridge. *fs*, The fronto-sphenoid suture. *ps*, The parieto-sphenoid suture. *ss*, The squamoso-sphenoid suture. *pm*, The parieto-mastoid suture. 1, Frontal line. 2, Parietal line. SF, MF, IF, The supero-, mid-, and infero-frontal subdivisions of the frontal area. SAP, The supero-antero-parietal area. IAP, The infero-antero-parietal area. SPP, The supero-postero-parietal area. IPP, The infero-postero-parietal area. O, The occipital area. Sq, The squamoso-temporal area. AS, The ali-sphenoid area.

RELATIONS OF THE CONVOLUTIONS AND FISSURES OF THE BRAIN TO THE AREAS OF THE SKULL.

§ 676. The fissure of Sylvius (*Fig.* 197, SS) commences behind the posterior border of the lesser wing of the sphenoid, and courses upwards and backwards below the greater wing of the sphenoid, where it articulates with the anterior inferior angle of the parietal bone, and then appears in the lower part of the inferior antero-parietal region. The fissure of Rolando (*Fig.* 197, *R*) lies in the antero-parietal region, both in its superior and inferior divisions, its upper extremity being as much as two inches and its lower one and a half inch behind the respective ends of the coronal suture. The coronal suture does not, therefore, correspond to the boundary between the frontal and parietal lobes of the brain.

The parieto-occipital fissure is situated on an average about 0·7 to 0·8 inch in front of the apex of the lambdoidal suture (*Fig.* 197, PO).

Contents of the Respective Areas.

The *frontal area* is occupied by the frontal lobe, but does not cover the whole of it, the posterior extremities of the three frontal convolutions lying behind the coronal suture. The frontal area therefore corresponds to the part of the frontal lobe supplied by the anterior cerebral artery, and which is not excitable to stimulation. The superior, middle, and inferior frontal areas of the skull correspond respectively to the superior, middle, and inferior frontal convolutions, with the exception of their posterior extremities.

The *upper antero-parietal area* (*Fig.* 197, SAP) contains the upper two-thirds of the ascending frontal (AP) and ascending parietal (S) convolution, and the posterior extremities of the superior (1·2in.) and middle frontal (1·3in.) convolutions. At the upper posterior angle of this area part of the postero-parietal lobule is visible, and below this, part of the supra-marginal lobule may appear.

The *lower antero-parietal area* (*Fig.* 197, IAP) contains the lower third of the ascending parietal (1in.) and ascending frontal (AP) convolutions, and the posterior extremities (1in.) of the inferior frontal convolution. A small portion of the supra-marginal gyrus is visible at the upper posterior angle of this area, and below it a small portion of the superior temporo-sphenoidal convolution.

The *upper postero-parietal area* (*Fig.* 197, SPP) contains the greater part of the postero-parietal lobule. Below it lies the upper portion of the angular gyrus (SPP), and part of the supra-marginal gyrus (×). Posteriorly the annectant gyri blend with the occipital lobe.

The *lower postero-parietal area* (*Fig.* 197, IPP) contains part of the supra-marginal gyrus, and behind it part of the angular gyrus, and below this the posterior or upper ends of the temporo-sphenoidal convolutions.

The *occipital area* (*Fig.* 197, O) indicates the situation of the occipital lobe, but is not co-extensive with it, inasmuch as a portion extends anteriorly beyond the lambdoidal suture into the postero-parietal area.

The *squamoso-temporal area* (*Fig.* 197, Sq) contains the greater portion of the temporo-sphenoidal convolutions, but the superior temporo-sphenoidal convolution ascends into the lower parietal areas.

Fig. 197.

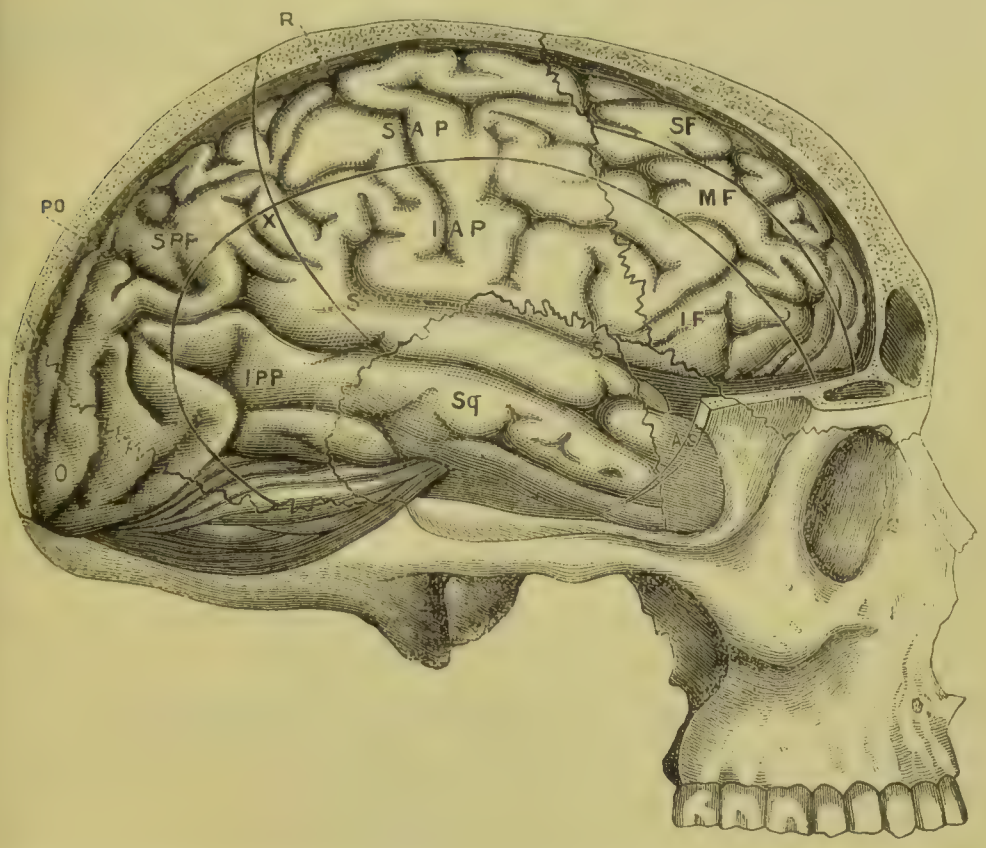


Fig. 197 (Turner). *Diagram showing the Relations of the Convolutions of the Skull.* R, The fissure of Rolando, which separates the frontal from the parietal lobe. PO, The parieto-occipital fissure between the parietal and occipital lobes. SS, The fissure of Sylvius, which separates the temporo-sphenoidal from the frontal and parietal lobes. SF, MF, IF, The supero, mid-, and infero-frontal subdivisions of the frontal area of the skull; the letters are placed on the superior, middle and inferior frontal convolutions. SAP, The supero-antero-parietal area of the skull: S is placed on the ascending parietal convolution, AP on the ascending frontal convolution. IAP, The infero-antero-parietal area of the skull: I is placed on the ascending parietal, AP on the ascending frontal convolution. SPP, The supero-postero-parietal area of the skull: the letters are placed on the angular convolution. IPP. The infero-postero-parietal area of the skull: the letters are placed on the mid-temporo-sphenoidal convolution. ×, The convolution of the parietal eminence, or supra-marginal gyrus. O, The occipital area of the skull: the letter is placed on the mid-occipital convolution. Sq, The squamoso-temporal region of the skull: the letters are placed on the mid-temporo-sphenoidal convolution. AS, The ali-sphenoid region of the skull: the letters are placed on the tip of the supero-temporo-sphenoidal convolution.

The *ali-sphenoidal area* (*Fig.* 197, AS) contains the lower or anterior extremity of the temporo-sphenoidal lobe.

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

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

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

§ 677. INTERNAL PARTS OF THE CEREBRUM.

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

Centrum Ovale.—A horizontal section made half an inch above the corpus callosum displays the white matter of each hemisphere surrounded on all sides by the grey matter of the convolutions. The white central mass in each hemisphere was named by Vicq. d'Azyr the *centrum ovale minus.* A section made at the level of the corpus callosum shows that the white substance of that part is continuous with the central white substance of each hemisphere. The large white medullary mass thus displayed is named the *centrum ovale majus.*

The *Corpus Callosum* connects the centres of the two hemispheres, and it approaches nearer their anterior than their posterior extremities. It terminates behind in a free rounded end—the *splenium,* whilst in front it forms a knee-shaped bend, and passes downwards and backwards as far as the lamina cinerea. It is thicker behind than in front, the middle part being the thinnest. It consists of bundles of nerve fibres, almost the whole of which pass transversely between the two hemispheres. The fibres may be traced into the white cores and grey matter of the convolutions, and apparently connect corresponding convolutions in the opposite hemispheres. A few fibres run longitudinally on the surface of the corpus callosum, named the *striæ longitudinales* or *nerves of Lancisi.*

Topography of the Centrum Ovale.—A systematic nomen-

clature of the various parts of the centrum ovale has been devised by Pitres. His system consists in making vertical sections of the brain at definite points, and naming the various parts of the medullary substance exposed in each section. A vertical section of the hemisphere at right angles to its longitudinal axis in the præ-frontal region gives the *præ-frontal section* (*Fig.* 198). The next section is made two centimètres

FIG. 198.

FIG. 198 (After Pitres). *Præ-frontal Section.*—1, 2, 3, First, second, and third frontal convolutions. 4, Orbital convolutions. 5, Convolutions on the internal aspect of the frontal lobe. 6, Præ-frontal fasciculi of the centrum ovale.

FIG. 199.

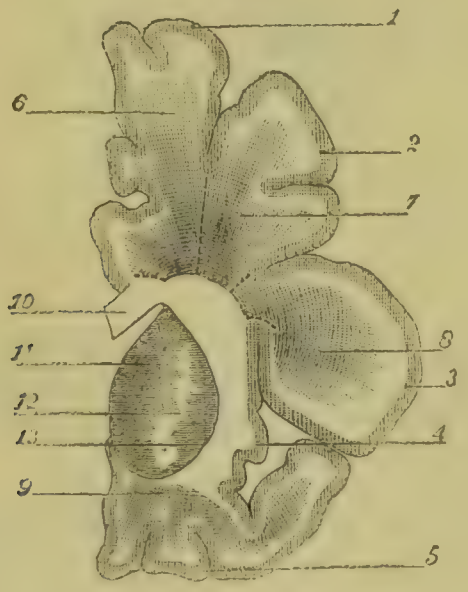


FIG. 199 (After Pitres). *Pedunculo-frontal Section.*—1, 2, 3, First, second, and third frontal convolutions. 4, Anterior extremity of the insular lobe. 5, Posterior extremity of the orbital convolutions. 6, Superior pedunculo-frontal fasciculus. 7, Middle pedunculo-frontal fasciculus. 8, Inferior pedunculo-frontal fasciculus. 9, Orbital fasciculus. 10, Corpus callosum. 11, Caudate nucleus. 12, Internal capsule. 13, Lenticular nucleus.

in front of the fissure of Rolando and passes through the bases of the three frontal convolutions, and is named the *pedunculo-frontal section* (*Fig.* 199). The medullary substance in this section is subdivided into a *superior, middle,* and *inferior pedunculo-frontal fasciculus,* corresponding with the respective frontal convolutions. The next section is made through the ascending frontal convolution, parallel with the fissure of Rolando, and is named the *frontal section.* It also passes through a small portion of the sphenoidal lobe. The medullary substance of this section is also subdivided into *superior, middle,* and *inferior frontal fasciculi* (*Fig.* 200). The fourth section

Fig. 200.

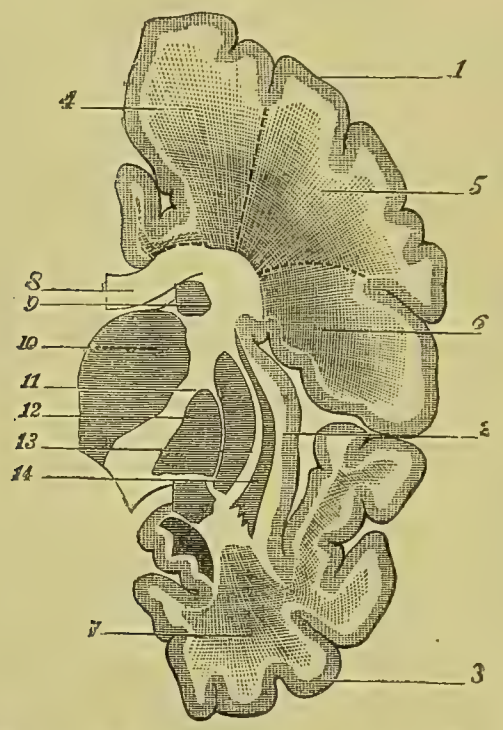


Fig. 200 (After Pitres). *Frontal Section.*—1, Ascending frontal convolution. 2, Insular lobule. 3, Sphenoidal lobe. 4, 5, 6, Superior, middle, and inferior frontal fasciculus. 7, Sphenoidal fasciculus. 8, Corpus callosum. 9, Caudate nucleus. 10, Optic thalamus. 11, Internal capsule. 12, Lenticular nucleus. 13, External capsule. 14, Claustrum.

is carried through the ascending parietal convolution, and is named the *parietal section.* It is subdivided into *superior, middle,* and *inferior parietal fasciculi* (*Fig.* 201). The next is the *pedunculo-parietal section,* made by dividing the hemisphere three centimètres behind the fissure of Rolando, and cutting the superior and inferior parietal lobules. It is sub-

FIG. 201.

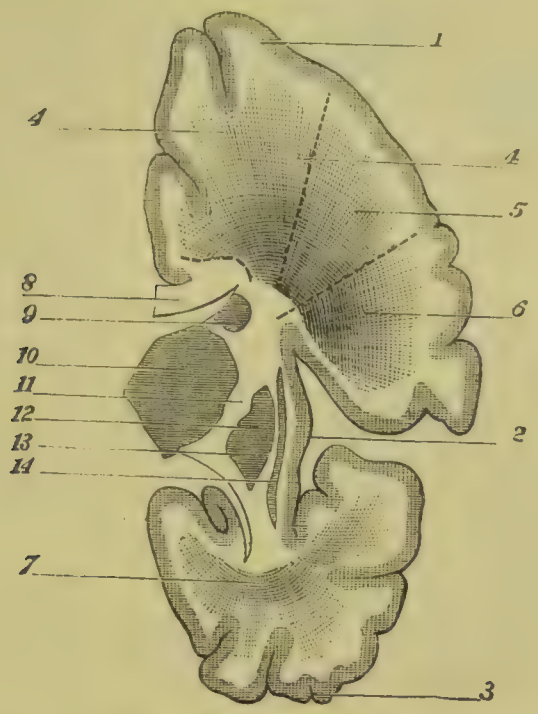


FIG. 201 (After Pitres). *Parietal Section.*—1, Ascending parietal convolution. 2, Insular lobe. 3, Sphenoidal lobe. 4, Superior parietal fasciculus. 5, Middle parietal fasciculus. 6, Inferior parietal fasciculus. 7, Sphenoidal fasciculus. 8, 9, 10, 11, 12, 13, 14, as in the preceding figure.

FIG. 202.

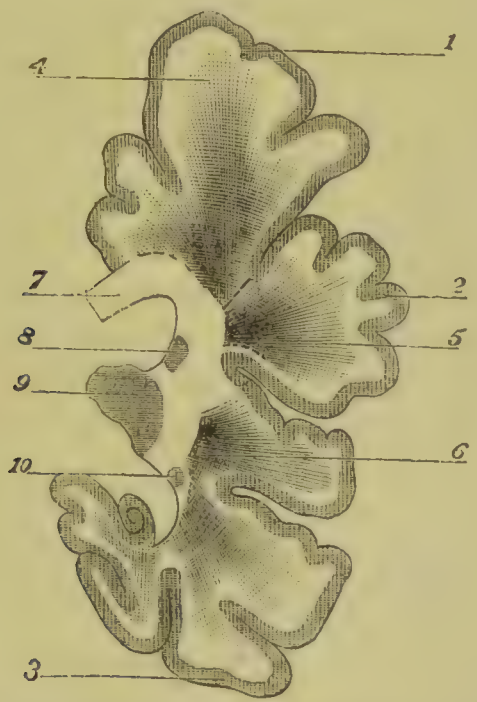


FIG. 202 (After Pitres). *Pedunculo-parietal Section.*—1, Superior parietal lobule. 2, Inferior parietal lobule. 3, Sphenoidal lobe. 4, Superior pedunculo-parietal fasciculus. 5, Inferior pedunculo-parietal fasciculus. 6, Sphenoidal fasciculus. 7, Corpus callosum. 8 and 10, Caudate nucleus. 9, Optic thalamus.

divided into *superior* and *inferior* pedunculo-parietal and *sphenoidal fasciculi* (*Fig.* 202).

The last is the *occipital section* (*Fig.* 203) in which no separate fasciculi are distinguished.

FIG. 203.

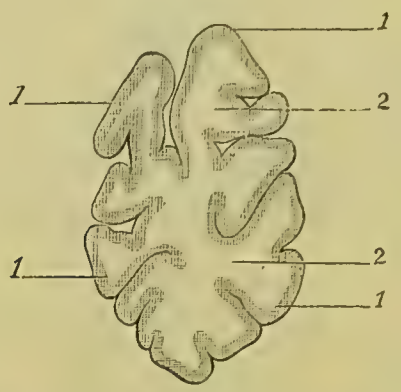


FIG. 203 (After Pitres). *Occipital Section.*—1, Occipital convolutions. 2, Occipital fasciculi of the centrum ovale.

Lateral Ventricles.—The lateral ventricle is divided into a *central space* or body, and three curved prolongations or *cornua.* The *anterior cornu* extends forwards and outwards into the frontal lobe, the *posterior* curves backwards, outwards, and inwards into the occipital lobe, and the *descending cornu* curves backwards, outwards, downwards, forwards, and inwards, behind and below the optic thalamus into the temporo-sphenoidal lobe.

On the floor of the central space may be seen from before backwards the *caudate nucleus,* and to its inner and posterior part a small portion of the *optic thalamus,* whilst between the two is a curved flat band, the *tænia semicircularis.* The *choroid plexus* rests on the upper surface of the optic thalamus, and immediately internal to it is the free edge *of the fornix.*

The anterior end of the caudate nucleus projects into the anterior cornu, while the posterior cornu has an elevation on its floor, named the *hippocampus minor,* and the *eminentia collateralis* lies between the posterior and descending cornua. The *hippocampus major* extends along the floor of the descending cornu, and terminates below in a nodular end, the *pes hippocampi.* Along its inner edge is a narrow white band prolonged from the posterior pillar of the fornix, named the *tænia hippocampi.* If the tænia be drawn aside the hippocampal fissure

is exposed, at the bottom of which the grey matter of the gyrus hippocampi may be seen to form a serrated border, named the *fascia dentata.* The choroid plexus enters the descending cornu through the great transverse fissures of the brain between the tænia hippocampi and optic thalamus. The lateral ventricle is lined by cylindrical epithelium, which rests on a layer of neuroglia, and is in many parts ciliated. This lining is continuous with that of the third ventricle through the foramen of Monro, the lining of the latter being continuous with that of the fourth ventricle through the aqueduct of Sylvius. A little fluid is contained in the cerebral ventricles.

Septum Lucidum.—If the corpus callosum be divided transversely about its middle, and the two halves reflected forwards and backwards respectively, the fornix and *septum lucidum* are exposed. This septum extends vertically between the corpus callosum above and the fornix below. It consists of two layers of grey matter, having an interval between them containing fluid, and covered by an epitheliated membrane. This space is the *fifth ventricle.*

The *fornix* is an arch-shaped band of nerve fibres which extends in the antero-posterior direction, its anterior end forming the *anterior pillars,* its posterior the *posterior pillars,* and its *body* the summit of the arch. It consists of lateral halves, but at the summit of the arch the two are joined together to form the body. The anterior pillars are separate from one another; they descend in front of the third ventricle to the base of the cerebrum, where they form the *corpora albicantia,* and then enter the substance of the optic thalamus. The posterior pillars are also separate; each curves downwards and outwards into the descending cornu of the ventricle, and forms the free border of the hippocampus major, which is named the *tænia hippocampi.*

The *velum interpositum* is a fold of pia mater which passes into the interior of the hemispheres through the great transverse fissure. It is triangular in shape, the base is in a line with the posterior end of the corpus callosum, the lateral margins are fringed by the choroid plexuses, and the apex, where the choroid plexuses blend with each other through the foramen of Monro, lies behind the anterior pillars of the fornix.

The *choroid plexuses* consist of highly vascular folds of membrane, and the epithelium of the ventricles is continued over their surface. These plexuses contain the small *choroidal arteries*, and supply the corpora striata, the optici thalami, and corpora quadrigemina, the blood from these bodies being returned by the *veins of Galen*. If the velum interpositum be raised from before backwards, the optic thalami, third ventricle, pineal gland, and corpora quadrigemina are exposed (*Fig.* 204).

The *third ventricle* is a cavity situated in the mesial plane, between the optici thalami; its roof is formed by the velum interpositum and the body of the fornix, its floor by the posterior perforated space (*pons Tarini*), the corpora albicantia, the tuber cinereum, infundibulum, and optic commissure; its anterior boundary by the anterior commissure and laminæ cinerea; its posterior boundary by the corpora quadrigemina and posterior commissure. The cavity of the ventricle is small, and it is crossed at its middle by the *middle* or *soft commissure*, which consists of grey matter and connects the two inner surfaces of the optici thalami together. If the anterior pillars of the fornix be separated, the *anterior white commissure* may be seen entering the *lenticular nuclei*. The white fibres of the *posterior commissure* pass across between the two optic thalami in front of the corpora quadrigemina.

BASAL GANGLIA.

§ 678. The ganglia of the base of the cerebrum are the corpora striata, the optici thalami, the corpora geniculata, the corpora quadrigemina, and the locus niger.

(1) *The corpora striatum* is situated in front and to the outer side of the optic thalami, and consists of two masses of grey matter, separated from each other by bands of medullated fibres, which pass from below upwards through its substance. The upper mass projects into the lateral ventricle, and is called the intra-ventricular portion or *caudate nucleus*.

The caudate nucleus consists of a club-shaped portion directed forwards, and a slender tail-like extremity directed backwards, the two together forming almost a complete ring, which encircles the optic thalamus and internal capsule, like a loop or surcingle. The body of the nucleus grows

smaller as it extends backwards in the upper part of the ventricle, and soon runs into the tail-like prolongation; when the latter reaches the posterior end of the optic thalamus it curves downwards into the inferior horn of the ventricle and runs forward to its anterior extremity, when it

FIG. 204.

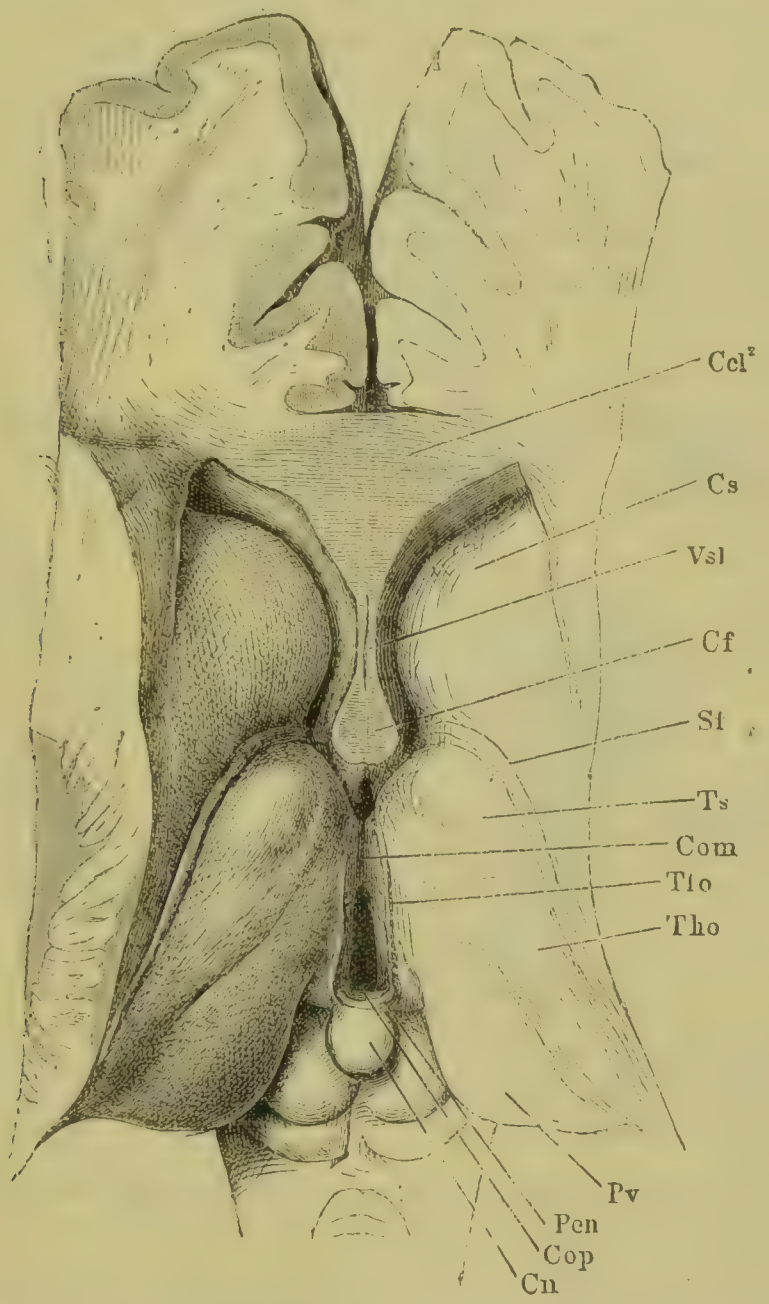


FIG. 204 (From Henle's Anatomie). *Basal Ganglia viewed from above.*

Ccl², Knee of the corpus callosum.
Cs, Corpus striatum.
Vsl, Ventricle of the septum lucidum.
Cf, Crura of the fornix.
Sf, Tænia semicircularis.
Ts, Anterior tubercle of the optic thalamus.
Com, Cop, The middle and posterior commissures respectively.
Tfo, Tænia thalami opt.
Pv, Pulvinar.
Tho, Optic thalamus.
Cn, Pineal gland.
Pcn, Peduncles of the pineal gland.

terminates in an enlarged extremity almost exactly opposite the point where it started in the anterior horn. The head of the caudate nucleus is continuous with the lenticular nucleus and with the grey matter of the anterior perforated space. The extremity of the surcingle, on the other hand, is connected with a deposit of grey matter forming the anterior wall of the inferior horn of the ventricle, named the *amygdala.* The tænia semicircularis accompanies the concave border of the surcingle, and runs forwards along the roof of the inferior horn of the ventricle to its anterior end, and there terminates in the amygdala (Dalton). In a vertical transverse section of the brain through the optic thalamus the superior portion of the surcingle is visible above the lenticular nucleus and internal capsule, while the inferior portion appears as an isolated mass of grey matter below the level of the lenticular nucleus and near the outer part of the inferior horn of the ventricle.

FIG. 205.

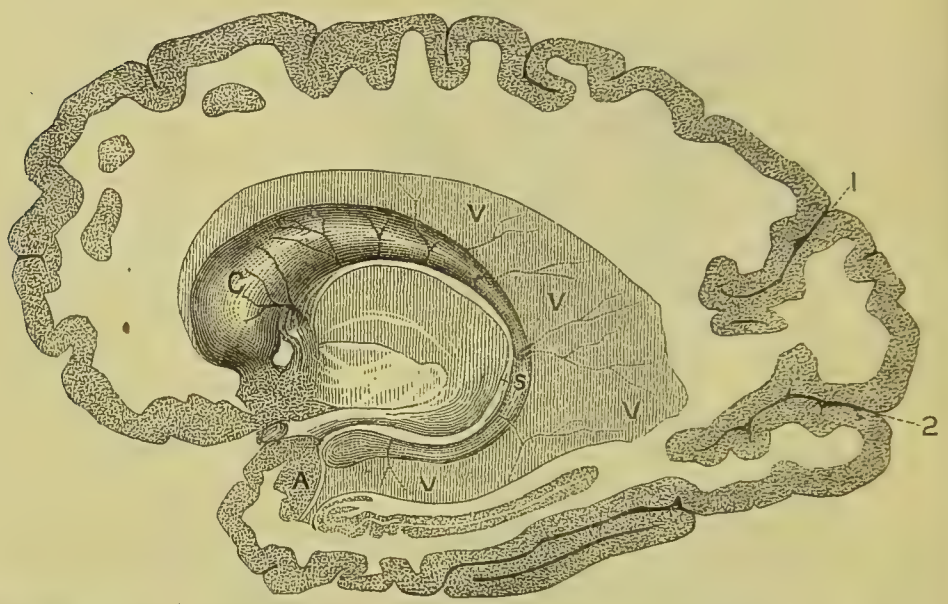


FIG. 205 (After Dalton). *Longitudinal and Vertical Section of the Right Hemisphere, showing the Cavity of the Lateral Ventricle and the Caudate Nucleus.*—C, Head of the caudate nucleus. S, Surcingle. V, Ventricle. A, Amygdala. 1, Parieto-occipital fissure. 2, Calcarine fissure.

The lower extra-ventricular portion, or *lenticular nucleus*, is separated from the intra-ventricular part by a layer of white substance named the *internal capsule*, while it is separated from the Island of Reil by a layer of white substance named the *external capsule*, and a grey lamina termed the *claustrum.* The lenticular nucleus, as its name implies, is of the form of a bi-convex lens on horizontal section, but on a vertical section through its middle it appears triangular, the apex being directed inwards. Two white bands which run parallel to the outer surface of the nucleus or the external capsule divide it into three zones named from within outwards the first, second, and third divisions of the lenticular nucleus.

(2) The *optic thalamus* is of an oval shape and rests on the crus cerebri of the same side. It is bounded externally by the corpus striatum and tænia semicircularis. The upper surface is free and is partly seen in the lateral ventricle, and is partly covered by the fornix, the former being called the *anterior tubercle* and the latter the *posterior tubercle* or *pulvinar*.

FIG. 206.

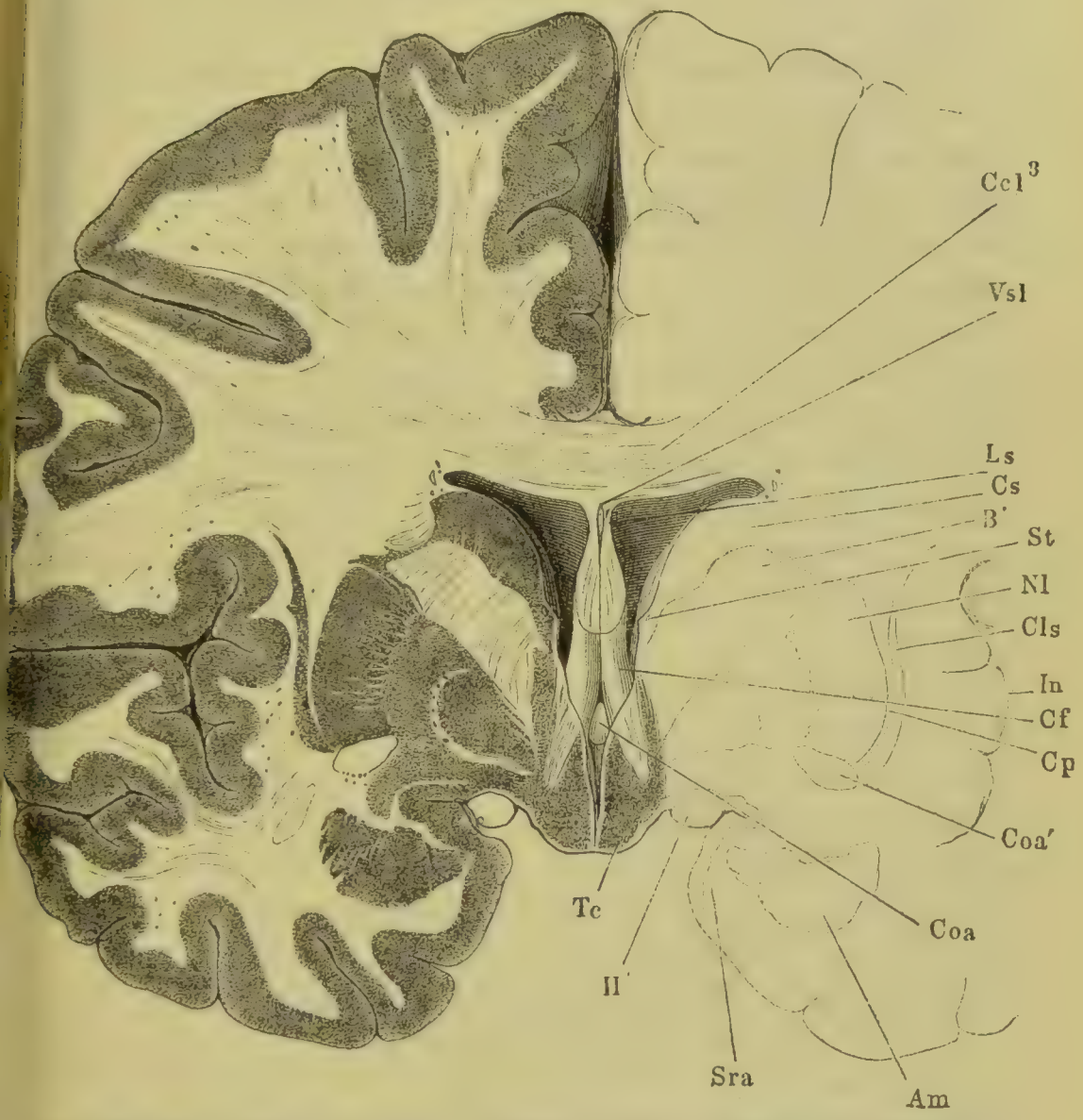


FIG. 206 (From Henle's Anatomie). *Vertical Section of the Brain immediately behind the Anterior Commissure of the Third Ventricle.*—Ccl^3, Corpus callosum; Vsl, The fifth ventricle; Ls, Lamina of the septum lucidum; Cs, Caudate nucleus; B', Internal capsule; St, Tænia semicircularis; Nl, Lenticular nucleus; Cls, Claustrum; In, Island of Reil; Cf, Interior pillars of the fornix; Cp, External capsule; Coa, Anterior commissure of the third ventricle; Coa', Anterior commissure as it winds back beneath the lenticular nucleus to reach the convolutions of the cortex; Am, Descending horn of the lateral ventricle; Sra, Substantia retic. alba.; II', Optic tract; Tc, Tuber cinereum.

The posterior surface is also free and projects into the descending cornu of the lateral ventricle. The inner surfaces of the two thalami form the lateral walls of the third ventricle, and are connected together by a transverse portion which forms the *middle* or *soft commissure* of the third ventricle. The inner surface is lined by grey matter which, according to Meynert, is distinct from that of the interior of the thalamus, and is probably the upward continuation of the central grey substance of the spinal cord.

The *internal capsule* consists of a thick band of medullated fibres, which separates the lenticular nucleus on the one hand from the caudate nucleus and optic thalamus on the other. On horizontal section the internal capsule is seen to consist of an anterior and posterior division, which form an obtuse angle with one another, the latter being called the *knee of the internal capsule.* The anterior division lies between the anterior and internal margin of the lenticular nucleus and the head of the caudate nucleus, and the posterior division between the posterior and internal margin of the lenticular nucleus and the optic thalamus; while the knee of the capsule is directed inwards towards the third ventricle, and forms by its projection a partial separation between the caudate nucleus and optic thalamus.

The *external capsule* consists, as already mentioned, of a thin band of white substance which bounds the lenticular nucleus externally and lies between it and the claustrum.

(3) The *corpora geniculata* consist of two small oblong and flattened eminences connected with the posterior extremity of the optic tract, named respectively *corpus geniculatum externum* and *internum.*

(4) The *locus niger* is a dark mass of grey matter which lies between the crust and tegmentum in the crus cerebri. It occupies nearly the whole diameter of the crus and extends from the anterior edge of the pons to the corpora albicantia.

The *pineal body* or *gland* is a reddish body, enveloped by the velum interpositum, and situated upon the anterior elevations of the corpora quadrigemina.

The *peduncles of the pineal body*, by means of which it is connected with the rest of the cerebrum, pass forwards, one on the inner side of each optic

thalamus, to join, along with the tænia semicircularis, the anterior pillar of the fornix of its own side.

(5) The *corpora quadrigemina* or *optic lobes* are situated behind and between the optici thalami, and rest upon the posterior surface of the crura cerebri. These bodies are divided into four eminences by a longitudinal and transverse fissure, the anterior pair being named *nates*, and the posterior *testes*. From each testis a white cord, the *superior peduncle of the cerebellum*, passes backwards to the cerebellum, while the *valve of Vieussens*, or *anterior medullary velum*, stretches between the pair of cerebellar peduncles.

The *aqueduct of Sylvius* is a narrow canal which passes beneath the corpora quadrigemina, and connects the third with the fourth ventricle. It is lined by a ciliated cylindrical epithelium.

DISTRIBUTION OF THE ARTERIES OF THE BRAIN.

§ 679. The arteries of the brain are derived from two great trunks—the vertebral and internal carotid arteries. The branches of the vertebrals and of the basilar trunk formed by their union supply the posterior and lesser portion of the brain, while the terminal branches of the internal carotid arteries supply the anterior and greater part of the brain. The branches distributed to the brain from the vertebral arteries may be called the posterior or vertebral, and those derived from the internal carotids the anterior or carotid arterial system.

The posterior cerebral arteries are the terminal branches of the basilar trunk. Each artery winds round the crus cerebri to reach the occipital lobe, and gives off a number of twigs—*the posterior median group* (*Fig.* 211, 2)—which pierce the posterior perforated space, and supply the internal surface of the optic thalamus, and the walls of the third ventricle.

Branches.—A choroid branch is given off to the velum interpositum, and small twigs pass into the substance of the crus cerebri as the vessel winds round it. A number of small branches, the *postero-lateral group* (*Fig.* 211, 4), enter the base of the brain behind the posterior border of the crus cerebri, and pass into the optic thalamus and corpora quadrigemina.

The *cortical branches* are three in number; the first, or anterior temporal artery, being distributed to the anterior part of the uncinate gyrus

and its vicinity ; the second, or posterior temporal artery, to the posterior part of the uncinate gyrus and the lower part of the temporo-sphenoidal lobe ; and the third, or occipital artery, to the inner and outer surfaces of the occipital lobe.

FIG. 207.

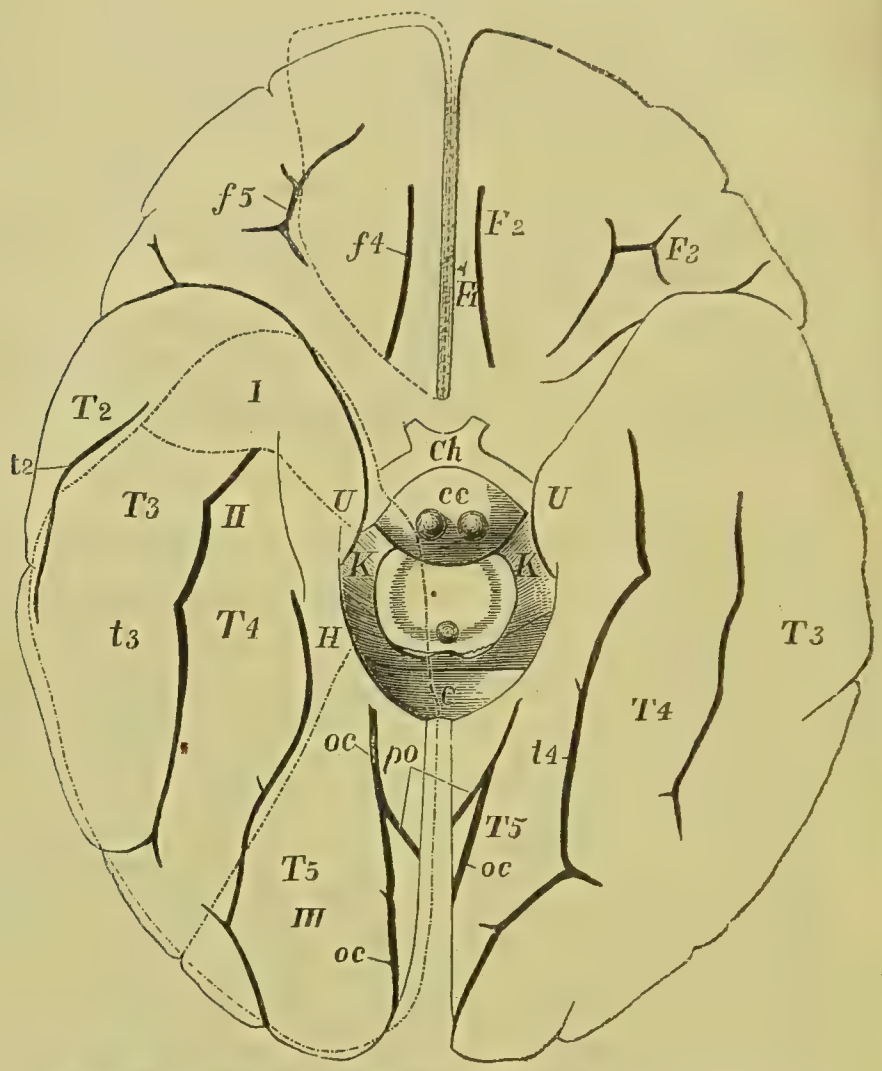


FIG. 207 (After Ecker and Duret). *View of the Brain from below.*

DISTRIBUTION OF VESSELS.

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

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

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

The *internal carotid artery* reaches the base of the brain close to the outer side of the optic commissure, and immediately breaks up into two branches—the anterior and middle cerebral arteries.

The *anterior cerebral artery* (*Fig.* 211, C A) runs forwards in the longitudinal fissure, and, turning round the corpus callosum, is distributed to the anterior part of the cerebrum. The arteries of the two sides are united at their commencement by a short transverse branch, the *anterior communicating artery*.

FIG. 208.

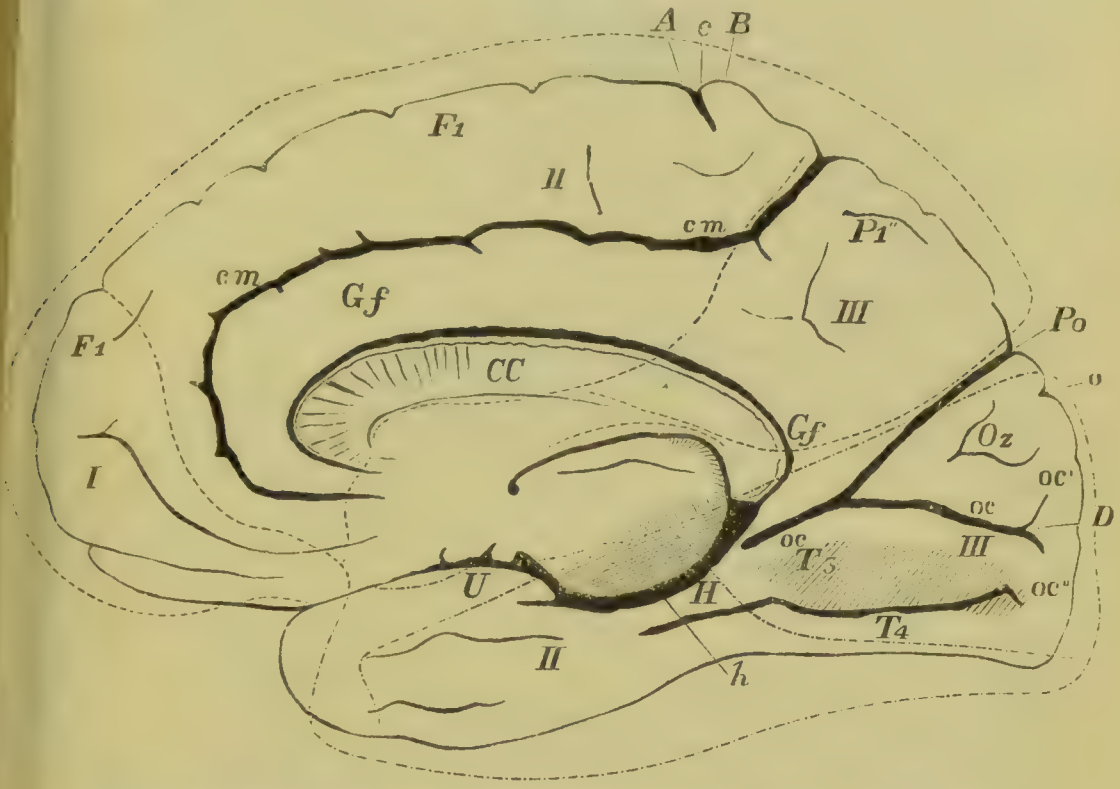


FIG. 208 (After Ecker and Duret). *Inner Surface of Right Hemisphere.*

DISTRIBUTION OF VESSELS.

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

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

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

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

Branches.—The *anterior median group* (*Fig.* 211, 1) are given off from the anterior communicating and the commencement of the anterior cerebral arteries; they supply the anterior part of the head of the caudate nucleus. The *cortical* branches are four in number—the first being distributed to the two internal orbital convolutions; the second to the anterior extremity of the marginal convolution, and to the superior and anterior portions of the middle frontal convolutions on the outer surface; the

third to the inner surface of the hemisphere as far as the extremity of the calloso-marginal fissure ; and the fourth to the quadrate lobule, the last supplying a branch to the corpus callosum.

The middle cerebral or Sylvian artery (*Figs.* 210, 211, S) runs in the fissure of Sylvius, and is the largest and most important branch of the internal carotid artery. It gives small branches—*the antero-lateral group* (*Fig.* 211, 3)—which pierce the anterior perforated space, and supply the corpus striatum and anterior part of the optic thalamus.

FIG. 209.

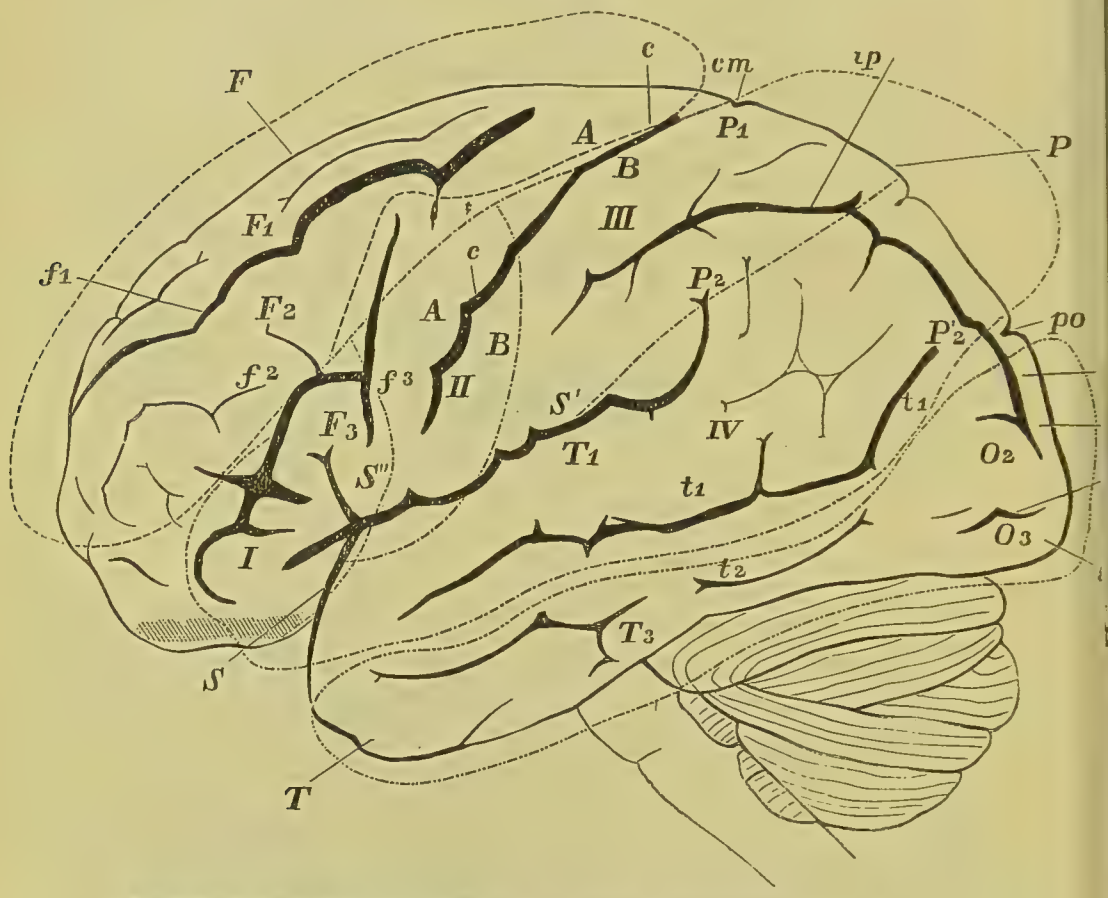


FIG. 209 (After Ecker and Duret). *Outer Surface of the Left Hemisphere.*

DISTRIBUTION OF VESSELS.

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

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

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

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

Branches.—A choroid branch is given off either by the middle cerebral or internal carotid arteries, which winds round the crus cerebri to reach the choroid plexus of the lateral ventricle. The main trunk divides into four branches. The first, or inferior frontal branch (*Fig.* 210, 1), is limited

FIG. 210.

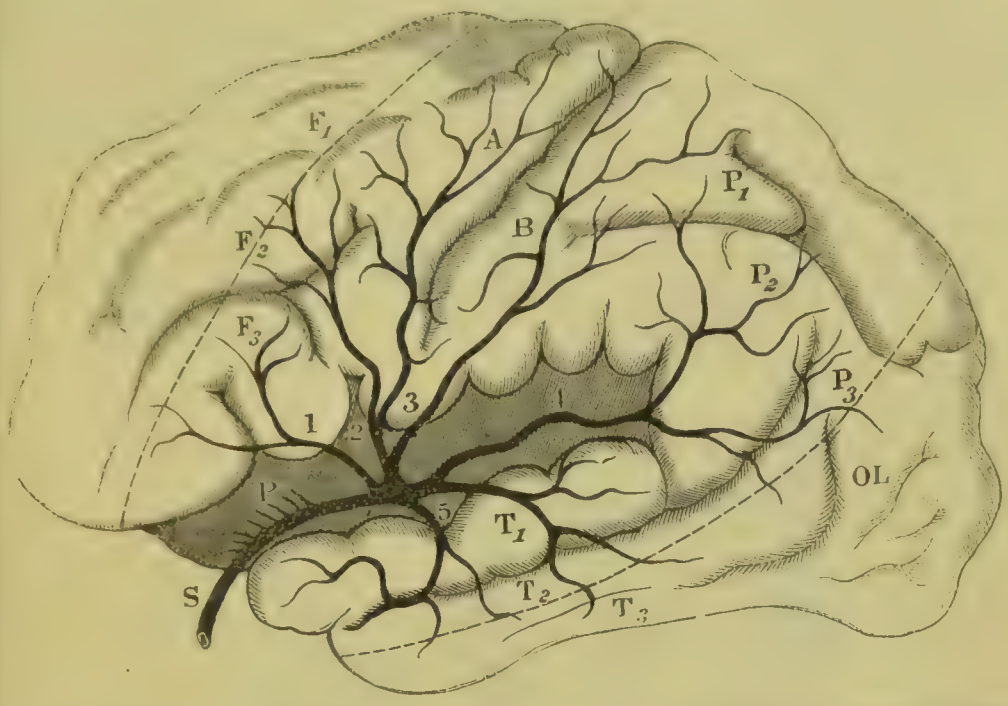


FIG. 210. *Diagram Showing the Area of Distribution of the Middle Cerebral Artery.* S, Sylvian or middle cerebral artery; P, Perforating branches; 1, Inferior frontal branch; 2, Ascending frontal branch; 3, Ascending parietal branch; 4 and 5, Parieto-sphenoidal and sphenoidal branches; A, Ascending frontal convolution; B, Ascending parietal convolution; F_1, F_2, F_3, First, second, and third frontal convolutions; P_1, P_2, P_3, First, second, and third parietal convolutions; T_1, T_2, T_3, First, second, and third temporo-sphenoidal convolutions; OL, Occipital lobe.

in its distribution to the outer part of the orbital surface and the adjacent inferior or third frontal convolution. The second, or *ascending frontal* branch (*Fig.* 210, 2), supplies the posterior part of the middle frontal and the chief part of the ascending frontal convolutions. The third, or *ascending parietal artery* (*Fig* 210, 3), passes into the fissure of Rolando, and supplies the rest of the ascending frontal and the ascending parietal convolutions as well as the anterior part of the superior parietal lobule. The fourth and fifth, or *parieto-sphenoidal and sphenoidal* branches (*Fig.* 210, 4 and 5), supply the inferior parietal lobule and the superior temporo-sphenoidal convolutions.

The posterior communicating artery is a long and slender vessel which connects the internal carotid with the posterior cerebral arteries.

The circle of Willis is formed by the union of the anterior and posterior arterial cerebral systems by means of the posterior communicating arteries. The free anastomosis which is thus formed enables the circulation of blood in the brain to be carried on when one of the main trunks is obstructed.

FIG. 211.

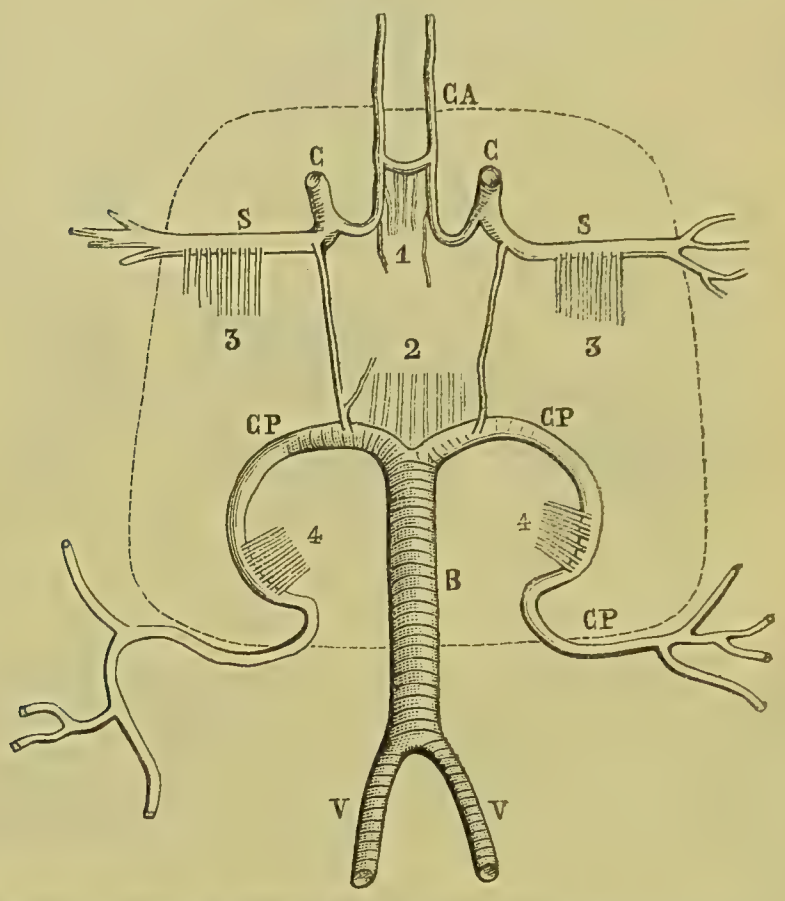


FIG. 211 (After Charcot). *Diagram of the Distribution of the Vessels at the base of the Cerebrum.*—CA, Anterior cerebral artery. S, S, Sylvian arteries. V, V, Vertebral arteries. B, Basilar. CP, CP, Posterior cerebral arteries. 1, 2, 3, 3, 4, 4, Groups of nutritive arteries. The line - - - - limits the ganglionic vascular area.

The following parts of the encephalon are situated within this vascular area: the optic commissure, laminæ cinerea, infundibulum and tuber cinereum, corpora albicantia, posterior perforated spot with part of the crura cerebri, and the origin of the third pair of nerves.

Cortical System of Arteries.—The arteries which supply the cortex of the brain ramify in the pia mater and are distributed to the grey matter of the convolutions and subjacent white matter. The terminal ramifica-

tions of the Sylvian artery may be taken as the type of the distribution of the cortical system of arteries. The main artery divides into the five secondary branches which have already been described, and each of these again subdivides into two or three tertiary branches. Each tertiary branch (*Fig.* 212, A) of the main artery subdivides into primary (*Fig.* 212, B), and secondary twigs (*Fig.* 212, C, C), and these form in the pia mater a vascular ramification from which the nutritive arteries of the brain are derived. Duret asserts that the tertiary branches of the main artery sometimes anastomose with similar branches of the neighbouring vascular territories, but the primary and secondary twigs of these branches do not anastomose amongst themselves.

FIG. 212.

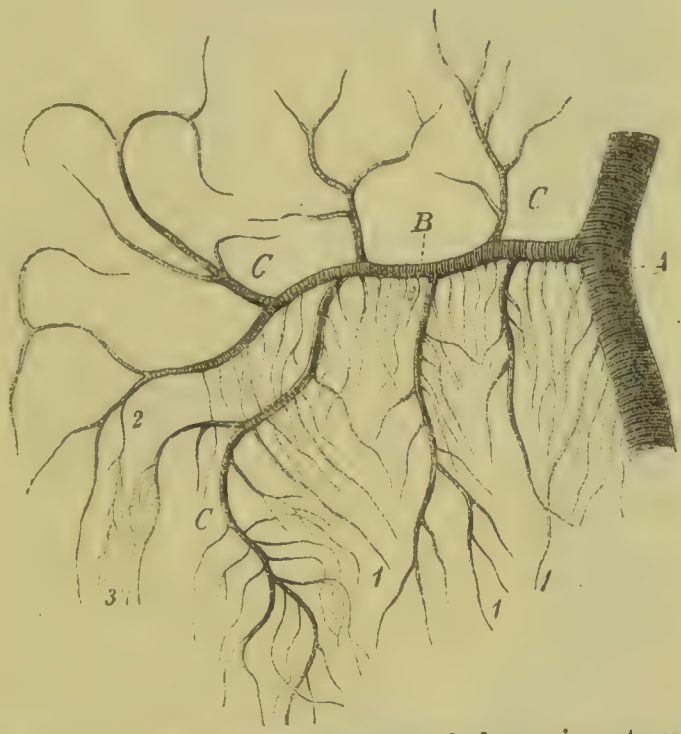


FIG. 212 (After Duret).—A, Tertiary branch of the main artery. B, Primary twigs. C, C, Secondary twigs. 2, 2, Cortical arteries. 3, Network of cortical arteries in the cerebral tissues.

Nutritive Arteries of the Brain.

The nutritive arteries are derived, not only from the extremities of the primary and secondary twigs, but a large number issue from the sides of these twigs, as well as from the sides of the tertiary branches of the main artery (*Fig.* 212, 1, 2). The nutritive arteries are of two kinds—(*a*) *the long or medullary*, and (*b*) *the short or cortical arteries.*

(*a*) The *medullary arteries* pass into the substance of the centrum ovale for a distance of three or four centimetres. They do not communicate with each other in their course except by fine capillaries, and consequently constitute so many small independent vascular territories. The terminations of these vessels approach the upward continuation of the gang-

lionic system of vessels, but the two systems do not appear to anastomose with one another. In a section of a convolution, twelve or fifteen medullary arteries may appear ; three or four of these pass into the free surface of the convolution (*Fig.* 213, 1), and pursue a vertical course ; those which enter the sides of the convolution pursue an oblique course through it, while those which pass into the bottom of the fissure again become vertical.

FIG. 213.

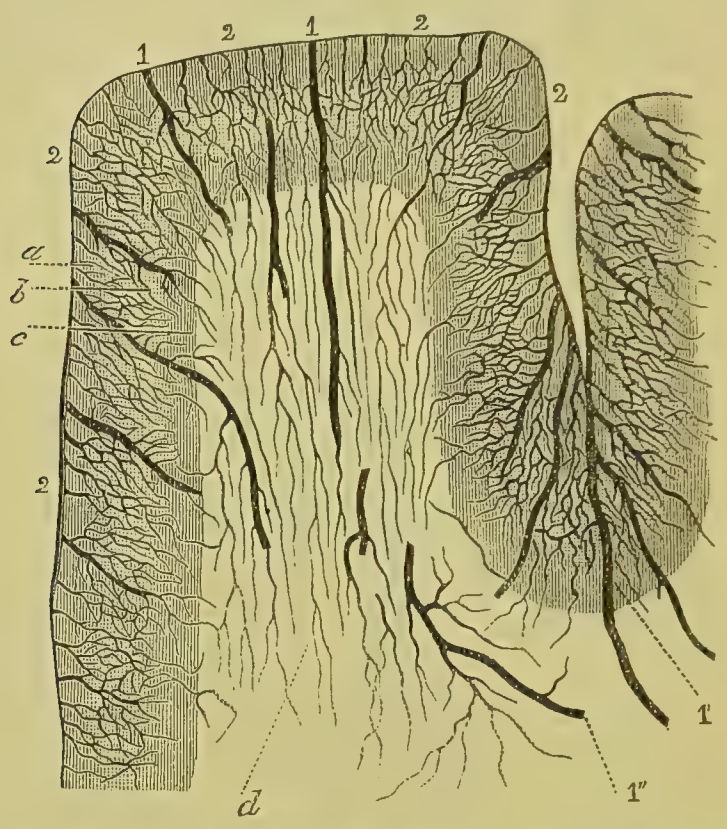


FIG. 213 (After Duret).—1, 1, Medullary arteries. 1′, Group of medullary arteries in the fissure between two neighbouring convolutions. 1″, Arteries of the system of arcuate fibres. 2, 2, 2, Arteries of the grey substance of the cortex. *a*, A large meshed capillary network situated under the pia mater. *b*, A smaller meshed capillary network situated in the middle layers of the cortex. *c*, Somewhat larger network in the internal layers adjoining the white substance. *d*, Capillary network of the white substance.

(*b*) The *cortical nutritive arteries* arise from the vascular network of the pia mater in the same way as the long arteries, but the former are thinner than the latter and pursue a shorter course. Some of these vessels pass through the whole thickness of the grey substance, and give small capillaries to the centrum ovale, while others terminate in the substance of the cortex. The vascular network in the convolutions possesses the following characteristics :—In the first layer, about one-half millimetre in thickness, the meshes of the network are large (*Fig.* 213, *a*); in

the second, corresponding to two layers of ganglionic cells, a very close and fine vascular network is formed (*Fig.* 213, *b*); in the third, corresponding to the internal layers of the cortex, a larger and coarser vascular network exists (*Fig.* 213, *c*); and in the fourth layer, or medullary substance, a still larger and coarser vascular network is observed.

The Central or Ganglionic System of Arteries.

These arteries consist of small branches which are given off from the trunks of the chief cerebral vessels; they pierce the base of the brain perpendicularly to reach the substance of the basal ganglia. These arteries form six main groups, which may be named the anterior and posterior median (*Fig.* 211, 1 and 2), the right and left antero-lateral (*Fig.* 211, 3, 3), and the right and left postero-lateral (*Fig.* 211, 4, 4) groups. An imaginary line passing round the circle of Willis, at a distance of two centimetres external to it, would completely surround all these vessels, and the area so limited may therefore be called the ganglionic vascular area (Charcot). All these vessels are *terminal arteries.* Some of these vessels are of sufficient importance, owing to their liability to rupture, as to deserve special description. The vessels derived from the middle cerebral artery—the antero-lateral group—after piercing the anterior perforated

FIG. 214.

FIG. 214 (From Duret). *Transverse Section of the Cerebral Hemispheres, about* 1 *cm. behind the Optic Commissure.*

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

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

space, ascend vertically to supply the corpus striatum, the internal capsule, and a portion of the optic thalamus. The following branches may be distinguished :—

(*a*) *Lenticular branches* consist of two or three small twigs, which ascend vertically and enter the substance of the lenticular nucleus, and are distributed to its two inner divisions and the adjoining portion of the caudate nucleus (*Fig.* 214, 5).

(*b*) The *lenticulo-striate branch* is much larger than either of the preceding arteries. It ascends along the external surface of the outer division of the lenticular nucleus, traverses the superior part of the internal capsule, and then passes from behind forwards into the substance of the caudate nucleus. It gives branches to the outer division of the lenticular nucleus, the internal capsules, and the caudate nucleus (*Fig.* 214, 4).

(*c*) The *lenticulo-optic branch* passes, like the lenticulo-striate artery, along the external surface of the outer division of the internal capsule, passes through the posterior part of the internal capsule, and terminates in the anterior and external part of the optic thalamus. The *anterior median* group of vessels derived from the anterior cerebral and anterior communicating arteries are small arteries ; they supply the anterior part of the caudate nucleus, and derive their chief importance from the fact that hæmorrhage from them may rupture into the ventricles and thus cause rapid death.

The *posterior cerebral artery* gives rise to two branches, which deserve special mention. (i.) The *posterior internal* artery of the optic thalamus, which is derived from the artery near its point of origin from the basilar, and is distributed to the internal surface of the optic thalamus. Hæmorrhage from this vessel often finds its way into the ventricular cavity. (ii.) The *posterior external* artery of the optic thalamus is derived from the posterior cerebral artery after it has wound round the peduncle. It passes upwards in the crus to the posterior part of the optic thalamus, where it terminates. It supplies the external geniculate bodies.

INTERNAL STRUCTURE OF THE CEREBRUM.

§ 680. The cerebrum is made up of (1) grey and (2) white matter.

(1) The *grey matter* is disposed in three great groups : (*a*) the grey matter which forms the central end of the cerebro-spinal tube ; (*b*) the grey matter of the basal ganglia ; (*c*) the grey matter of the cortex of the hemisphere.

(*a*) The *grey matter which forms the central end of the cerebro-spinal tube* has already been described up to the level of the opening of the aqueduct of Sylvius into the third ventricle, and the grey matter which surrounds the third ventricle

may be regarded as the upward continuation of the central tube. The grey matter of the third ventricle consists of a well-defined layer covering the inner wall of each optic thalamus and the masses situated at the base of the brain between and in front of the crura cerebri; viz., the posterior perforated space (pons Tarini), tuber cinereum, lamina cinerea, infundibulum, and part of the pituitary body.

(*b*) The *basal ganglia* consist of the locus niger, red nucleus of the tegmentum, corpora quadrigemina, the corpora geniculata, the optic thalami, and corpora striata.

The *locus niger* lies between the crus and tegmentum, and extends the whole diameter of the crus, and from the anterior edge of the pons to the corpora albicantia. It consists of nerve cells of various forms containing much dark pigment.

The *red nucleus of the tegmentum* is a round, reddish-grey centre, in structure somewhat similar to the olivary body of the medulla oblongata.

The *corpora quadrigemina* consist of grey and white matter, the former being continuous in front with the grey matter of the optic thalamus, and behind with that of the pons, and by means of the nucleus of the roof of the fourth ventricle with the corpus dentatum of the cerebellum.

The *Corpora Geniculata.*—The *external* geniculate body is densely filled with large branching and fusiform cells, and the fibres of the outer portion of the optic tract pass through it. The *internal* geniculate body contains numerous small nerve cells similar to those of the corpora quadrigemina.

The *optic thalamus* is composed of interlacing fibres mingled with grey matter. The nerve cells in the grey matter are both multipolar and fusiform.

The *middle* or *grey* commissure, connecting the two thalami, consists of small cells containing yellow pigment.

The *corpus striatum* is arranged in two chief masses, named respectively the caudate and lenticular nuclei. The caudate nucleus is connected below with the lamina cinerea, the anterior perforated space, and that part of the grey matter of the optic thalamus which is seen in the third ventricle. It contains large and small nerve cells, both possessing many branched processes.

No axis-cylinder process has been observed springing from the cells of the caudate nucleus.

The *lenticular nucleus* is continuous below with the caudate nucleus, and with the grey matter of the anterior perforated space. The two innermost zones contain numerous large branching nerve cells with yellow pigment. The cells are smaller in the outer division of the nucleus.

The *claustrum* is made up of fusiform and bipolar cells, somewhat resembling the cells of the vesicular column of Clarke on the one hand, and those of the fifth layer of the cortex on the other.

The *amygdaloid nucleus* is a small, round mass of grey matter, connected with the inferior part of the claustrum. It lies in front of the anterior extremity of the descending horn of the lateral ventricle, and is composed of fusiform cells similar to those of the claustrum.

(*c*) *The Grey Matter of the Cortex.*—When a convolution is divided vertically the grey matter is seen to be confined to the surface and to enclose a white core. The cortical substance consists of cells and fibres embedded in a matrix similar to the neuroglia of the spinal cord.

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

Layers of the Cortex.—The cortex of the cerebrum is divided into several layers, each of which possesses a definite histological character. The most commonly distributed form of structure is what Meynert has called the "five laminated type." The external layer consists of neuroglia and a layer of delicate

nerve tubes, along with a few scattered small nerve cells which are destitute of processes. The next layer is composed of small angular or pyramidal nerve cells with branching processes. The third layer contains large and small pyramidal cells with branching processes, arranged with their pointed extremities towards the surface of the convolutions, and separated into groups by bundles of the radiating fibres. In the innermost portion of this layer the pyramidal cells are larger than in the remaining portions, and it has therefore been described as a separate layer by Dr. Lockhart Clarke. In the cortex of the occipital lobe the deeper cells of the third layer are pyramidal in form, with their bases turned inwards towards the medullary substance, but their basal processes are directed laterally so as to connect adjacent cells, and none of them appear to be directed inwards to connect the cells with the fibres of the medullary substance. In the anterior portion of the frontal convolutions the disposition of these cells is somewhat similar, but a distinct basal process has occasionally been observed, which is directed towards the

FIG. 215.

FIG. 215 (After Meynert). *Transparent Section of a Furrow of the Third Cerebral Convolution of Man. Magnified* 100 *decimeters.*—1, Layer of the scattered small cortical corpuscles; 2, Layer of close-set, small pyramidal corpuscles; 3, Layer of large pyramidal cortical corpuscles (formation of the cornu Ammonis); 4, Layer of small, close-set, irregular-shaped cortical corpuscles (granule-like formation); 5, Layer of fusiform cortical corpuscles (claustral formation); *m*, the medullary lamina.

medullary substance of the convolution, and which afterwards becomes continuous with one of the fibres of the centrum ovale.

In the central convolutions of the brain Betz and Meziejewski have discovered cells which are two or three times the size of the pyramidal cells of the other regions of the cortex, and they have consequently named them *giant-cells.* In

FIG. 216.

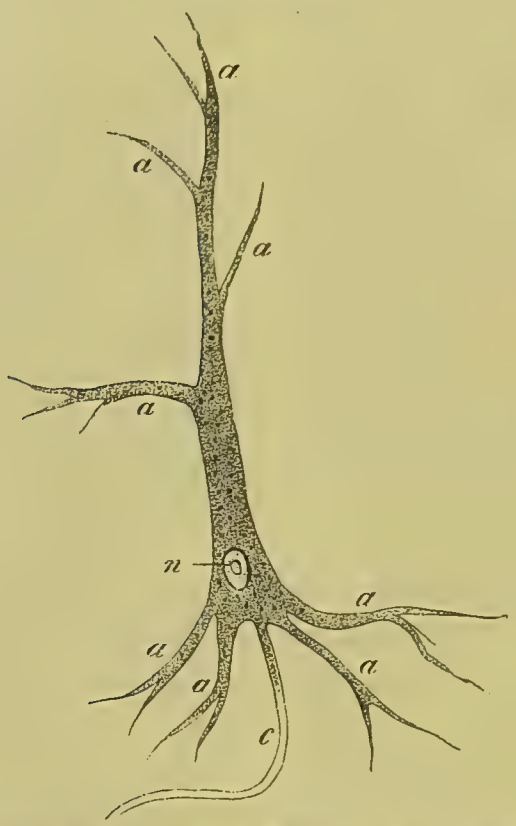


FIG. 216. *Pyramidal Giant-Cell.* — *n*, Nucleus; *a*, *a*, *a*, Branched processes; *c*, Unbranched basal process.

addition to the branched protoplasmic processes (*Fig.* 216, *a*, *a*) which connect neighbouring cells with one another, these cells possess a distinct axis-cylinder process (*Fig.* 216, *c*). The latter is always unbranched, and after becoming surrounded by a medullary sheath it forms the axis cylinder of a nerve fibre of the centrum ovale. Giant-cells have been observed in the paracentral lobule and in a portion of the postero-parietal, as well as in the ascending frontal and parietal convolutions, and posterior extremities of the three frontal gyri. These cells are disposed

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

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

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

(2) The *white matter* of the cerebrum consists of (*a*) transverse or commissural fibres; (*b*) longitudinal or collateral fibres; and (*c*) ascending or peduncular fibres.

(*a*) The *transverse* or *commissural fibres* consist of the following:—

(i.) The *transverse fibres of the corpus callosum* pass transversely from one side to the other, and connect corresponding convolutions in the hemispheres. These fibres lie on a plane superior to those of the corona radiata, and consequently the two systems of fibres interlace on their way to the convolutions.

(ii.) The *fibres of the anterior commissure* wind backwards through the lenticular nuclei to reach the convolutions around the Sylvian fissure.

(iii.) The *fibres of the posterior commissure* run through the optic thalami.

(*b*) The *longitudinal* or *collateral* system of fibres are the following:—

(i.) *Arcuate fibres* or *fibræ propriæ*, which are situated immediately beneath the inner surface of the cortex, and connect together the grey matter of adjacent convolutions.

(ii.) *Fibres of the gyrus fornicatus* take a longitudinal course immediately above the corpus callosum and form the white matter of that convolution. In front they bend round the corpus callosum, and become connected with the anterior perforated space. Behind they turn round the back of the same body, and are said to pass forwards to reach the anterior perforated space, so that these fibres completely surround the corpus callosum. Offsets from these fibres pass upwards and backwards to reach the summits of the secondary convolutions derived from the gyrus fornicatus near the longitudinal fissure.

(iii.) *Longitudinal septal fibres* lie on the inner surface of the septum lucidum and extend into the gyrus fornicatus.

(iv.) The *fasciculus uncinatus* passes across the bottom of the Sylvian fissure, and connects the convolutions of the frontal and temporo-sphenoidal lobes.

(v.) The *longitudinal inferior fasciculus* connects the convolutions of the occipital with those of the temporal lobe.

(vi.) The *longitudinal fibres of the corpus callosum (nerves of Lancisi)* connect the anterior and posterior ends of the callosal convolution.

(*c*) *The Ascending or Peduncular Fibres.*—The fibres which connect the central grey tube with the encephalon have already been traced as far as the crura. The upward continuation of the fibres of the anterior root-zones of the cord terminate in the optic thalamus. The posterior longitudinal fasciculus lies in front of the nucleus of origin of the third nerve, and when the aqueduct of Sylvius opens into the third ventricle, the fibres of the fasciculus bend outwards in the posterior commissure of the third ventricle to reach the inner wall of the optic thalamus, where they appear to terminate. Meynert describes these fibres as passing downwards and outwards to form part of the fillet of the crus cerebri, but examination of the crus in the embryo does not bear out this statement. The fibres of the posterior longitudinal fasciculus are medullated at an early period of embryonic life, but in a nine months embryo no medullated fibres having the course described by Meynert can be seen in the crus cerebri. The fibres of the posterior commissure, on the other hand, are the first fibres of the cerebrum to assume a medulla (Flechsig). The upward continuation of the external portion of the anterior root-zone of the cord lies in the crus cerebri to the outside of the third nerve and posterior longitudinal fasciculus, and the fibres of this area are continued upwards into the optic thalamus, where they form a thin

stratum of fibres which separates the grey matter which lines the third ventricle from the rest of the optic thalamus. A portion of the upward continuation of the external part of the anterior root-zone of the cord bends backwards in the pons to reach the corpora quadrigemina.

FIG. 217.

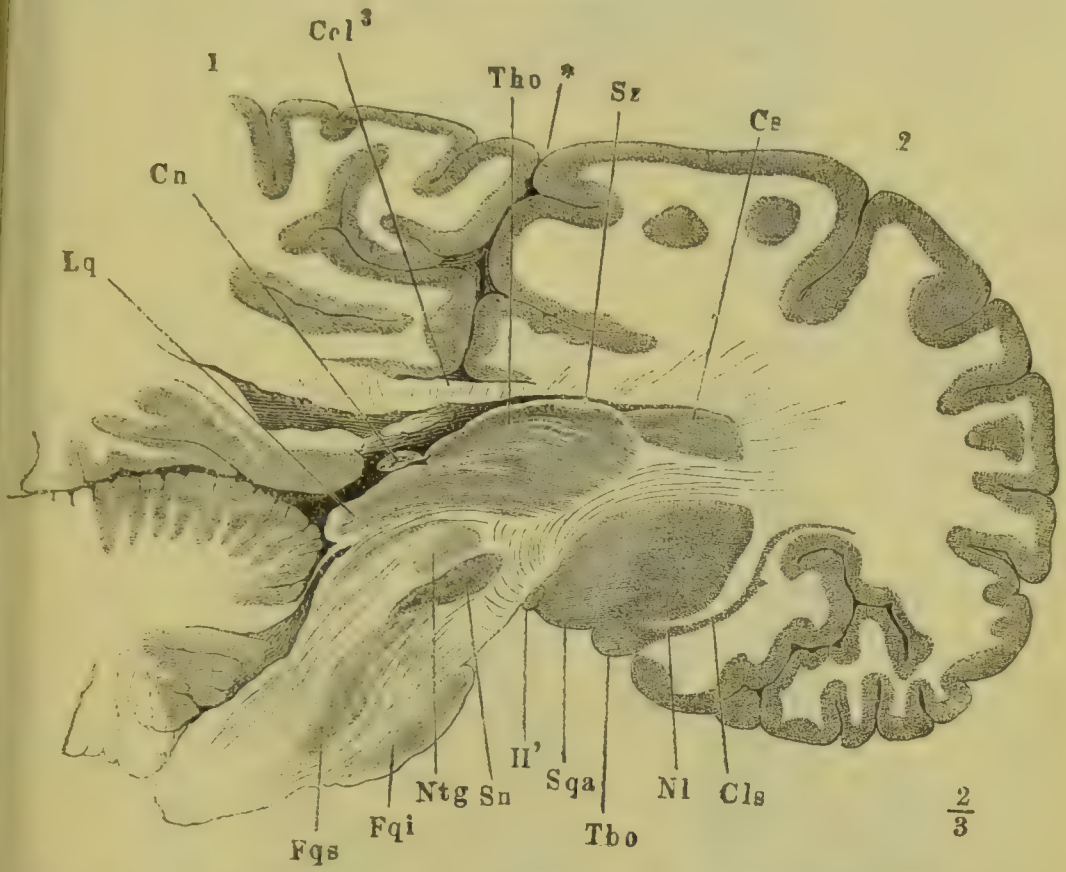


FIG. 217 (From Henle's Anatomie). *Vertical Section of the Brain parallel to the course of the Ascending Fibres of the Right Cerebral Peduncle.*—*, Great longitudinal fissure; 1, Left; and 2, Right hemisphere; Lq, Lamina quadrigemina; Cn, Pineal Gland; Ccl³, Corpus callosum; Tho, Thalamus: Sz, Striatum zonale of the thalamus; Cs, Caudate nucleus; Nl, Lenticular nucleus; Tbo, Tuber olfactorium; Cls, Claustrum; Sn, Locus niger; Ntg, Red nucleus of the tegmentum; Fqs and Fqi, Superior and inferior transverse fibres of the pons; II', Optic tract.

The corpora quadrigemina are connected with the optic thalami by nervous tracts, named *brachia*. The cerebellum is connected with the corpora quadrigemina by the superior peduncles. A large number of the fibres of the superior peduncles of the cerebellum decussate in the tegmentum, so that the fibres of the one side cross to become connected with the red nucleus of the opposite side. Some of these fibres

probably terminate in this nucleus, while others appear to pursue an uninterrupted course to the brain. The course of the fibres of the superior peduncles of the cerebellum is not well ascertained beyond the red nucleus. Some anatomists think that these fibres terminate in the optic thalamus, while others believe that they pass uninterruptedly as a thin stratum of fibres between the optic thalamus and the internal capsule, and through the corona radiata to reach the grey matter of the central convolutions.

FIG. 218.

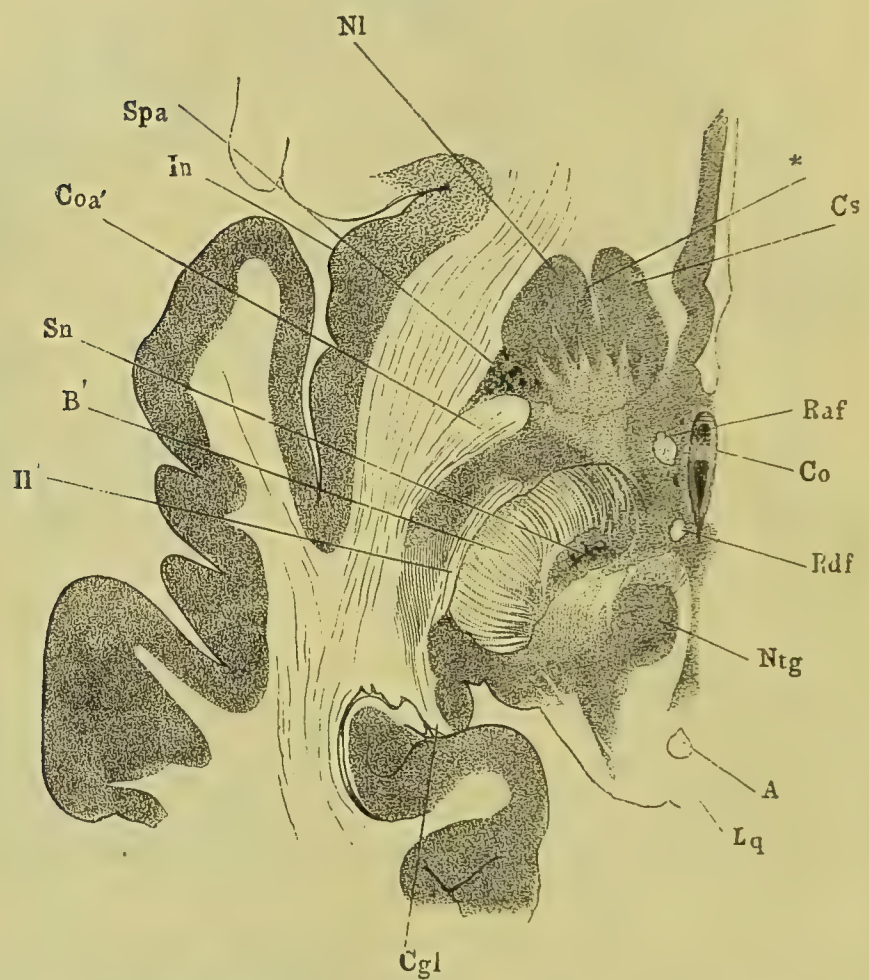


FIG. 218 (From Henle's Anatomie). *Horizontal Section of the Hemisphere of the Brain, close to its Inferior Surface.*—Lq, Lamina quadrigemina; A, Aqueduct of Sylvius; Ntg, Red nucleus of the tegmentum; Rdf, and Raf, Descending and ascending roots of the fornix; Co, Optic commissure, seen through the floor of the third ventricle; Cs, Caudate nucleus of the corpus striatum; Nl, The lenticular nucleus; *, Division between the two nuclei of the corpus striatum; Spa, Anterior perforated space; In, Island of Reil; Coa', Anterior commissure; Sn, Substantia nigra; B', Transverse section of the crusta; II', Optic tracts; Cgl, External geniculate body.

The Internal Capsule and Corona Radiata.—The crust of the cerebral peduncle consists of bundles of longitudinal fibres which have ascended mainly from the anterior pyramid of the medulla. The crust of the peduncle is, however, much larger

FIG. 219.

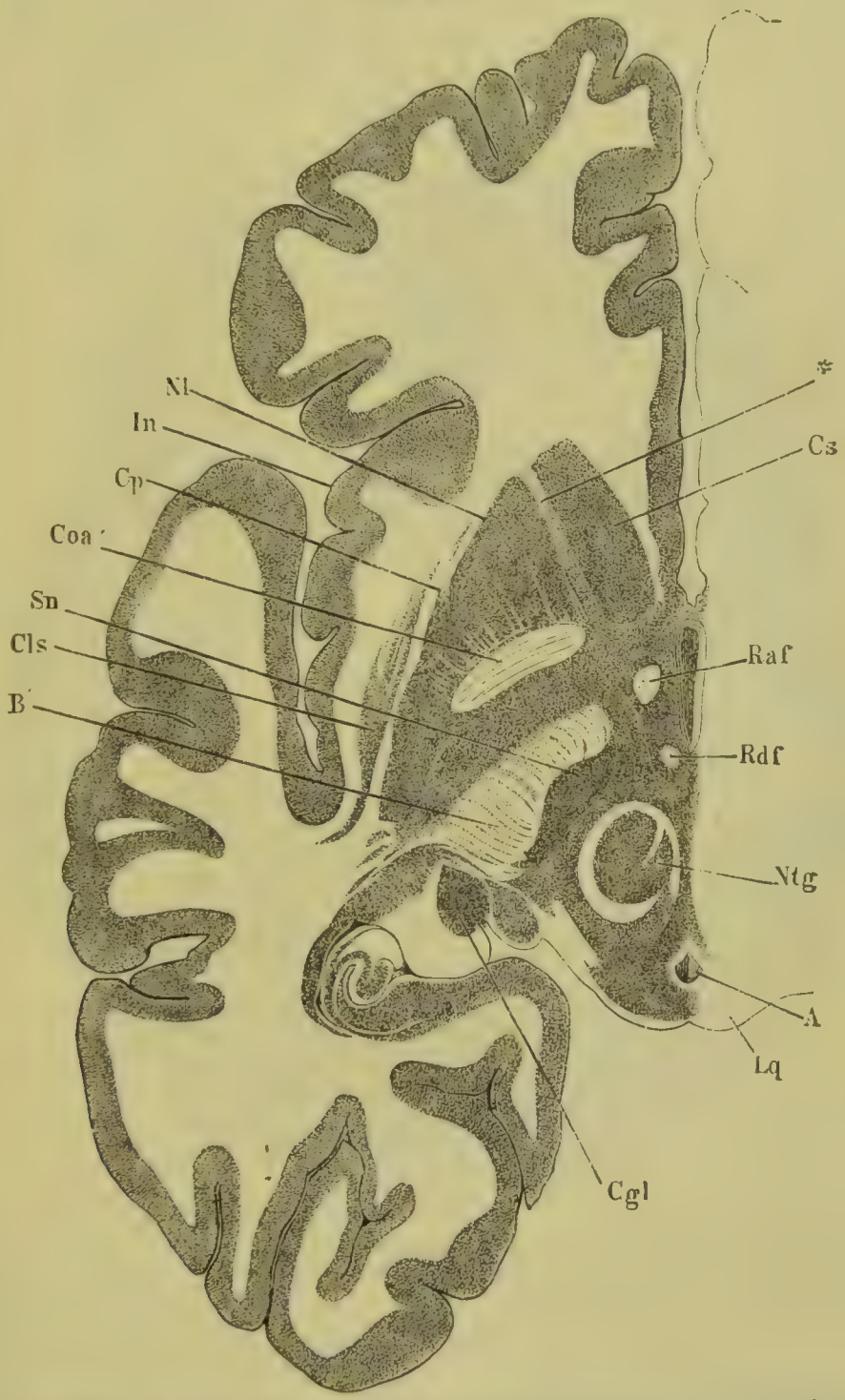


FIG. 219 (From Henle's Anatomie). *Transverse Section of the Hemisphere of the Brain, at a little higher elevation than Fig.* 218.—Cp, External capsule; Cls, Claustrum. The remaining letters indicate the same as *Fig.* 218.

than the anterior pyramid, hence the fibres of the latter must have been reinforced in their ascent through the pons. The crust of the peduncle is quadrilateral in form, but in ascending to the hemispheres it becomes flattened from above downwards, and from within outwards, and the fibres spread out like a fan, the edges of which are directed forwards and

FIG. 220.

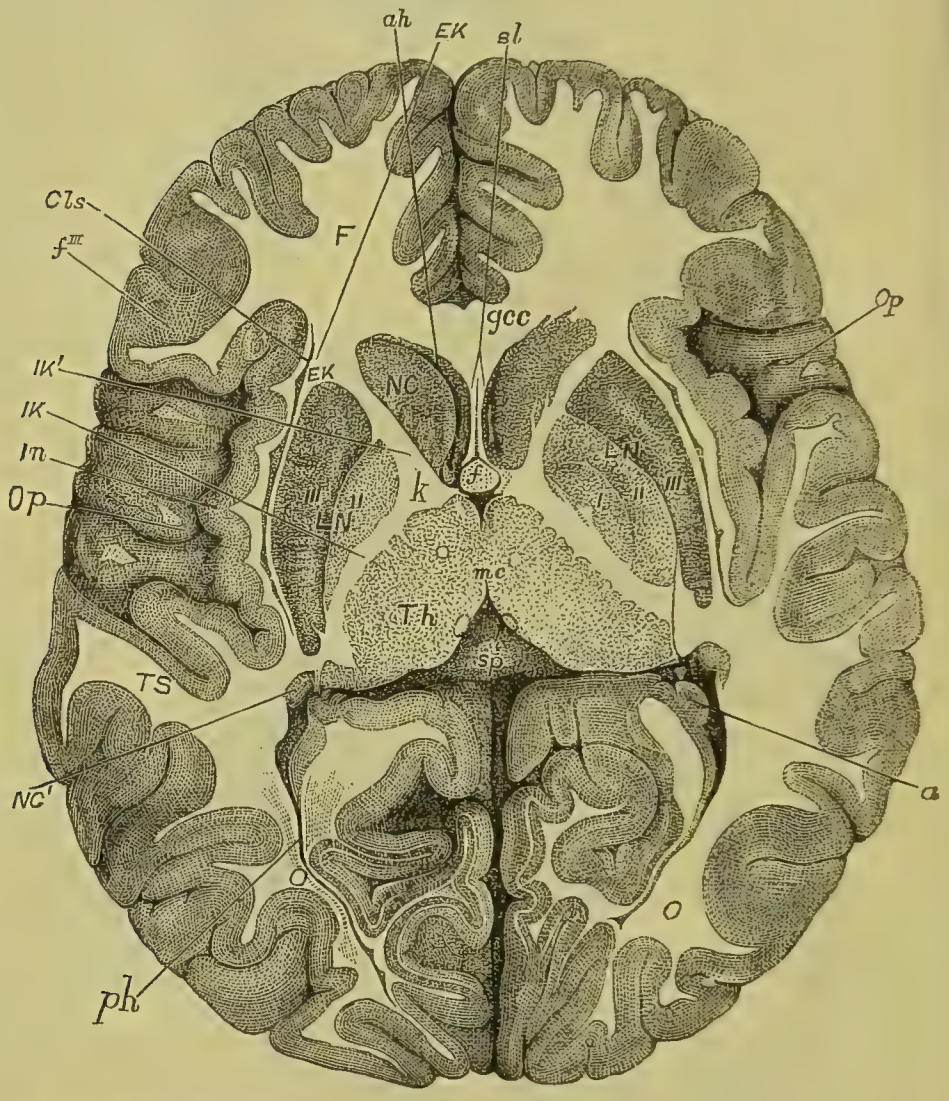


FIG. 220 (From Flechsig). *Horizontal Section of the Brain of a Child nine months of age, the right side being at a somewhat lower level than the left half.*—*F*, Frontal, *TS*, Temporo-sphenoidal, and *O*, Occipital lobes; *Op*, Operculum; *In*, Island of Reil; *Cls*, Claustrum; *f'''*, Third frontal convolution; *Th*, Optic thalamus; *NC*, Caudate nucleus; *NC'*, Tail of caudate nucleus; *LN*, Lenticular nucleus; *I*, *II*, *III*, First, second, and third divisions of the lenticular nucleus; *EK*, External capsule; *IK*, Posterior division, *IK'*, Anterior division, and *K*, Knee of the internal capsule; *ah*, *ph*, Anterior and posterior horns respectively of the lateral ventricles; *gcc*, Knee of the corpus callosum; *sp*, Splenium; *mc*, Middle commissure; *f*, Fornix; *sl*, Septum lucidum; *a*, Cornu Ammonis.

backwards. The fan formed by these fibres is bent into the form of an incomplete hollow cone, having its concave surface directed downwards and outwards, and its convex upwards and inwards. As the fibres ascend they pass at first between the optic thalamus and lenticular nucleus, but higher up they pursue their course beneath and to the outside of the thalamus and caudate nucleus, and over the lenticular nucleus. On horizontal section of the hemisphere, close to the inferior surface of the brain, the crusta is seen to be of an irregularly quadrilateral form, with its long axis directed from before backwards and from within outwards (*Fig.* 218, B′). At a higher level the crust, or what may now be regarded as the internal capsule, is of the same general form as in the preceding section, but its long axis is somewhat lengthened in proportion to its short axis (*Fig.* 219, B′). Still higher up the internal capsule has spread out from before backwards, while the anterior half forms an obtuse angle with the posterior. The angle where the halves meet is called the *knee* (*Fig.* 220, *K*), while the divisions themselves are called the *anterior* (*Fig.* 220, *IK′*) and *posterior segments* (*Fig.* 220, *IK*) of the internal capsule.

Corona Radiata.—On emerging from the basal ganglia the fibres of the internal capsule radiate in all directions to reach the cortex of the hemisphere, hence these have been described by Reil under the name of *corona radiata*, and the point at which the fibres emerge from between the ganglia is called the foot of the corona radiata.

The following fibres may be distinguished in the crusta and internal capsule:—

(1) The sensory peduncular tract and optic radiations of Gratiolet, the latter joining the internal capsule from the optic thalamus; (2) the pyramidal tract; (3) fibres in the crust connecting the central grey tube and the corpus striatum; (4) fibres issuing from the external surface of the optic thalamus to join the internal capsule; (5) fibres issuing from the external surface of the caudate nucleus; (6) fibres ascending from the superior and internal surface of the lenticular nucleus; (7) fibres already described ascending from the superior peduncle of the cerebellum; (8) fibres from the corpus callosum (Wernicke).

(1) *Sensory peduncular fibres and optic radiations of Gratiolet.*—The posterior root-zones and columns of Goll terminate, as we have already seen, in the triangular and clavate nuclei; and the connection between

these nuclei and the olivary body, and of the latter with the cerebellum, has already been sufficiently described. It has also been seen that the sensory fibres cross in the spinal cord, but Meynert describes a sensory crossing which takes place in the lower part of the medulla oblongata. According to this author, fibres issue from the nuclei of the cuneate and slender fasciculi which pursue an arcuate course round the central grey column, and become mixed with the fibres of the lateral column as they bend forwards to decussate. As already noticed, Flechsig thinks that these fibres curve round the olivary body of the same side, and enter its substance, while Meynert thinks that they form the outer fasciculus of the anterior pyramid of the medulla oblongata, and ascend with the latter up through the pons to reach the crus cerebri. Debove and Gombault describe an additional crossing of sensory fibres higher up in the medulla. These fibres pursue an arcuate course from the triangular and clavate nuclei, pass forwards to the outside of the olivary body, and then become subdivided into small

FIG. 221.

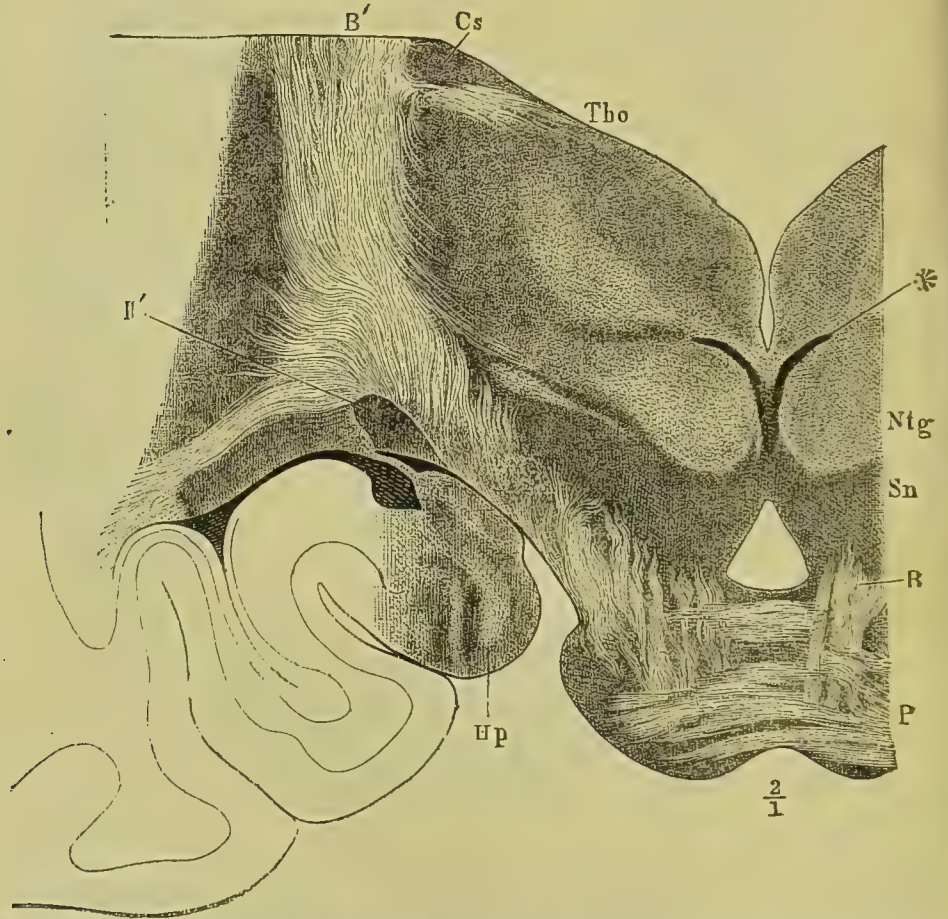


FIG. 221 (From Henle's Anatomie). *Transverse and Oblique Section of the Basal Ganglia slanting upwards and forwards from the anterior edge of the Pons* (P).—B, Crust of the crus cerebri; B′, Radiation of the peduncular fibres into the hemisphere; Sn, Locus niger; Ntg, Red nucleus of the tegmentum; *, Upper portion of the *formatio reticularis;* Tho, Thalamus opticus; Cs, Caudate nucleus; II′, Optic tract; Hp, Hippocampus.

asciculi, which penetrate into the posterior and external aspects of the nterior pyramid, and finally curve upwards, becoming mixed with the notor fibres. It is very probable that these sensory fibres occupy the posterior and external portion of the pyramidal tract in its ascent through the pons, inasmuch as bundles of fibres exist here which are not so distinctly medullated in a nine months human embryo as those lying in

FIG. 222.

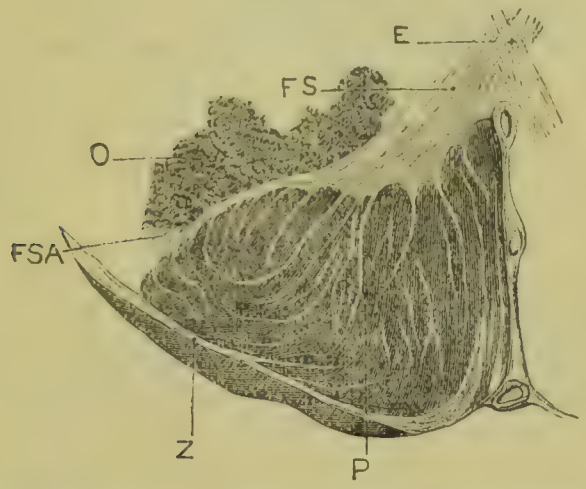


FIG. 222 (After Debove and Gombault) —*Section of the Anterior Pyramid* (P) *of the Medulla Oblongata, on a level with the middle part of the crossing of the Sensory Fibres.*—FS, Sensory fibres; FSA, Posterior and external sensory fasciculus which does not penetrate into the substance of the pyramid; E, Crossing of the sensory fibres; O, Nucleus of the pyramid; Z, Stratum zonale.

front of them. It has been at least ascertained that the sensory fibres occupy the external fourth of the crusta, and about the posterior third of the posterior segment of the internal capsule in their ascent towards the cortex of the brain. These fibres do not appear to be in any way connected with the optic thalamus and lenticular nucleus, but pass onwards between them to reach the cortex of the brain. In the posterior third of the posterior segment of the internal capsule the sensory fibres bend abruptly backwards, and then radiate to reach the convolutions of the occipital and temporo-sphenoidal lobes. The fibres of this tract are never medullated in an embryo of nine months, and can be readily traced upwards in the outer segment of the crusta and posterior segment of the internal capsule. In addition to the fibres which ascend from the spinal cord, medulla oblongata, and pons, the sensory tract in the internal capsule contains fibres which connect the first and second cerebral nerves with the cortex of the brain.

The *optic tracts* take origin in the basal ganglia by an internal, middle, and external root.

The *internal* root consists of a bundle of fibres which passes between the external geniculate body and outer edge of the crusta, and penetrates into the substance of the internal geniculate body, appearing to end in the anterior pair of corpora quadrigemina. Huguemin has recently main-

tained that this root is connected with the posterior pair of corpora quadrigemina, either directly or through the medium of the external geniculate body.

The *middle* root terminates in the external geniculate body.

The *external* root passes to the outside of the external geniculate body and penetrates the inferior peduncle of the optic thalamus about 12mm. in front of the posterior border of the pulvinar. By extirpating the eyeballs of young hares Gudden found that, when the animals were killed some months subsequently, the anterior pair of corpora quadrigemina, the optic thalami, and the external geniculate bodies were atrophied; while the posterior pair of corpora quadrigemina and the internal geniculate bodies were unaffected. In man, however, both the anterior and posterior pair of corpora quadrigemina have been found diminished in size in cases of long standing atrophy of the optic nerves.

These various roots of the optic nerves appear to be connected with the cortex of the brain by means of the fibres which have been named the *optic radiations of Gratiolet.* This bundle of fibres issues from the posterior and external border of the optic thalamus and is closely applied to the peduncular sensory tract in its passage through the internal capsule; these fibres radiate backwards and upwards to be connected with the convolutions of the occipital lobe.

The *olfactory lobe,* according to Meynert, divides in front of the anterior perforated space into an internal and external olfactory convolution. The external convolution coalesces with the temporal extremity of the gyrus fornicatus or the subiculum cornu ammonis. The internal convolution is continuous with the frontal end of the gyrus fornicatus, beneath which it may be recognised for some distance as a distinct longitudinal elevation.

A considerable portion of the white substance of the olfactory lobe traverses the corpus striatum until it meets the anterior commissure coming in the opposite direction. The olfactory fibres are supposed to cross in the anterior commissure, corresponding to the crossing of the fibres of the optic nerves in the chiasma. After crossing these fibres appear to ascend upwards and backwards and to join the fibres of the optic radiations of Gratiolet, and pass along with them to the convolutions of the cortex of the occipital or temporo-sphenoidal lobe. The posterior third of the posterior segment of the internal capsule, therefore, contains the peduncular sensory fibres and the fibres which connect the optic nerves, and the olfactory bulb with the cortex of the brain.

(2) *The Pyramidal Tract.*—The course of the fibres of the pyramidal tract has already been traced upwards through the spinal cord, medulla oblongata, and pons. It remains to trace the course of these fibres through the crusta, internal capsule, and corona radiata to their destination in the convolutions of the cortex. We have also found that the greater number of the fibres of the pyramidal tract in the cord are medullated in a nine months human embryo, while a large proportion of the fibres which join the tract in the medulla oblongata and pons are non-medullated. The ex-

ernal portion of the anterior pyramids of the medulla contains on the one hand medullated without any admixture of non-medullated fibres, the nternal and anterior margin of the pyramid on the other hand contains non-medullated without any admixture of medullated fibres, while an area lies between these in which the two kinds of fibres are mixed. The first

FIG. 223.

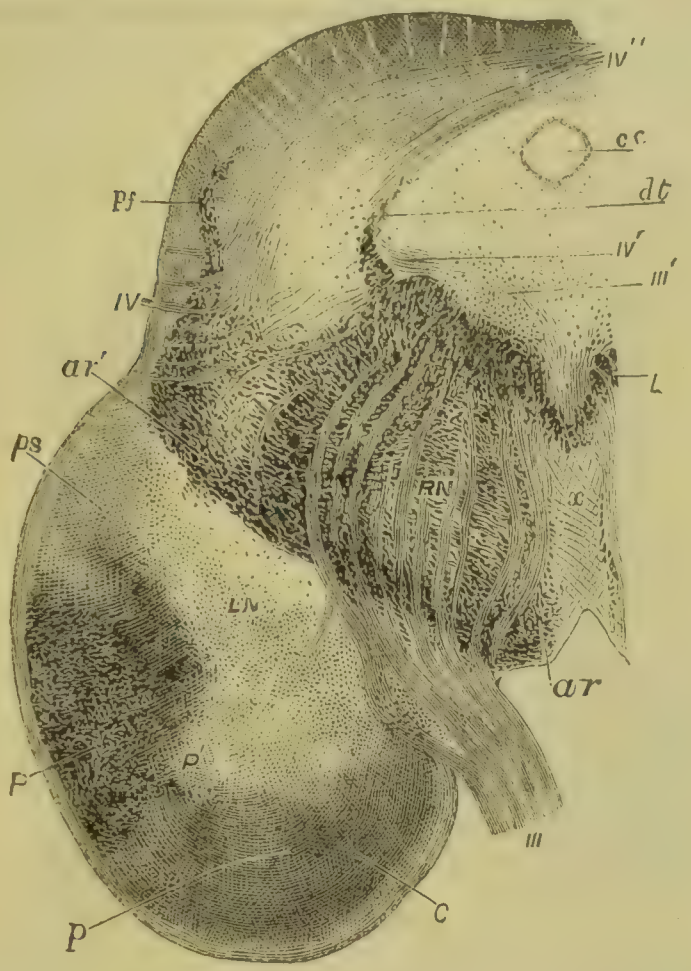


FIG. 223 (Modified from Krause). *Transverse Section of the Crus Cerebri on a level with the anterior pair of Corpora Quadrigemina, from a nine months embryo.*—*cc*, crusta; *P*, fundamental, *P'*, mixed, and *p*, accessory portion of the pyramidal tract; *LN*, locus niger; *RN*, red nucleus of the tegmentum; *L*, posterior longitudinal fasciculus; *ar* and *ar'*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord; III, third nerve; III', nucleus of the third nerve; IV, fourth nerve; IV', nucleus of the fourth nerve; IV'', crossing of the fibres of the fourth nerves to opposite sides; *dt*, descending root of the trigeminus; *cc*, aqueduct of Sylvius; *x*, crossing of the fibres of the superior peduncles of the cerebellum; *pf*, fasciculus of medullated fibres proceeding to the anterior pair of corpora quadrigemina.

of these regions may be called the *fundamental*, the second the *accessory*, and the third the *mixed area*. We have already seen that in the pons the accessory portion of the pyramidal tract lies internal to the fundamental portion, and in the crusta they occupy the same relative positions. The fundamental portion of the tract occupies the greater portion of the middle

third (*Fig.* 223, *P*), and the accessory portion the larger part of the internal third of the crusta (*Fig.* 223, *p*). The mixed area of the tract lies partly in the middle third of the crusta between the fundamental area and the locus niger, and winds round to the inside of the fundamental and between it and the accessory area (*Fig.* 223, *P'*). Speaking broadly, the fundamental fibres ascend in the middle (*Fig.* 224, P) and the mixed fibres in the anterior third of the posterior segment of the internal capsule (*Fig.* 224, P'), while the accessory fibres ascend in the anterior segment of the capsule (*Fig.* 224, p).

FIG. 224.

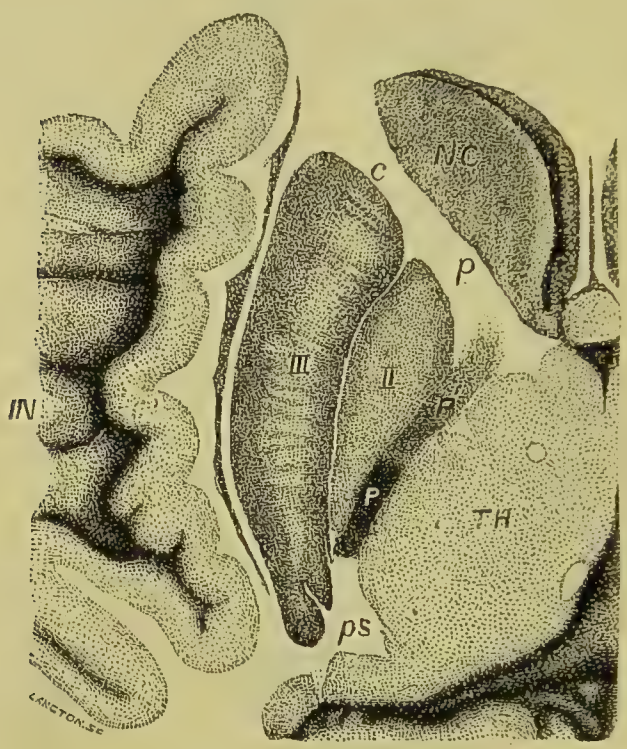


FIG. 224. *Horizontal Section of the Basal Ganglia and Internal Capsule of a Nine Months Embryo.*—LN, Lenticular nucleus; II, III, Second and third segments of the nucleus respectively; NC, Caudate nucleus; Th, Optic thalamus; In, Island of Reil; ps, Peduncular sensory tract and optic radiations of Gratiolet; P, Fundamental, P', Mixed, and p, Accessory portion of pyramidal tract; C, Fibres from the corpus callosum (?).

The fibres of the pyramidal tract, on emerging from between the basal ganglia, ascend in the corona radiata, and are distributed to the convolutions of the cortex in the following manner:—The fundamental fibres pass to the central convolutions near the margin of the great longitudinal fissure. These convolutions are, briefly, the parietal lobule, the paracentral lobule, the superior extremities of the ascending frontal and parietal convolutions, and probably also the posterior extremity of the first frontal convolution. These convolutions are, as we have already seen, those in which the largest pyramidal cells of the fourth layer of

the cortex have been found. The accessory fibres are distributed to the convolutions that constitute the operculum. These convolutions are the posterior extremity of the third frontal and the inferior extremities of the ascending frontal and parietal convolutions, and correspond to those in which the smaller-sized pyramidal cells with axis-cylinder processes have been observed. The mixed pyramidal tract is distributed to the convolutions between the two other areas. These convolutions are the posterior extremity of the second frontal and the middle of the ascending and parietal convolutions. What connection exists between the pyramidal tract and the supra-marginal and angular gyri has not been ascertained.

(3) *Fibres connecting the Central Grey Tube with the Corpus Striatum.*—The first and second divisions of the lenticular nucleus are connected with the crusta by a band of radiating fibres, which in their ascent are disposed in two thin bands, named the *striæ medullares*, and which run parallel to

FIG. 225.

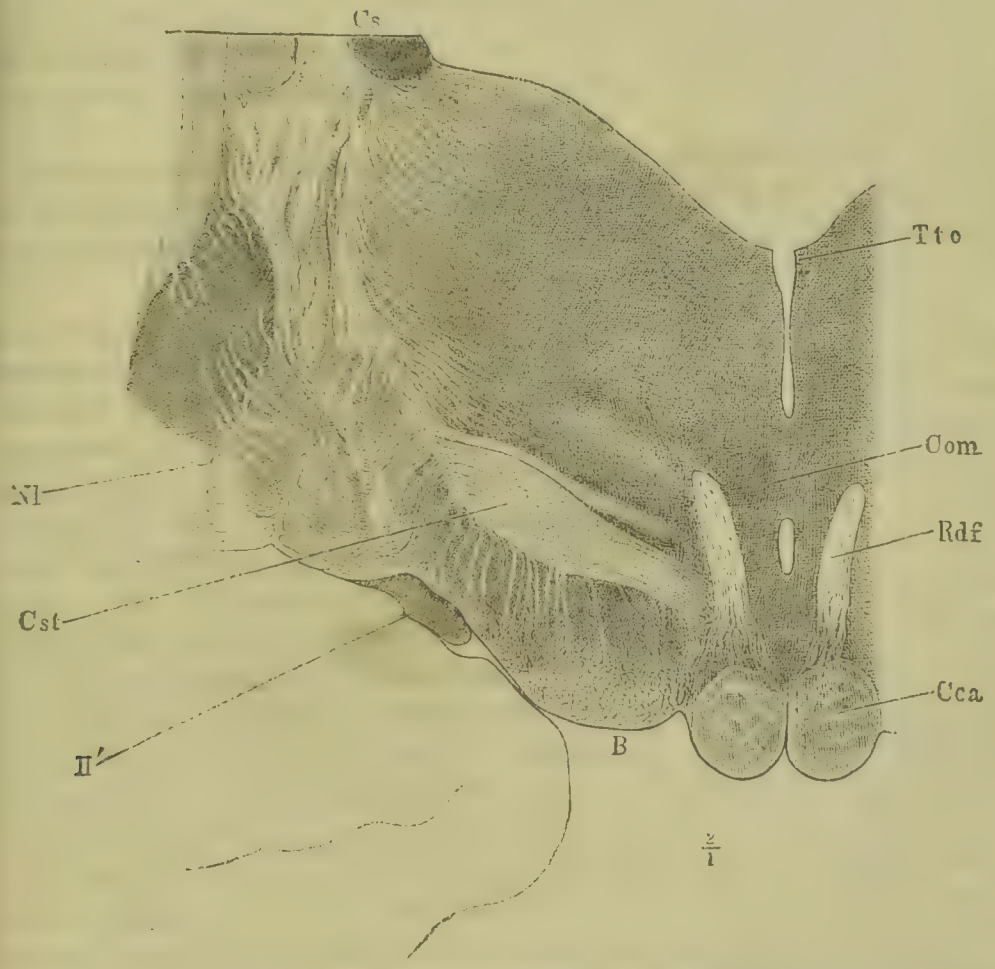


FIG. 225 (From Henle's Anatomie). *Transverse and Vertical Section of the Basal Ganglia on a level with the Corpora Caudicantia.*

Cca, Corpora albicantia.
Rdf, Descending roots of the fornix.
Com, Anterior commissure.
Tto, Tænia of the optic thalamus.
Cs, Caudate nucleus.
Nl, Lenticular nucleus.
Cst, Corpus subthalamicum.
II', Optic tracts.
B, Crust of the cerebral peduncle.

the outer surface of the nucleus, and divide it into three zones. Many of these fibres terminate in the substance of the nucleus, while others pass through it probably without interruption. A large number of these fibres at least pass transversely through the internal capsule, interlacing with its ascending fibres, and becoming connected with the optic thalamus and caudate nucleus. Fibres appear to enter the crusta from the locus niger, and it is not improbable that the latter nucleus is to be regarded as the means of communication between the anterior root-zone of the spinal cord and the corpus striatum.

(4) *Fibres issuing from the External Surface of the Optic Thalamus to join the Internal Capsule.*—The optic radiations of Gratiolet already described belong to this system of fibres, inasmuch as they issue from the external surface of the posterior portion of the thalamus. Other fibres issue from the external surface of the anterior two-thirds of the thalamus and join those of the pyramidal tract on their way to the cortex. The anterior radiating fibres of the thalamus are probably distributed to the convolutions of the frontal lobe, and the central radiating fibres to the convolutions of the parietal lobe, while as we have already seen the posterior radiating fibres are distributed to the convolutions of the occipital lobe.

(5) *Fibres issuing from the External Surface of the Caudate Nucleus.*—These fibres are described as issuing from the external surface of the caudate nucleus, and as passing into the corona radiata immediately above and internal to the radiating fibres of the optic thalamus.

(6) *Fibres issuing from the Superior and Internal Surface of the Lenticular Nucleus to join the Ascending Fibres of the Internal Capsule.*—A large number of fibres issue from the superior and internal surface of the lenticular nucleus, and pass transversely through the internal capsule, interlacing with its longitudinal fibres. Other fibres are described as pursuing an ascending course parallel with the longitudinal fibres of the internal capsule. The latter fibres are supposed to radiate in all directions on gaining the corona radiata to become connected with the cortex. It is right, however, to add that the latest anatomical researches throw considerable doubts upon the existence of the radiating fibres which anatomists have described as connecting the caudate nuclei and the third division of the lenticular nucleus with the cortex. Wernicke states that neither the caudate nucleus nor the third division of the lenticular nucleus are directly connected with the cortex by radiating fibres, and he thinks that they must be regarded as independent ganglia, like the grey matter of the cortex itself. The first and second divisions of the lenticular nucleus form ganglia of interruption, which connect the caudate nucleus and the third division of the lenticular nucleus with the central grey tube.

(7) *Fibres Ascending from the Superior Peduncle of the Cerebellum.*—The red nucleus of the tegmentum is connected, as already described, with the fibres ascending in the superior peduncle of the cerebellum of the opposite side. Fibres appear to ascend from the red nucleus to the

optic thalamus, and Flechsig supposes that some of the fibres of the superior peduncle of the cerebellum of the opposite side pass uninterruptedly through the red nucleus and along the internal surface of the fibres of the pyramidal tract to be distributed to the central convolutions of the cerebrum.

(8) *Fibres issuing from the Corpus Callosum and Descending into the Internal Capsule.*—Wernicke states that the fibres of the corpus callosum which form the anterior wall of the anterior horn of the lateral ventricle wind backwards along the external border of the caudate nucleus, where they become mixed with the longitudinal fibres of the internal capsule He is unable to trace them further.

(9) *Fibres of the External Capsule.*—The fibres of the external capsule either ascend from the crusta, pass along the inferior surface of the lenticular nucleus, and bend abruptly upwards round its inferior external angle to reach the external surface, or they take origin in the cells of the nucleus, and after issuing from its inferior surface pursue the course just described. These fibres ascend along the external surface of the lenticular nucleus forming the thin stratum of white matter between it and the claustrum (*Fig.* 220, *EK*), and on reaching the corona radiata they radiate to reach the convolutions of the cortex. The external surface of the lenticular nucleus and the external capsule are simply in contact with one another, and there appear to be no connections formed between the fibres of the one and the cells of the other. The two surfaces are, indeed, separated in some places by blood-vessels ascending from the middle cerebral artery.

Besides those of the internal capsule and corona radiata, other fibres connect the basal ganglia and the cortex of the brain. These fibres consist of the *fornix*, *tænia semicircularis*, *pedunculus septi*, and a considerable proportion of the fibres which constitute the *collar* or *fillet of the crus*.

The *fornix* arises in the optic thalamus. Its fibres of origin are connected with the *tænia semicircularis* and the peduncles of the pineal gland. They descend to the under surface of each thalamus, and after forming a loop in the corpora albicantia they ascend upwards and forwards in the walls of the third ventricle as the anterior pillars of the fornix. The fibres of each crus then pass backwards in the body of the fornix, and end as the tænia hippocampi in the gyrus of the same name.

The *tænia semicircularis* connects the apex of the temporal lobe with the whole length of the internal margin of the caudate nucleus. The fibres which penetrate into the anterior region of the head of that nucleus are named *striæ corneæ*.

The *pedunculus septi* connects the cortical substance of the septum lucidum with the basal mass of the corpus striatum.

The Collar or Fillet of the Crus Cerebri.—A bundle of fibres forms at the

posterior, inferior, and external angle of the optic thalamus, which winds downwards, outwards, and forwards round the posterior margin of the ascending fibres of the crusta. These fibres are named the *inferior peduncle* of the optic thalamus, and constitute the posterior portion of the collar or fillet of the crus; they spread out on the roof of the descending cornu of the lateral ventricle and pass forwards to the convolutions of the anterior extremity of the temporo-sphenoidal lobe. It is probable that some of them also radiate backwards to reach the convolutions on the inferior surface of the occipital lobe. Other fibres appear to issue from the anterior, inferior, and external angle of the thalamus, which wind round the anterior border of the crusta, and terminate in the lenticular nucleus, or pass to the convolutions of the temporo-sphenoidal lobe. These fibres form the anterior portion of the collar of the crus.

§ 681. *Development of the Brain.*—The cerebral end of the cerebro-spinal tube is at first uniform in appearance with the spinal part, but it soon expands into three vesicular dilatations—the *primary cerebral vesicles*. These vesicles are named, from their relative positions, anterior, middle, and posterior, and the structures which go to form the several sub-divisions of the encephalon are produced in their walls.

The *posterior cerebral* vesicle first bends forwards to form the medulla oblongata, and then backwards to form the cerebellum, the pons being developed at the angle where these two parts are continuous with one another. The cerebellum consists at first of a central lobe, and the lateral lobes are only developed in the mammalia.

The *middle cerebral* vesicle bends forwards from the posterior one, its central hollow becoming the aqueduct of Sylvius; the *optic lobes* are formed in its roof, and the *crura cerebri* in its floor.

The *anterior cerebral* vesicle bends downwards from the middle vesicle, and its central hollow becomes the third ventricle. The optic thalami form in its lateral walls, and the pineal body in its upper and posterior wall. The lamina cinerea closes the vesicle in front. The posterior part of the anterior vesicle gives off from each side a flask-shaped prolongation—the *primary optic* vesicle—which subsequently forms the optic tract with the optic nerve and retina.

The antero-lateral part of the cerebral vesicle is prolonged forwards into two hollow processes, the *hemisphere-vesicles*, from which the cerebral hemispheres are subsequently developed. These vesicles are separated from one another by a *median longitudinal fissure*, whilst the hollow in the interior of each forms the *lateral ventricle*. On the floor of this vesicle a grey mass forms which may be named the *basal nucleus*, and which subsequently develops into the corpus striatum. The remaining portions of the walls of the vesicle form the cortex of the brain, the basal nucleus and cortex being continuous in the part which subsequently forms the anterior perforated space. When after a time fibres shoot down from the cortex to reach the central grey tube, and shoot upwards

from the central grey tube, corpora quadrigemina, and optic thalamus to reach the cortex, they pursue the shortest course by passing through the *basal nucleus*, so that the latter becomes divided into an inferior and external (the lenticular), and a superior and internal portion (the caudate nucleus), the two being continuous with one another and with the cortex of the cerebrum in the anterior perforated space. The development of the *basal nucleus* therefore renders it probable that the corpus striatum is a modified portion of the cortex of the brain, thus confirming the view recently adopted by Wernicke. The fibres of which the fornix consists now appear on the inner wall of the hemisphere-vesicle, while the transverse fibres of the *corpus callosum* pass above the plane of the fornix to connect the cortex of one hemisphere with that of the other. Between the corpus callosum and the upper surface of the fornix, anteriorly, two thin layers of grey matter belonging to the inner surface of each *hemisphere-vesicle* are enclosed. These together form the laminæ of the septum lucidum, and the cavity which separates them becomes the fifth ventricle. Each hemisphere-vesicle gives off from its anterior part a hollow process which develops into the *olfactory bulb*.

The longitudinal or collateral system of fibres, which connects the occipital lobe on the one hand and the temporal and frontal lobes on the other, form a relatively thick white layer in their passage through the middle lobe, which cuts off the fifth from the remaining layers of the cortex of the Island of Reil, the detached portion being known as the *claustrum*. It consists, as we have seen, of fusiform cells analogous to those found in the fifth layer in other areas of the cortex, and which are probably associated in the latter with the system of arcuate fibres.

The Convolutions.—The walls of the cerebral hemispheres consist at first of two smooth shell-like lamellæ which include the cavities afterwards named the lateral ventricles. The first traces of the convolutions appear about the fourth month, the primary *sulci* appearing as slight depressions on the smooth surface. The Sylvian fissure begins as a cleft between the anterior and middle lobes about the fourth month, and is the first fissure to make its appearance after the great longitudinal fissure. Soon afterwards the fissure of Rolando appears; it is followed by the parieto-occipital, and at a somewhat later period by the calloso-marginal fissure. After the fifth month, the secondary fissures develop rapidly, and all the convolutions and fissures make their appearance towards the seventh and eighth months. The hemispheres do not cover the optic thalami until the third month, at the fourth they reach the corpora quadrigemina, and at the sixth month they cover a great part of the cerebellum.

The convolutions of the human brain are divided into *primary or fundamental* and *secondary or accessory*. The disposition of the fundamental convolutions is fixed, and corresponds closely with the arrangement of the convolutions in the brain of the monkey; but the disposition of the accessory convolutions is variable, and they must be regarded as being superadded to the former in the course of evolution. The arrangement of the

convolutions of a human embryo at the sixth month corresponds closely to that of the brain of the adult monkey.

The fundamental convolutions are distributed along the margin of the great longitudinal and other primary fissures, while the accessory convolutions border the secondary fissures.

The grey matter of the accessory is connected with that of the fundamental convolutions by means of arcuate fibres, while the former is not directly connected with the ascending and radiating fibres of the internal capsule.

Another circumstance worth observing is that the grey matter of the summits of the convolutions is developed before that at the bottom of the fissures (Broadbent). It must be remembered that the growth of the brain is restrained at an early period of embryonic life by the skull. The distribution of the blood-vessels to the brain is such that the surface of the hemisphere must be more freely supplied with blood than the medullary substance, and consequently the former will grow at a more rapid rate than the latter. If then the cortex grow at a more rapid rate than the medullary substance, and be at the same time restrained from growing freely outwards by the skull, the surface of the organ must be thrown into folds. The young nerve cells and fibres grow in the neighbourhood of the vessels and the older ones are thrust away from them. It follows that when two vessels run a more or less parallel course on the surface of the brain the younger nerve cells and fibres lie near each vessel, while the older ones occupy a position midway between them. If the surface of the brain were free to grow in all directions like the skin, the vessels themselves would be thrust further apart during growth and the surface would remain more or less smooth; but as the surface of the brain grows under pressure, the vessels cannot be thrust from each other at a sufficiently rapid rate to keep progress with the growth of nerve tissue between them. The consequence is that either the growth of the nerve tissue between the two vessels must be arrested, or a fold must be formed so that the grey matter between them may pursue a curved instead of a straight course. If this be an approximately accurate account of the way in which the cerebral convolutions are formed, it will be readily seen that the earlier-formed nerve cells and fibres occupy the summits of the convolutions, while the later-formed cells and fibres occupy the bottom of the fissures. It may be noticed, in confirmation of this view, that the summits of the convolutions are alone directly connected with the peduncular and radiating fibres, while the grey matter of the fissures is only connected with them indirectly through that of the summits by means of arcuate fibres. The depth of the fissures may, therefore, be taken along with the complexity of the arrangement of the convolutions and other circumstances as a measure of the degree of development of the brain. The distribution of the blood-vessels of the cortex would also lead us to expect that the superficial layer next the pia mater is the embryonic layer of the grey matter, while the earlier-formed portions occupy the

position furthest removed from the vessels, and consequently consist of the internal layers with caudate cells.

§ 682. *Differences between the brain of the adult man and that of the higher mammalia and human infant.*—The usual information given in anatomical works with regard to the size and weight of the human brain as compared with that of animals will be passed over here in order to insist upon less conspicuous, but probably not less important, differences.

There has been a widely-spread belief that the large development of the frontal lobes is peculiarly characteristic of the brain of man, but some years ago Dr. Carpenter drew attention to the fact that in the lower forms of animals the cerebellum is entirely uncovered by the cerebrum, that it is only partially covered by the posterior lobes in the more intelligent animals, such as dogs and monkeys, and that it is only in man that the posterior lobes of the cerebrum completely overlap the cerebellum. Dr. Carpenter argued from this that the increase of the posterior lobes is more characteristic of advance in development than that of the anterior lobes. But he ignores the fundamental facts of development, while regarding superficial appearance. If we take the fissure of Rolando, or the central sulcus in animals, as the line which separates the frontal from the parietal lobe, it will be noticed how small a portion of the brain lies anterior to the sulcus in such animals as the rabbit (*Fig.* 226, *r*), which

FIG. 226.

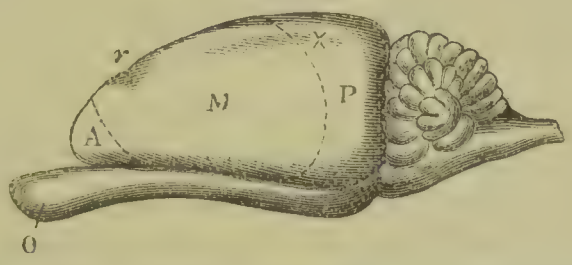


FIG. 226 (Modified from Ferrier). *Brain of Rabbit.*—O, Olfactory bulb; *r*, Central sulcus; ×, Parallel sulcus; M, Motor area; A, Anterior or psychical area; P, Posterior or sensory area.

is one of the lowest animals in which the sulcus is developed. Even in the dog and monkey only a relatively small part of the brain lies anterior to this sulcus compared to the large mass which lies behind it. And a study of the development of the human brain shows that the occipital and parietal lobes increase rapidly at an early period in the development of the embryo, while the frontal lobes increase chiefly during the later months of fœtal life. The portion which lies in front of the sulcus of Rolando in a six months human fœtus (*Fig.* 227, *r*) is small, while the sulcus itself is directed vertically upwards in a line with the anterior ascending limb of the fissure of Sylvius; the superior extremity being somewhat anterior to the inferior extremity. In the brain of the human adult the superior extremity of the sulcus of Rolando is pushed backwards, owing to the great increase of the frontal lobes, so

that a vertical line drawn from it would pass through the posterior extremity of the horizontal limb of the Sylvian fissure. During the development of the human brain the superior extremity of the sulcus of Rolando therefore suffers a backward displacement in order to make room for the increasing size of the anterior area of the cortex; and, similarly, in the evolution of the human brain from the simian type the occipital lobes have undergone a posterior displacement in order to make room for the relatively large increase of size of the frontal lobes, hence the covering of the cerebellum is not caused directly by an increased size of the occipital, but indirectly by an increased size of the frontal lobes.

Fig. 227.

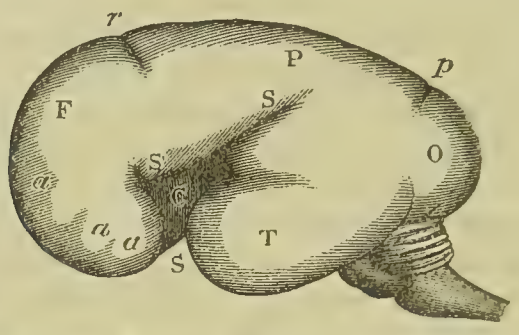


Fig. 227 (From Quain, after Wagner). *External Surface of the Fœtal Brain at Six Months.*—F, Frontal lobe. P, Parietal lobe. O, Occipital lobe. T, Temporal lobe. *a*, *a*, *a*, Slight appearance of the several frontal convolutions. S, Sylvian fissure; S′, its anterior division. C, Convolutions of the island. *r*, Fissure of Rolando. *p*, External part of the vertical fissure.

Another remarkable feature in which the human brain differs from that of animals is the manner in which the Island of Reil is completely surrounded, and hidden out of view by deep convolutions. This is brought about by the large development of the posterior extremity of the inferior frontal, the inferior extremities of the ascending frontal and parietal convolutions, and of the supra-marginal, angular, and inferior temporo-sphenoidal gyri. It appears to me that the cortex of the central lobe, starting from the grey matter of the anterior perforated space, is the embryonic portion of the cortex of the brain, just as the central grey column is the embryonic portion of the grey matter of the spinal cord. The anterior perforated space is a point where the grey matter of the two nuclei of the corpus striatum and of the cortex of all the lobes of the brain meet, and it may therefore be regarded as the starting point of the whole of the grey matter derived from the primary cerebral vesicles. On the supposition that the portion of the central lobe which lies in the line of distribution of the Sylvian artery is the embryonic portion of the convolutions of the central or motor area of the brain, it may be expected that the earlier-formed portions of these convolutions will be thrust upwards towards the great longitudinal fissure, while the later-formed portions approach nearer and nearer to the root of the artery. According to

this supposition therefore the fundamental portions of the convolutions supplied by the Sylvian artery will be found near the great longitudinal fissure, and the accessory portion low down, near the root of the artery, the latter corresponding to the convolutions named the operculum, which is so highly developed in man.

The great development of the supra-marginal and angular gyri is also a characteristic feature of the brain of man.

FUNCTIONS OF THE ENCEPHALON.

The functions of the medulla oblongata have already been described in detail, and those of the pons, corpora quadrigemina, and crura cerebri in a general way.

§ 683. *Functions of the Cerebellum.*—The cerebellum is, according to the view adopted in these pages, an organ of compound co-ordination in space, and regulates the continuous muscular actions which are necessary for the maintenance of certain attitudes in space. Flourens observed that when a small portion of the cerebellum was removed from a pigeon, the animal's gait became unsteady, and that when larger portions were taken away, the movements became much more disorderly. Section of the middle peduncle gives rise to a forced movement, the animal rolling round its longitudinal axis, and the rotation being generally towards the side operated upon. Injury of the lateral lobe of the cerebellum, and probably of the fibres of the peduncle as they pass transversely through the pons, produces the same forced movements as section of the middle peduncle. Nothnagel concludes from experiments on rabbits that lesions which injure the fibres uniting the two sides of the organ occasion the greatest amount of motor disturbance.

Ferrier found that electric stimulation of the cortex of the cerebellum in animals caused movements of both eyes, with associated movements of the head, limbs, and pupils.

§ 684. *Functions of the Basal Ganglia.*—The most generally received hypothesis, especially in England, with respect to the functions of these ganglia is that the optic thalami are concerned in the upward transmission and elaboration of centripetal impulses; and the corpora striata in the downward transmission and elaboration of centrifugal impulses. The

impulses elaborated by the thalami may either be transmitted anteriorly and laterally to the corpora striata, or upwards to the grey matter of the cortex, while the incitement to action may come to the corpora striata either from the thalami directly or from the cortex. When the impulses are transmitted directly from the thalami to the corpora striata, and from the latter downwards to the cord, then the basal ganglia may be regarded as the superordinates of the central grey tube; but when the impulses are transmitted from the thalami to the cortex, and from the latter to the corpora striata, the basal ganglia, although still the superordinates of the central grey tube, are the subordinates of the grey matter of the cortex.

Very serious objections have been urged against the view that the thalamus is the sensory ganglion of the opposite half of the body, but these have been fairly answered by Dr. Broadbent, to whose writings we are indebted for two most fruitful discoveries in the application of physiological principles to the elucidation of the phenomena of diseases of the nervous system.

The first objection is, that lesion of the thalamus does not impair sensation in the same degree that motor paralysis is caused by injury of the corpus striatum; but the reply is, that centripetal currents are more diffusely conducted than centrifugal currents, and that this feature is as characteristic of the grey matter of the posterior horns of the cord as it is of the thalamus. Another objection is, that if the thalamus be the common sensory ganglion, lesion of it ought to cause not only hemianæsthesia, but also unilateral blindness and deafness. To this objection Dr. Broadbent replies by extending his principle of the bilateral association of the nerve-nuclei of muscles bilaterally associated in action to the functions of the nerves of special sense. Bilateral association of sensation ought to involve fusion of sensory nuclei, and the combination of sounds reaching the ears, and of light reaching the retinæ, being completely fused into one sensation, the two auditory and the two optic nuclei ought to be fused practically into one, so that unilateral deafness or blindness from injury to one thalamus becomes thus impossible. Another objection to this view is, that while lesion of the thalamus is frequently unaccompanied by complete hemianæsthesia, it is sometimes accompanied by motor paralysis of the opposite side of the body; from this it has been argued that the thalamus is a superior centre for reflex action (Crichton Browne). It must, however, be remembered that the pyramidal fibres of the internal capsule lie almost immediately external and inferior to the thalamus, so that disease of the latter may readily implicate the former, and then paralysis of the opposite side result.

The hypothesis, therefore, that the thalamus is a centre for the com-

pound co-ordination of centripetal impulses is not open to any insuperable objections, and accords better with facts than any other theory of its function. The compound co-ordinated centripetal impressions may be transmitted directly to the corpus striatum, and reflected downwards to the anterior horns and anterior root-zones of the cord, thus causing a compound reflex action, or upwards to the cortex of the brain, where the impressions become correlated with feeling. There are no sufficient grounds for believing that the activity of the thalamus implies consciousness, even of the most rudimentary kind. A compound differs from a simple reflex action not only as being more complex, but also as consisting of a succession of *different* actions. The act of sucking in an infant is a complex act, but it consists of a series of *similar* complex actions in response to a series of similar impressions, and this action may be taken as a good example of reflex actions in general. But when a chicken has just burst the shell, and almost immediately begins to pick grains of food off the ground, the necessary actions are not only complex, but consist of a succession of *different* complex actions in response to different complex impressions. There is no reason to believe that the latter action is a conscious one, any more than that of sucking in an infant; but while the latter is a simple reflex action, and co-ordinated in the central grey tube, the former is a compound reflex action, and co-ordinated in the basal ganglia acting in association with the central grey tube and probably also with the cerebellum. When impressions are made upon a large number of the end organs of the afferent nerves, these, after being first co-ordinated in the posterior part of the grey matter of the central grey tube, undergo, on ascending, a second co-ordination in the thalami, whereby they are integrated in various ways, and reduced to something like serial order. When the centripetal impulses so arranged are transmitted to the corpora striata, and reflected downwards, they give rise to a succession of muscular contractions; when again they are transmitted to the cortex—which is, as we have already remarked, the organ of doubly compound co-ordination in time—their serial order adapts them for evoking the rhythmical sequences of centrifugal impulses which regulate complex psychical actions.

The *corpus striatum*, on the other hand, is a centre for the compound co-ordination of centrifugal impulses for the opposite half of the body. When it acts in obedience to impulses received from the optic thalamus, it is an organ of compound reflex action. All the actions which are regarded as inherited instincts, or which through long-continued repetition have assumed the character of acquired instincts, are of the nature of compound reflex actions; they are or have become independent of consciousness, and are co-ordinated in the basal ganglia. But the corpus striatum is supposed to act in obedience to impulses received

through the cortex of the brain. We have already seen that there is an uninterrupted connection between the cortex and grey matter of the cord by means of the pyramidal fibres, and we must now endeavour to differentiate the functions of the cortex when it acts through the latter fibres and through the corpus striatum respectively. A simple illustration will make this clear. When a child is learning to write, the muscles of the thumb, index, and middle fingers are moved in separate groups, so that the fingers are ultimately brought to a proper attitude for holding the pen. Subsequently the separate groups of muscles are brought successively into action, whereby the point of the pen is moved upwards, downwards, and laterally, so as to produce the elementary strokes of writing. These actions, described in subjective terms, are not simply conscious, but involve that active consciousness which constitutes attention, and they are also deliberate, the outward sign of deliberation being slowness of execution. The centrifugal impulses which initiated these movements may be presumed to have passed through the pyramidal fibres. After long-continued habit, however, the actions involved in writing are to a large extent, if not wholly, unconscious, and demand no deliberation, and this absence of deliberation is accompanied by extreme rapidity of execution. The centrifugal impulses regulating these actions are co-ordinated in the corpus striatum, under the guidance of a relatively small number of impulses from the cortex. This illustration also shows that the progress of education is from actions which are at first regulated through the pyramidal fibres, to actions which are regulated through the corpus striatum.

The characteristics of the actions regulated through the pyramidal fibres are, that they are complex, slowly executed, and grouped in an unusual manner; while the characteristics of the actions which are regulated through the corpus striatum are, that they are quickly executed, and arranged in frequently repeated combinations. Now, the slowly executed movements grouped in unusual ways precede in the order of development the quickly executed and habitual movements, and the structural correlative of this fact is, that in the course of development the pyramidal fibres assume a medullary sheath some time before the fibres in the crusta which connect the cord with the corpus striatum. All the complex movements which animals manifest in response to emotional disturbances are

organised in the corpora striata. The attitude expressive of fear and anger assumed by a cat when threatened by a dog may be taken as a familiar example of such actions. Mr. Darwin's description of this attitude is that the cat "arches its back in a surprising manner, erects its hair, opens its mouth, and spits." The regulation of the muscular movements concerned in producing this attitude is organised mainly in the corpora striata, but the incitement to the action of these centres in such a case comes from the cortex.

My friend Dr. Noble, of Manchester, whose work "On the Human Mind in its relations with the Brain and Nervous System" was so much in advance of the time in which it was written, was the first to suggest that the movements which are in relation with the desires and emotions are regulated through the basal ganglia acting in subordination to the cortex of the brain; but this view, like many of his other opinions, did not then attract the attention it deserved.

To illustrate the functions of the basal ganglia, let us suppose that an impression is made on the retina by a minute object, such as a fly, approaching the eye. The eyelids immediately close. This action is purely reflex, and is determined by the corpora quadrigemina and cord, uninfluenced by the basal ganglia. Part of the disturbance, however, is conveyed to the optic thalami, and by them co-ordinated in such a way that on reaching the cortex of the brain they give rise to a sensation, or even to an indistinct perception, but the closure of the lids is quite independent of, and prior in time to, the sensation or perception. Let us now suppose that the impression on both retinæ is made by a larger body, such as a cricket-ball, at a considerable distance from the eyes, but moving towards them. The disturbances produced are conducted inwards by the optic nerves and the afferent nerves of the ocular muscles, and after being elaborated by the sensory part of the grey matter of the pons and corpora quadrigemina, some of them pass upwards to reach the cortex through the sensory fibres of the internal capsule, while others are conducted to the thalami, and after having undergone a second elaboration and reduction to something like serial order, they also are transmitted to the cortex. The mental correlative of the cortical disturbance is a perception of the object and of its position in space, and of the rate and direction of its motion. Centrifugal impulses may now be sent from the cortex to the inferior centres, which will eventuate in a series of movements, either to catch the ball or to avoid collision with it. One man, in whom no special aptitude has been organised with respect to the motion of the ball, may simply move his head to one side to avoid collision. The slower the execution is the more sure we are that it has not been frequently repeated in the previous experience of the individual, and that it has been determined by conscious and volitional impulses. In such a case the volitional or centrifugal impulses are conducted outwards through the pyramidal fibres, and the corpora striata have had nothing to do with it. Another man, or rather a woman, on

seeing the ball may exhibit the outward manifestations of alarm by facial expression or screaming, and execute a series of locomotive actions far greater than necessary to avoid collision with the ball, and the greater these outward manifestations are, the more certain we may be that the sensory impressions on reaching the cortex have caused a profound emotional disturbance, and that the centrifugal impulses reach the periphery through the corpora striata. But a third man, instead of endeavouring to avoid collision, may put up both hands so as to catch the ball. Now, the centrifugal impulses may pass in this case either through the pyramidal fibres or corpora striata, according to circumstances. If the action have been frequently repeated so that it be done with precision, and without a feeling of conscious effort, its regulation is organised in the corpora striata ; and if it be done awkwardly, and with the inward feeling and outward manifestation of a conscious effort, then the centrifugal impulses have passed through the pyramidal fibres.

It must, however, be admitted that the foregoing account of the functions of the basal ganglia is by no means fully established. We have already stated that the internal surface of the optic thalamus is lined by a layer of grey substance which represents the upper end of the central grey tube, and that the upward continuations of the anterior root-zone of the cord terminated in this ganglion, and consequently it must be presumed that a portion at least of the thalamus is endowed with motor functions. The opinion that the optic thalami is a high reflex centre has been ably sustained by Dr. Crichton Browne on pathological grounds. The anatomical difficulties which stand in the way of regarding the corpus striatum as an intermediate ganglion between the cortex of the brain and the central grey tube are also very great. The latest researches of Wernicke appear to show, as we have seen, that neither the lenticular nor the caudate nucleus possess radiating fibres ; and if this be the case, the corpus striatum must be regarded as a nerve centre co-ordinate with and not subordinate to the cortex. Ferrier observed that when the corpora striata were stimulated by a strong interrupted current, the muscles of the opposite side of the body became strongly contracted ; but it is impossible to prevent even weak currents through the corpus striatum from affecting the fibres of the internal capsule, and the spasm of the opposite side would be probably caused by irritation of the fibres of the pyramidal tract. We shall hereafter see that when hemiplegia occurs from hæmorrhage into the corpus striatum, the patient recovers if the fibres of the pyramidal tract remain uninjured. Nothnagel found that destruction or injury to a particular part of the caudate nucleus gave rise in the rabbit to remarkable forced movements.

§ 685. *Functions of the Cortex of the Cerebrum.*—The cortex of the cerebrum is probably the exclusive seat of psychical action, and there seem to be no grounds for believing that the activity of any other portion of the encephalon is necessarily connected with

even the crudest consciousness. But before we can refer certain states of consciousness to definite processes in the cortex of the cerebrum, it is necessary to have a classification of mental phenomena, for no decided progress can be made in interpreting the results of experiments on the cortex of the brain until the true nature of a psychical action is defined and some rational classification of psychical states is adopted by physiologists.

Nature of Psychical Actions.—We have already seen that simple reflex adapted actions consist of a series of *similar* complex movements evoked by a series of similar impressions, and that compound reflex adapted actions consist of a series of *different* complex movements evoked by a series of different impressions; and we must now endeavour to show wherein true psychical action differs from simple and compound reflex actions. Reflex actions, both simple and compound, consist of three factors: (1) conduction to a nerve centre of an impression made on the surface; (2) reduction to order of these impressions in the centre; and (3) conduction of these outwards, with the muscular contractions resulting from them. But, as has been frequently stated by Mr. Herbert Spencer, four factors may be distinguished in every psychical action. To quote Mr. Spencer's own language, "there is (*a*), that property of the external objects which primarily affects the organism—the taste, smell, or opacity; and, connected with such property, there is in the external object that character (*b*) which renders seizure of it, or escape from it, beneficial. Within the organism there is (*c*), the impression or sensation which the property (*a*) produces, serving as stimulus; and there is, connected with it, the motor change (*d*), by which seizure or escape is effected. Now psychology is chiefly concerned with the connection between the relation *a b*, and the relation *c d*, under all those forms which they assume in the course of evolution. Each of the factors, and each of the relations, grows more involved as organisation advances. Instead of being single, the identifying attribute *a*, often becomes, in the environment of a superior animal, a cluster of attributes, such as the size, form, colours, motions, displayed by a distant creature that is dangerous. The factor *b*, with which this distant combination of attributes is associated, becomes the congeries of characters, powers, habits,

which constitutes it an enemy. Of the subjective factors, *c* becomes a complicated set of visual sensations co-ordinated with one another and with the ideas and feelings established by experience of such enemies, and constituting the motive to escape; while *d* becomes the intricate, and often prolonged, series of runs, leaps, doubles, dives, &c., made in eluding the enemy."

Classification of Psychical States.—Various classifications of mental states might be adopted, but the best is clearly that which involves the fewest assumptions and theoretical implications, and which will enable us at the same time to connect mental phenomena with the facts of development and experimental physiology.

"It would be the greatest benefit to mental science," says Max Müller, "if all such words as perception, intuition, remembering, ideas, conception, thought, cognition, senses, mind, intellect, reason, soul, spirit, etc., could for a time be struck out of our philosophical dictionaries, and not be admitted again till they had undergone a thorough purification." This passage expresses a state of mind which has been felt by almost everyone who has seriously engaged in psychological study; and Mr. Herbert Spencer, whose great works have formed an era in philosophy and psychology, has, with his usual breadth of treatment, adopted a classification which does in a great measure avoid the use of these words, except indeed where the use of them admits of accurate definition. We shall avail ourselves of this classification in our future remarks.

Mr. Spencer subdivides all mental states into *volitions, cognitions,* and *feelings;* and the first of these subdivisions may be disposed of in a few words. "Will," says Mr. Herbert Spencer, "is a simple homogeneous mental state, forming the link between feeling and action, and not admitting of subdivisions."

"*Cognitions,*" says Mr. Spencer, "are those modes of mind in which we are occupied with the *relations* that subsist among our feelings." They are divisible into four great sub-classes.

(1) "*Presentative cognitions,* or those in which consciousness is employed in localising a sensation impressed on the organism."

(2) "*Presentative-representative cognitions*, or those in which consciousness is occupied with the relations between a sensation or group of sensations and the representations of those various other sensations that accompany it in experience" (perceptions).

(3) "*Representative cognitions*, or those in which consciousness is occupied with the relations among ideas or represented sensations, as in all acts of recollection" (concrete ideas).

(4) "*Re-representative cognitions*, or those in which the occupation of consciousness is not by representation of special relations that have before been presented to consciousness, but those in which such represented special relations are thought of merely as comprehended in a general relation—those in which the concrete relations once experienced, in so far as they become objects of consciousness at all, are incidentally represented along with the abstract relation which formulates them" (abstract ideas). "It is clear," Mr. Spencer adds, "that the process of representation is carried to higher stages as the thought becomes more abstract."

Feelings, or those modes of mind in which we are occupied, not with the relations subsisting between our sentient states, but with the sentient states themselves, are divisible into four parallel sub-classes.

(1) *Presentative feelings* are those in which a corporeal impression is regarded as pleasure or pain (sensations).

(2) *Presentative-representative feelings* are those in which a sensation or a group of sensations arouses a vast group of represented feelings (emotions).

(3) *Representative feelings*, comprehending the ideas of the emotions when they are called up, apart from the appropriate external excitements, such as the emotions excited by a vivid description.

(4) *Re-representative feelings* are those more complex sentient states that are less the direct results of external excitements than the indirect or reflex results of them, such as the love of property, which consists of the represented advantages of possession in general, which is not made up of certain concrete representations, but of the abstracts of many concrete representations.

"The classification," Mr. Spencer proceeds, "here roughly indicated, and capable of further expansion, will be found in harmony with the results of decided analysis aided by development. Whether we trace mental progression through the grades of the animal kingdom, through the grades of mankind, or through the stages of individual growth, it is obvious that the advance, alike in cognitions and feelings, is, and must be, from the presentative to the more and more remotely representative. It is undeniable that intelligence ascends from those simple perceptions in which consciousness is occupied in localising and classifying sensations, to perceptions more and more compound, to simple reasoning, to reasoning more and more complex and abstract, more and more remote from sensation. And in the evolution of feelings there is a parallel series of steps. Simple sensations; sensations combined together; sensations combined with represented sensations; represented sensations organised into groups in which their separate characters are very much merged; representations of those representative groups in which the original components have become still more vague. In both cases the progress has necessarily been from the simple and concrete to the complex and abstract; and as with the cognitions so with the feelings, this must be the basis of classification."

It is not, perhaps, possible in the present state of our knowledge to separate the cortex of the brain into areas exactly corresponding to the various subdivisions of Mr. Herbert Spencer's classifications. The cortex may, however, be subdivided into areas which will correspond with the leading features of this classification.

1. The cortex of the brain must maintain some connection with the surface of the body, by means of which impressions made on the latter occasion molecular changes in the former. The parts at which the cortex is connected with the centripetal system of nerves may be called *sensory inlets*, and if the portion of the cortex containing these inlets can be isolated from the remaining portions of the cortex, there can be no serious objections to calling it the *sensory area of the cortex*. And, indeed, if the inlets from the various senses can be more or less isolated from one another, each may be called a

sensory centre. We have already seen that the posterior third of the posterior division of the internal capsule contains centripetal fibres for the opposite half of the body, and that these radiate in the centrum ovale to terminate in the convolutions of the occipital and temporo-sphenoidal lobes, or the area of the cortex, which is supplied by the posterior cerebral artery.

That the fibres of the tract which ascends in the external third of the crusta and posterior part of the external capsule are sensory has been proved by the experiments of Veyssière, and confirmed by Carville and Duret, Raymond, and others. Veyssière showed that section of the posterior part of the internal capsule lying between the lenticular nucleus and optic thalamus was followed by hemianæsthesia of the opposite side of the body.

FIG. 228.

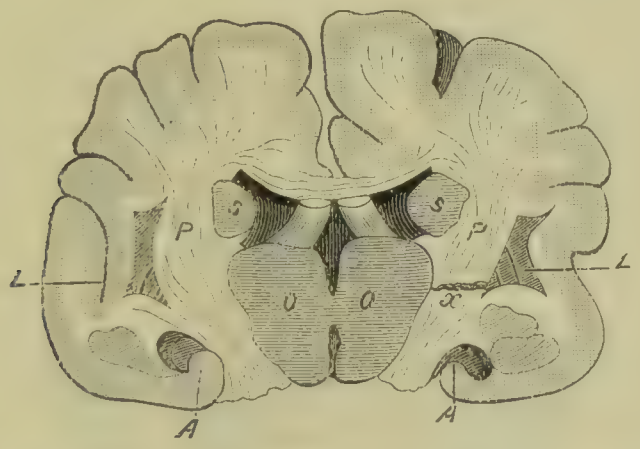


FIG. 228 (After Carville and Duret). *Transverse Section of the Brain of a Dog on a level with the Corpora Albicantia.*—*O*, *O*, Optic thalami; *S*, *S*, Caudate nuclei; *L*, *L*, Lenticular nuclei; *P*, *P*, Posterior region of the internal capsule; *x*, Section of the posterior part of the internal capsule determining hemianæsthesia; *A*, *A*, Cornu Ammonis.

2. The cortex of the brain must be connected with the muscular system, in order that the reactions of the organism upon its environment may be regulated in correspondence with the impressions made upon it. The parts at which the cortex is connected with centrifugal fibres may be called *motor outlets*, and if the portion of the cortex which contains these can be isolated from the remaining portions of the cortex it may be called the *motor area.* And if the motor outlet for a particular movement can be isolated from the outlets for other movements there can be no great harm in calling it a *motor centre.*

A cortical motor centre then constitutes the link between cortical activity on the one side, and voluntary muscular contractions on the other; and volition being the link between feeling and action, the cortical motor centres may be regarded as the structural counterparts of volitions.

We have already seen that the pyramidal tract contains the centrifugal fibres from the cortex of the brain, and this has also been determined experimentally by Veyssière, who found that section of the anterior two-thirds of the internal capsule was followed by hemiplegia of the opposite side, unaccompanied by sensory paralysis.

Fig. 229.

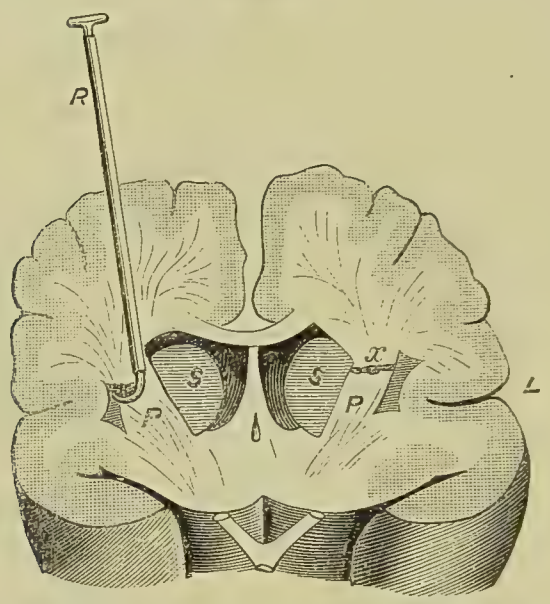


Fig. 229 (After Carville and Duret). *Transverse Section of the Brain of the Dog, five millimetres in front of the optic commissure.—S, S,* The caudate nuclei of the corpora striata; *P, P,* Peduncular fibres (the internal capsule); *L,* Lenticular nucleus; *R,* Stylet, by means of which Veyssière produced section of the internal capsule at (*x*).

But the pyramidal tract is not, according to most anatomists, the only outlet from the cortex of the brain. Leaving out of account the centrifugal fibres which probably connect the cortex of the cerebrum with the cerebellum, there still remain the fibres which connect the cortex of the cerebrum with the central grey tube through the intermediation of the corpus striatum. The cortical actions which are regulated through the corpus striatum and pyramidal tract are often, although not always, antagonistic to one another. The excitation of the cortex which is the correlative of feeling, whether the latter be pleasurable or painful, always tends to find a vent in immediate action, while a great portion of our voluntary efforts are directed to restrain action, and to postpone the im-

mediate gratification of the feelings in order to accomplish remote ends. Excitation, for instance, of the cells in the cortex which are in immediate contact with the terminations of the centripetal nerves in the summits of the convolutions of the posterior area of the cortex tends to be conducted immediately outwards along the centrifugal fibres which connect these convolutions with the corpus striatum. If these excitations are conducted at once outwards, they give rise to movements which have been named *sensori-motor;* but if the excitations, instead of being conducted at once outwards, pass from the cells in connection with the termination of one bundle of centripetal fibres (vision) to those in connection with another bundle (tactile), so that the *relation* between the two feelings comes into prominence, then a presentative cognition is formed. When, for instance, the centripetal impulse received in a convolution from the irritation caused by a thorn in the finger is brought into connection with the impressions received through the optic and other centripetal nerves, and which, on reaching the cortex, becomes the correlative of the consciousness of the finger itself, then a cognition of the relationship of presentative feeling is formed. Now, a presentative cognition does not usually, like a presentative feeling, immediately result in action. The excitation expends itself in the former in producing excitation of other groups of cells in the cortex, the transition from one group to another giving rise to other presentative and representative cognitions, until finally the motor area is reached, and the excitation passes out along the pyramidal fibres. Subjectively considered, the cognition of the thorn and finger would call up other cognitions connected with these by previous experiences, as that of a pin, and probably the highly representative cognitions of the general properties of the lever, until finally the pin is voluntarily grasped and rightly applied for the removal of the thorn. This action is very different from that which impels a dog to lick with his tongue the foot in which a thorn is lodged. The latter is a sensori-motor or doubly compound reflex action, and in immediate relation with the cortical excitation which causes the feeling of pain, while the former results from a series of complex cortical excitations, some of which check the tendency to immediate action, until by-and-by complex actions result which are guided by wide experience and adapted to remote ends. The movements which result immediately from the feelings have been called sensori-motor, percipio-motor, and ideo-motor, on the supposition that they occurred in response to the cognitions; but it would be better to call the movements which result from a presented feeling a doubly compound reflex action, that from a presentative-representative feeling a trebly compound reflex action, and that from a representative feeling a quadruply compound reflex action, and so on in an ascending scale, according to the degree of the complexity of the feeling. When, however, a series of cognitions intervene in the mental operations between the feeling which prompts a movement and the movement itself, the resulting muscular adjustment is a voluntary one, and is regulated through the pyramidal fibres. When, for instance,

Leverrier, prompted by the highly representative feeling of a desire for discovery, directed his telescope to a certain spot in the heavens, and discovered Neptune, the requisite muscular adjustments necessary for carrying out this action were preceded in his mind by a long series of involved and highly representative cognitions; and these muscular adjustments themselves were, to a large extent, voluntary.

3. The region of the cortex supplied by the anterior cerebral artery still remains to be connected with some kind of mental activity. We have seen that the area supplied by the posterior cerebral artery is the sensory area, and consequently excitation of this area is the correlative of the presentative and presentative-representative cognitions and feelings, while excitation of the area supplied by the middle cerebral artery is the correlative of volition. Excitation of the cortical area supplied by the anterior cerebral artery is the correlative again of the representative and re-representative cognitions and feelings. It is somewhat difficult to find a name which will be expressive of the functions of this area, and if we consent to call it the *ideational* area, it must be remembered that it is no less likely to be the anatomical substratum of the higher emotions than of the higher intellectual operations.

§ 686. *Anatomical Substratum of Consciousness.*—It is well recognised that a large number of psychical actions may take place in an unconscious manner. Leaving out of consideration the phenomena of dreaming and somnambulism, we may instance such a familiar fact as that a man may read aloud whole pages of a book while his mind is engaged in solving a difficult problem, and he is wholly unconscious of what he is saying, yet the muscular movements engaged in reading are co-ordinated in the cortex of the cerebrum. If, under these circumstances, the eye falls on an unusual word, consciousness is directed to it for a moment, and the reading may then go on unconsciously as before. It would therefore appear that impressions which have been frequently repeated in experience may pass up to the cortex and give rise to complicated motor impulses from the cortex without being attended by consciousness; but that when the impressions made on the sensory organ present an unusual combination, consciousness is aroused. Unusual combinations

of sensory impressions are, therefore, probably conducted to and through the cortex in channels which are only partially open, while the habitual combinations pass in channels which are open and well defined. In intellectual efforts the highest consciousness is aroused when the mind is contemplating new combinations of presentative and representative impressions, or, to translate this into the language of physics, when the organism is adjusting itself to new combinations of circumstances and events. In other words, the highest intellectual consciousness is aroused during the time that a new organisation in the cortex of the brain is being superadded to the existing one, while excitation of the portion of the cortex which is already thoroughly organised is attended by little or no consciousness.

It cannot be supposed that the large cells, with the distinct processes and definite connections found in the internal division of the third layer of the cortex, will readily undergo structural changes in the healthy adult, and it is much more probable that any new alteration of structure in the cortex will proceed from the small cells of the external layers of the cortex. The first layer may probably be regarded as an embryonic layer without any active nerve functions, and consequently the second layer and external portion of the third layer of the cortex, the cells of which do not possess definite connections with one another or with nerve fibres, must be regarded as the areas, excitation of which is attended by the highest consciousness.

Experiments on animals have proved, as we have seen, that the fibres which pass through the posterior third of the posterior division of the internal capsule are sensory, but the sensory area of the cortex is also connected with the periphery, through the optic thalamus and its radiating fibres. It is probable that impressions which have been frequently repeated in experience pass through the optic thalamus and its radiating fibres, and that they give rise to little or no consciousness on reaching the cortex. It may be presumed, on the other hand, that unusual combinations of impressions are conducted through the posterior fibres of the internal capsule, and give rise on reaching the cortex to distinct consciousness.

§ 687. *Experimental Determination of the Functions of the Cortex of the Brain.*

1. *Motor Centres.*

Experiments by Stimulation.—When the cerebrum is removed slice by slice there is a gradual loss of intelligence and volition, and consequently Flourens, who conducted these experiments, concluded that the brain acted, as a whole, without any special functions being assigned to special parts. Hughlings-Jackson, however, drew attention to the fact that focal disease of the cortex of the brain may occasion epileptiform convulsions, localised to particular groups of muscles. Hitzig and Fritsch showed that the local application of the galvanic current to particular parts of the cerebral convolutions gives rise to definite movements of various groups of muscles. These experiments were extended and rendered more definite by Ferrier, who used the faradic instead of the galvanic current as a means of stimulation. The motor centres as determined by Ferrier in the monkey are represented in *Figs.* 230 and 231, while the corresponding parts in the human brain are shown in *Figs.* 232 and 233.

FIG. 230.

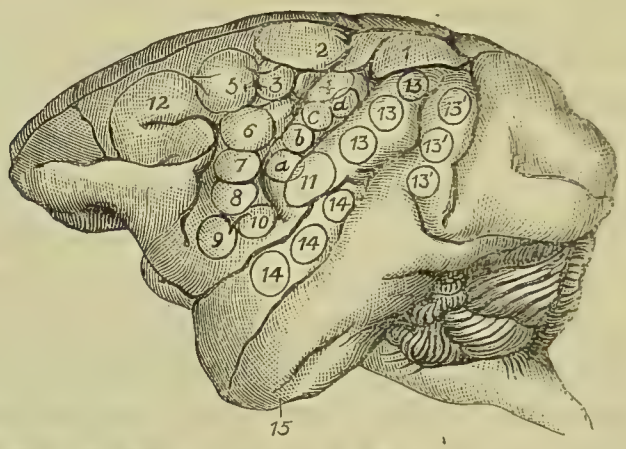


FIG. 230 (After Ferrier). *The Left Hemisphere of the Monkey.*

Burdon-Sanderson states that the motor reactions to cortical stimulation are not prevented from taking place by a horizontal incision carried some distance from the surface. This simply shows that a faradic current applied to the surface of the brain is conducted into the centrum ovale and stimulates the ends of the divided pyramidal fibres, but it does not show that the cortex is non-excitable.

Burdon-Sanderson also found that local stimulation of the white matter immediately surrounding the corpus striatum produces localised movements similar to those caused by stimulation of the corresponding cerebral surface. This experiment, like the last, shows that the fibres of the pyramidal tract are excitable, but it proves nothing with regard to the excitability or non-excitability of the cortex. If the motor area of the

cortex be removed, excitation of the subjacent white substance causes the same movements as excitation of the motor centre itself. In such a case the ends of the fibres of the pyramidal tract which issued from the motor centre are now exposed, and excitation of them causes the same kind of motor reaction as that caused by excitation of the motor centre itself. If the animal, however, survive the operation, the pyramidal fibres undergo secondary descending degeneration, and excitation of the scar or

FIG. 231.

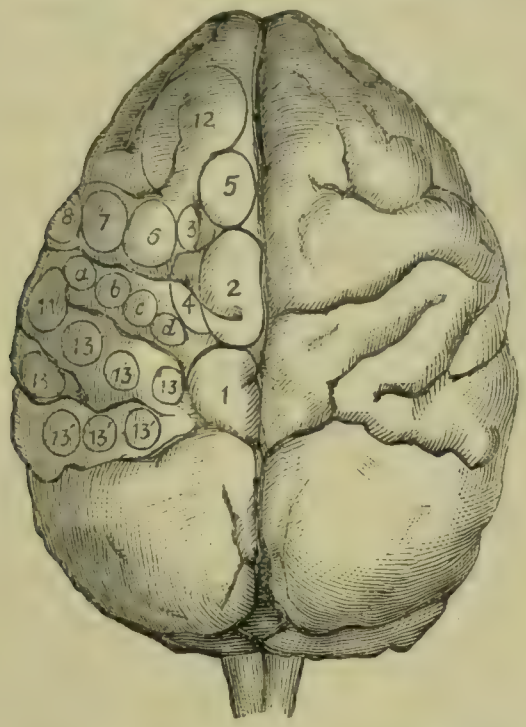


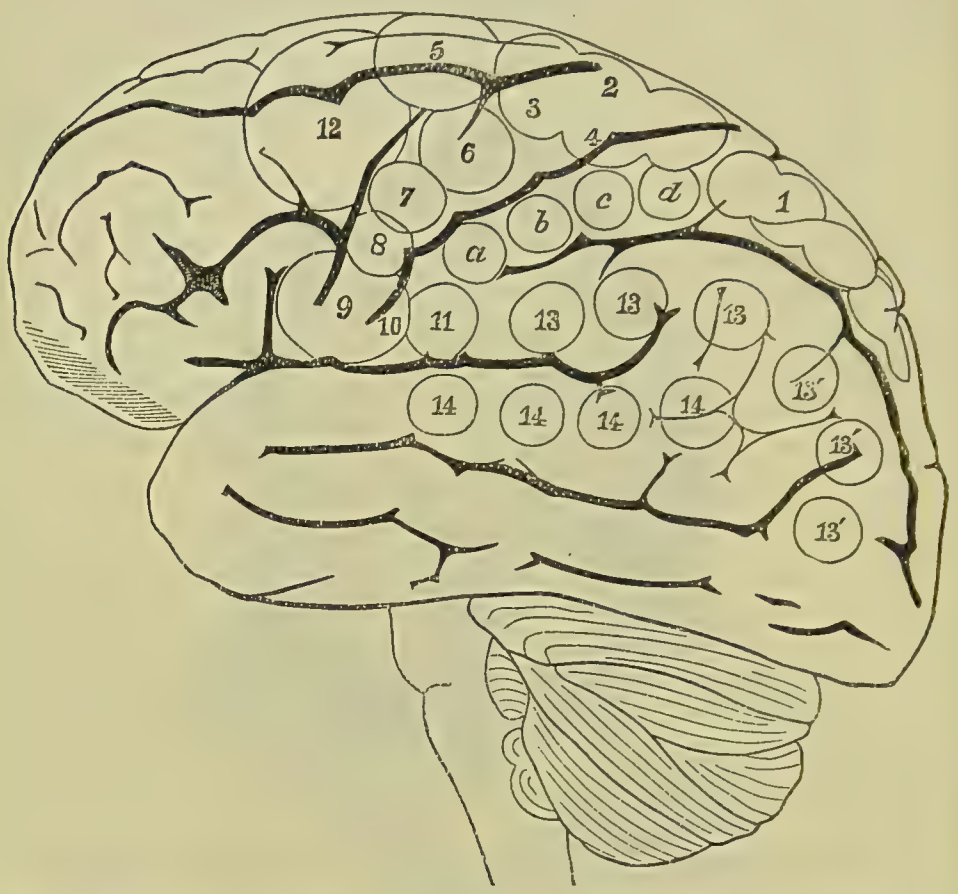
FIG. 231 (After Ferrier). *Upper Surface of the Hemisphere of the Monkey.*

1, Advance of the opposite leg as in walking.
2, Complex movements of the thigh, leg, and foot, with adapted movements of the trunk.
3, Movements of the tail.
4, Retraction and adduction of the opposite fore limb.
5, Extension forward of the opposite arm and hand, as if to reach or touch something in front.
Circles (*a*), (*b*), (*c*), (*d*), Individual and combined movements of the fingers and wrists, ending in clenching of the fist.
6, Supination and flexion of the forearm, by which the hand is raised towards the mouth.
7, Action of the zygomatics, by which the angle of the mouth is retracted and elevated.
8, Elevation of the ala of the nose and upper lip, with depression of the lower lip, so as to expose the canine teeth on the opposite side.
9, Opening of the mouth with protrusion of the tongue.
10, Opening of the mouth with retraction of the tongue.
11, Retraction of the angle of the mouth.
12, Eyes opening widely, pupils dilating, head and eyes turning towards the opposite side.
13 and 13′, Eyeballs moving to the opposite side. Pupils generally contracting.
14, Sudden retraction of the opposite ear.
15, Subiculum cornu Ammonis. Torsion of the lip and nostril on the same side.

its neighbourhood no longer produces the particular movements characteristic of the destroyed area (Albertoni and Michieli).

Experiments by Destruction of Portions of the Cortex.—It has been observed that removal or destruction of a motor centre is followed

FIG. 232.



FIGS. 232 *and* 233 (After Ferrier). *Side and Upper Views of the Brain of Man.*

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

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

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

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

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

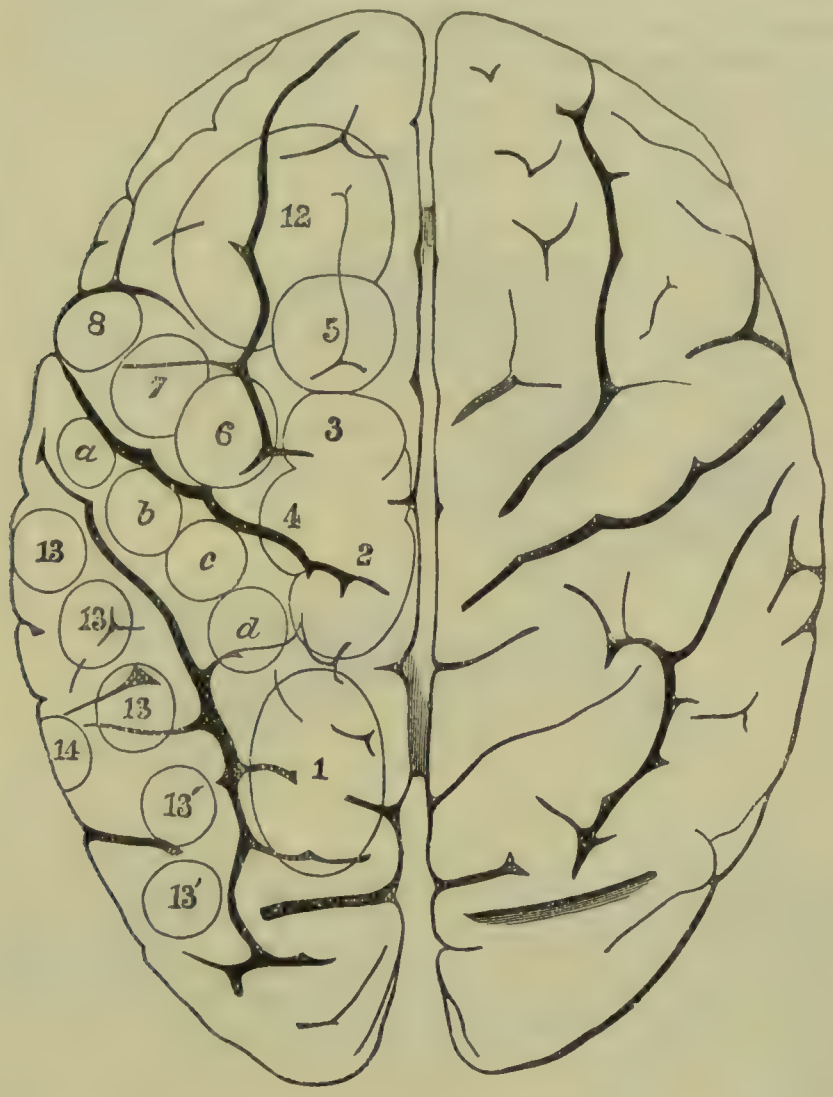
6 (On the upper part of the ascending frontal convolution), Supination and flexion of the opposite forearm.

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

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

by inability to execute the movements assigned to the area (Hitzig, Ferrier), but it has subsequently been found that the paralytic symptoms disappear when the animal operated upon survives some days (Nothnagel, Hermann, Goltz). Hermann removed cortical motor centres from dogs,

FIG. 233.



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

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

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

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

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

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

and found that the paralysis, which immediately followed the operation, disappeared in a few days. These results were afterwards confirmed by Carville and Duret, who also found that the restoration of motor power could not have been due to the corresponding centre of the opposite hemisphere, inasmuch as subsequent destruction of the latter produced the usual paralysis on the side opposite to the lesion, but did not cause a repetition of the paralysis on the side opposite to the first lesion. These authors suppose that portions of the same hemisphere took up the functions of the destroyed centre.

FIG. 234.

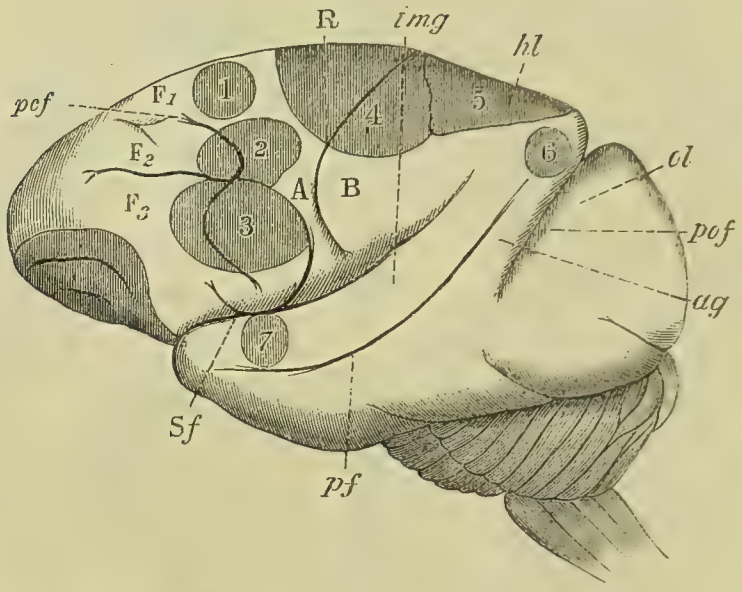


FIG. 234 (After Broca and Gomier). *External Convex Surface of the Brain of the Adult Monkey.—Fissures:* R, Fissure of Rolando: Sf, Fissure of Sylvius; *pf*, Parallel fissure; *pof*, External perpendicular or parieto-occipital fissure; *pcf*, Præ-central fissure. *Convolutions:* A, Ascending frontal convolution; B, Ascending parietal convolution; F_1, F_2, F_3, First, second, and third frontal convolutions; *ag*, Angular gyrus; *img*, Infra-marginal gyrus; *hl*, Horizontal lobule; *ol*, Occipital lobe. *Motor Centres:* 1, Movements for rotation of head and neck; 2, Movements of muscles of the face; 3, Movements of the tongue and jaws; 4, Movements of anterior extremity; 5, Movements of posterior extremity; 6, Movements of the ocular muscles; 7, Movements in relation with sense of hearing.

Goltz removed parts of the cerebral surface by washing the nervous substance away by a stream of water, and he came to the conclusion that the paralytic phenomena did not depend so much upon the locality as the extent of the injury. He also found that the paralysis disappeared in a short time, whatever might be the portion of brain removed. He was able in one case to remove the greater part of one hemisphere, and yet recovery of motor power took place, clumsiness in the execution of certain movements alone remaining; this Goltz attributed to a deficiency of tactile sensibility. He thinks that the paralytic phenomena are caused by an inhibitory action produced by the injury on lower centres, similar to the

temporary paralysis of the automatic centres in the lumbar portion of the spinal cord produced by section in the dorsal region.

But examples of a localised destroying lesion of the cortex of the brain in man have now multiplied to such an extent that there is no room for doubt with regard to the main symptoms caused by them, whatever may be the interpretation. When a patient has been unable to move his right arm and hand for months, and when after death a destructive lesion of the cortex of the opposite hemisphere is observed strictly limited to the middle of the ascending frontal and parietal convolutions, and when cases of this nature occur with sufficient frequency to show that the connection between the lesion and symptoms during life is not accidental, it is idle for any physiologist to deny that the paralysis was due to destruction of the cortex in that area, inasmuch as only temporary paralysis would be caused by a similar lesion in the dog. The fact that paralysis following cortical lesions in the dog is only temporary shows that differences must exist with regard to the relation which obtains between the highest nerve centres and muscular movements in man and the dog respectively. And it is not difficult to point out where some of these differences lie.

FIG. 235.

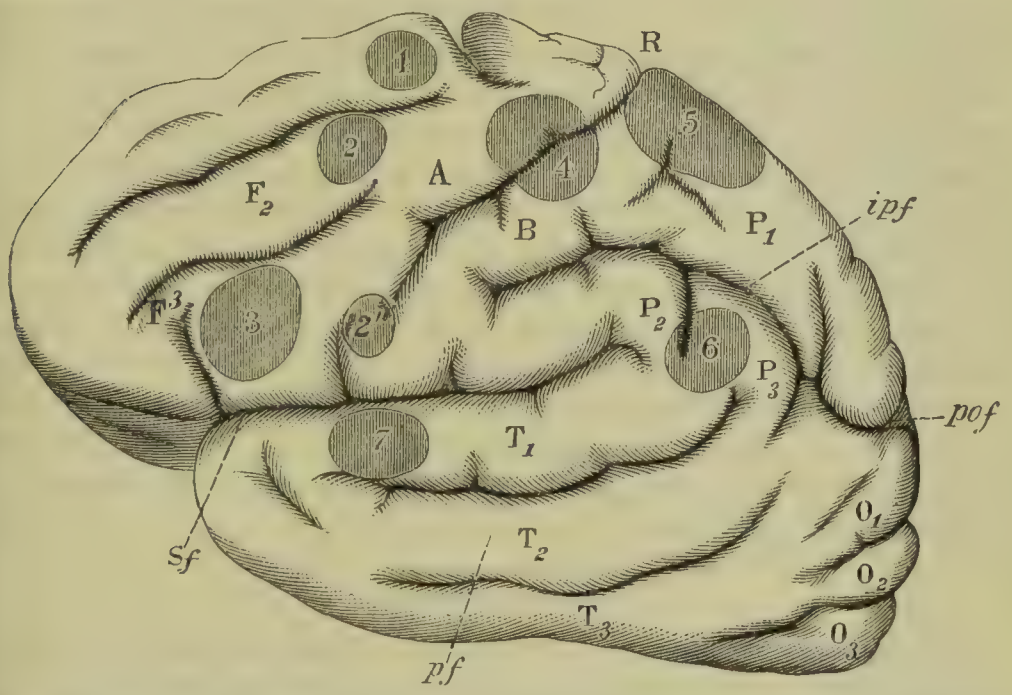


FIG. 235. *External Convex Surface of the Human Brain.—Fissures:* R, Fissure of Rolando; S*f*, Fissure of Sylvius; *pf*, Parallel fissure; *ipf*, Interparietal fissure; *pof*, External parieto-occipital fissure. *Convolutions and Lobules*: A, Ascending frontal; B, Ascending parietal convolutions; F_1, F_2, F_3, First, second, and third frontal convolutions; P_1, Superior parietal lobule; P_2, Supra-marginal gyrus; P_3, Angular gyrus; O_1, O_2, O_3, First, second, and third occipital convolutions; T_1, T_2, T_3, First, second, and third temporo-sphenoidal convolutions. *Motor Centres:* 1, Movements for rotation of head and neck; 2, Movements of the upper facial muscles; 2′, Movements of the lower facial muscles; 3, Movements of the tongue and jaws; 4, Movements of superior extremity; 5, Movements of inferior extremity; 6, Movements of the ocular muscles; 7, Movements in relation with the sense of hearing.

We have already seen that paralysis of the external rectus muscle of the one side and of the internal of the other occurs in most cases of sudden hemiplegia in man, causing a conjugate deviation of the eyes away from the paralysed side. This paralysis disappears in a few days, and is almost exactly similar to what occurs in the paralyses of cortical lesions caused in the dog. And even injury to the deep-seated parts of the brain in the dog, such as the crus cerebri, does not cause a hemiplegia at all comparable to the hemiplegia which occurs in man. In lesion of the right crus cerebri in the dog there is only a very partial hemiplegia. When standing the animal carries his body towards the right, his eyes are directed to the right, and his head is also rotated to the right, and if the animal move he goes round in a circle after his tail (Broadbent). It would not be more preposterous to tell us that because injury of the crus cerebri causes a *mouvement de manège* in the dog it cannot therefore cause hemiplegia in man, as to say that because rapid recovery from the paralysis caused by cortical lesions takes place in the dog the affections caused by similar lesions in man is not due to the destruction of a cortical centre. The disappearance of paralysis of the limbs in the dog corresponds exactly to the disappearance of conjugate deviation of the eyes in man, and the explanation which suffices for the one will probably suffice for the other (§ 90). But even Goltz admits that some movements in the dog become more or less permanently paralysed. For instance, he may use his forepaw to drag bones and other morsels of food from under a table, and he may also be taught to perform special tricks with his paws ; all such special movements become more or less permanently lost after portions of the cortex have been removed. This shows that the purely voluntary actions are more or less permanently lost, while paralysis of the automatic actions concerned in ordinary locomotion rapidly disappears. Goltz found that the animals operated on could after a time be trained or educated to perform special actions with their paws, a fact which shows that a new organisation takes place more readily in the brain of the dog than in that of man, but it is quite probable that new structural arrangements may also take place to a certain extent in the brain of man after partial injury.

2. *Sensory Centres.*

It has already been seen that the centripetal fibres terminate amongst the cells of the second and third layers of the cortex without forming any direct connection with them, while the fibres of the pyramidal tract take origin in the axis-cylinder processes of the giant-cells of the internal portion of the third layer. It may, therefore, be suspected that the centripetal currents will pass in a much more diffused manner through the cortex than the centrifugal, just as it is found that the former pass in a more diffused manner through the spinal cord than the latter. It is not therefore likely that the sensory inlets are as definitely localised as the motor outlets.

Experiments by Stimulation.—On stimulating the angular gyrus Ferrier obtained various movements of the eye and associated movements of the head, and he regarded the phenomena observed as being "merely reflex movements on the excitation of subjective visual sensation." He, therefore, concluded that the angular gyrus and surrounding grey matter constituted the centre of vision. On somewhat similar grounds he placed the auditory centre in the superior temporo-sphenoidal convolution, the centres of taste and smell at the extremity of the temporo-sphenoidal lobe, and that of touch in the gyrus uncinatus and hippocampus major. But these experiments, although exceedingly interesting and important as being the first to break ground in a new territory, are by no means conclusive. Dr. Ferrier himself, indeed, did not rest satisfied with them, but proceeded to verify his hypotheses by the extirpation or destruction of the portions of the cortex which he supposed to be the sensory centres.

Experiments by Extirpation or Destruction of Sensory Centres.—The most remarkable result obtained by Ferrier in his first experiments was afforded by destruction of the angular gyrus. When the angular gyrus of the left hemisphere was destroyed, it was found that the animal was blind on the right eye soon after the operation, but recovered sight completely on the following day. In another case the angular gyri of both hemispheres were destroyed and the animal became completely blind in both eyes. In no case was any motor paralysis observed.

The admitted objections to these experiments are that Ferrier did not keep his animals alive a sufficiently long time to ascertain if a return of vision occurred. Goltz found in his experiments that when a considerable portion of the cortex of the brain was removed the animals, although not blind, manifested a peculiar imperfection of vision. The animal operated upon could use his sight in avoiding obstacles, but often failed to recognise his food, and appeared quite indifferent when threatened with the whip. He also found that recovery from this condition was possible, at least to a considerable extent, by means of educational exercises.

Munk believes again in the existence of a "visual area," situated in the occipital lobes, and of much larger extent than that of Ferrier. He maintains that removal of this area causes blindness, and that extirpation of small portions of it gives rise to blindness of localised areas of the retina. He believes that there are three visual spheres in the cortex of the occipital lobe corresponding to three visual areas in the retina. The external part of the retina of the left eye is connected with the external part of the cortical visual centre in the left hemisphere, while the internal and central portions of the retina of the right eye are respectively connected with the internal and central portions of the visual centre of the opposite or left hemisphere. He also thinks that the upper part of the retina is connected with the front, and the lower part with the posterior aspect of the visual centre of the opposite side. Removal of both visual centres causes, according to this observer, complete

or *absolute* blindness. Partial removal of these areas on the other hand gives rise to the visual defect called attention to by Goltz, in which the animal can see and avoid objects, but does not recognise his food as such. This Munk calls *psychical* blindness (Seelenlähmungen, Seelenblindheit). He finds that after a time the animals recover from psychical blindness, provided the whole visual area be not removed. He thinks that the recovery is due to a process by which there is a deposition of new visual experiences in the rest of the visual area. The physical part of the restoration might probably be spoken of with greater justice as the formation of new structural arrangements in the visual areas. Munk describes an auditory area, which however differs from that of Ferrier, and he regards the whole front part of the brain as forming a large "sensory" area, in which separate sensory centres may be distinguished.

FIG. 236.

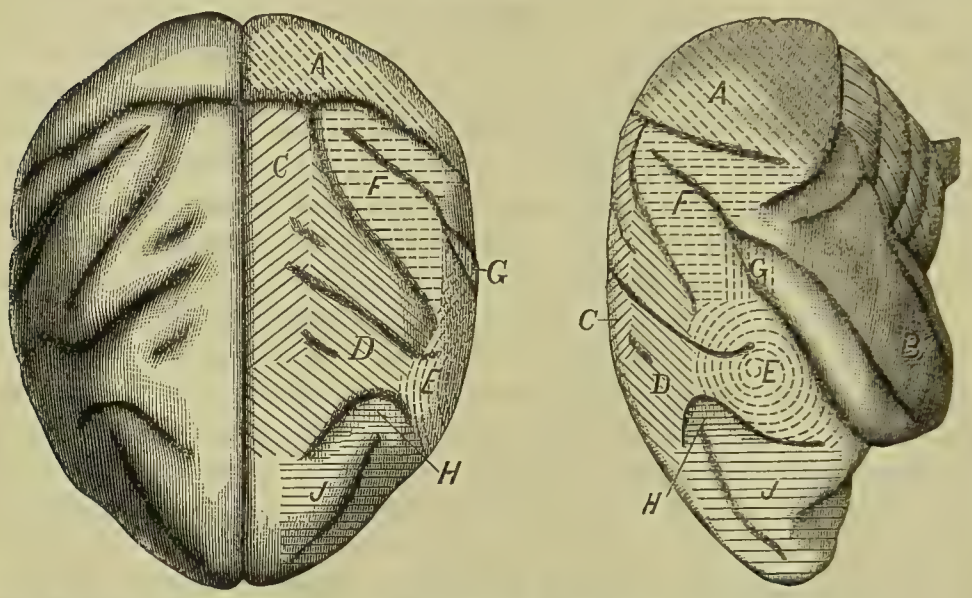


FIG. 236 (After Munk). *Upper Surface of the Brain of the Monkey.—Sensory Areas :* A, of the eyes; B, of the ears; C, of the sensibility of the lower extremity; D, Anterior extremity; E, Head; F, Ocular muscular apparatus; G, Region of ears; H, Neck; I, Body.

An elaborate paper on the cerebral visual centres was read before the physiological section at the meeting of the British Medical Association, at Cambridge, in August last, by Professors Ferrier and Gerald F. Yeo. Large portions of the brains of monkeys were removed, the operations being conducted antiseptically, so that there was a total absence of encephalitis. The following is an abstract of the chief results obtained :—

1. Removal of both occipital lobes did not cause any recognisable disturbance of vision, or other bodily or mental derangement, provided the lesion did not extend beyond the parieto-occipital fissure.

2. Complete destruction of one angular gyrus causes temporary loss of vision of one eye, lasting only a few hours. The restoration of vision is

not due to the integrity of the other angular gyrus. If both angular gyri be destroyed *simultaneously* total blindness ensues in both eyes, but does not last more than three days, although vision may be impaired for months. If the angular gyri be destroyed *successively*, several weeks elapsing between the operations, the animal sees quite well with both eyes in a few hours.

3. Simultaneous destruction of the angular gyrus and occipital lobe on one side causes evident loss of vision in both eyes towards the side opposite the lesion (hemiopia), but recovery from this condition takes place at the end of a week.

4. Destruction of the left angular gyrus (recovery), and subsequently of the right angular gyrus and occipital lobe, produces left hemiopia, from which the animal recovers in a fortnight.

5. Destruction of both occipital lobes, followed after a time by destruction of the left angular gyrus, causes transient blindness followed by indistinctness of vision of right eye, with subsequent complete recovery.

6. Destruction of both angular gyri and occipital lobes causes total and permanent blindness in both eyes, without any impairment of the other senses or of motor power.

3. *Præ-frontal or Ideational Area of the Cortex.*

Experiments by Stimulation.—Electrical irritation of the præ-frontal region of the cortex in the monkey causes no motor reaction (Ferrier).

Experiments by Extirpation.—Complete destruction of the præ-frontal lobes in the monkey causes no paralysis of motion and no sensory disturbance, but the character of the animal suffers great deterioration subsequently to the operation. "Removal or destruction by the cautery of the antero-frontal lobes," says Dr. Ferrier, "is not followed by any definite physiological results. The animals retain their appetites and instincts, and are capable of exhibiting emotional feeling. The sensory faculties—sight, hearing, touch, taste, and smell—remain unimpaired. The powers of voluntary motion are retained in their integrity, and there is little to indicate the presence of such an extensive lesion, or a removal of so large a part of the brain. And yet, notwithstanding this apparent absence of physiological symptoms, I could perceive a very decided alteration in the animal's character and behaviour, though it is difficult to state in precise terms the nature of the change. The animals operated on were selected on account of their intelligent character. After the operation, though they might seem to one who had not compared their present with the past fairly up to the average of monkey intelligence, they had undergone a considerable psychological alteration. Instead of, as before, being actively interested in their surroundings, and curiously prying into all that came within the field of their observation, they remained apathetic or dull, or dozed off to sleep, responding only to the sensations or impressions of the moment, or varying their listlessness with restless and purposeless wanderings to and fro. While not actually deprived of

intelligence, they had lost to all appearance the faculty of attentive and intelligent obeservation." The conclusions which Dr. Ferrier has drawn from his experiments on animals are fully borne out, as we shall subsequently see, by the results of diseases and injuries of the præ-frontal lobes in man. The whole evidence shows that, although destruction of these lobes is not followed by decided sensory or motor disturbances, yet that the later evolved (representative and re-representative) cognitions and emotions are seriously impaired.

§ 688. *Localisation of the Mechanisms which regulate the Fundamental and Accessory Actions.*

We have already seen that the fundamental portions of the convolutions of the motor area of the brain are found near the great longitudinal fissure, while the accessory portions of these convolutions are found in the convolutions of the operculum; and it may therefore be expected that the fundamental motor actions will be regulated from the former, and the accessory functions from the latter. Several lines of evidence converge in support of this view. The large giant-cells are found in the convolutions near the great longitudinal fissure, while these cells diminish in size as we descend towards the convolutions of the operculum. But we have already seen that the size of the motor ganglion cells of the anterior grey horns of the cord is determined by the size of the muscles whose movements they regulate more than by any other circumstance, and it is very likely that a similar relation exists between the giant-cells of the cortex and the muscles with which they are connected. But the fundamental actions are, as a rule, produced by the contractions of large muscles, such as those of the trunk and lower extremities, and consequently we may expect that they will be regulated by means of the large cells of the central convolutions near the great longitudinal fissure; while, on the other hand, the accessory actions are produced by small muscles, such as those of the hand, larynx, and face, and we may expect that they will be regulated through the smaller cells of the convolutions of the operculum. Again, the fibres of the pyramidal tract, which are medullated in a nine months embryo—the fundamental fibres—are connected with the central convolution near the great longitudinal fissure; while the non-medullated fibres—the accessory fibres of the tract—are connected with the convolutions of the operculum. The fibres which connect the posterior portion of the third frontal convolution with the internal capsule and crusta are not medullated before fourteen weeks after birth (Flechsig). We have already seen that a large proportion of the accessory fibres of the pyramidal tract terminate in the medulla oblongata, and in all probability the majority of them are concerned in regulating the special movements of articulation and facial expression. A glance at Dr. Ferrier's diagrams (*Figs.* 232 and 233) of the motor centres of the human brain shows that the movements of the trunk and lower extremities are regulated from the central convolutions near the great longitudinal fissure; that those of the

arms are regulated from the middle of the ascending frontal and parietal convolutions; and that those of the face, tongue, and hand are regulated from the convolutions of the operculum. The facts of development and of experimental physiology, therefore, concur to show that the fundamental actions are regulated from the central convolutions near the great longitudinal fissure, and the accessory functions from the convolutions of the operculum. It must also be remembered that the grey matter at the bottom of the fissures is developed subsequently to that of the summits, and consequently the former represents an organisation which has been superadded to the latter in the course of evolution. But the portion of the cortex of the Island of Reil which adjoins the convolutions of the operculum is the great area in which new structure is superadded to the motor region of the cortex. As the grey matter in the neighbourhood of the anterior perforated space increases in superficial extent, the external aspect of the cortex of the central lobe is thrust upwards and outwards so as to develop the convolutions of the operculum; and each addition of grey matter to the latter convolutions represents an additional complexity in the pre-existing structure corresponding to an additional complexity of previous muscular adjustments. Each increment which is added to the inferior extremities of the central convolutions by the upward growth of the cortex of the Island of Reil increases the length of the former; but as their upper extremities are prevented from moving freely upwards by the skull, their lower extremities are thrown into a fold, and consequently the depth of the sulcus which separates the Island of Reil from the convolutions of the operculum may be accepted as an indication of the degree of development of the accessory portion of the motor area of the cortex.

§ 689. *Localisation of the Cortical Centres of General and Special Sensations.*

We have seen that in the spinal cord the conducting paths of the common sensations passed directly into the posterior grey horn, through the middle of the fan formed by the fibres of the posterior roots on their entry into the cord; while, on the other hand, the conducting paths of the special cutaneous sensations are thrust inwards and outwards, so as to occupy positions outside the margins of the posterior horns. A somewhat similar process appears to take place during the development of the cortex in relation to the common and special cutaneous sensations and the special senses. According to the latest experiments of Ferrier and Munk the centre of vision—the most special of all the senses—is situated on the outer convex surface of the occipital lobe in the area of the terminal distribution of the posterior cerebral artery, while the centre of tactile sensation is situated in the *hippocampal* region, close to the root of the same artery. It is probable that the sensation of pain is too much diffused in the cortex to admit of any definite localisation. Both the auditory centre—the superior temporo-sphenoidal convolution—and the olfactory centre—the subiculum cornu Ammonis—although situated nearer the root of the artery than the visual centre, yet occupy positions

near the terminal distribution of some of the branches of the posterior cerebral artery, and certainly further removed from its root than the centre of tactile sensation.

§ 690. *Localisation of Function in the Præ-frontal Area of the Cortex.*

If the higher mental operations be carried on in the anterior area of the cortex, this region must contain the plexuses of cells and fibres, which, when excited, become the correlatives of the representative and re-representative cognitions and feelings. No progress has been made in localising the functions of this area of the cortex. It is, however, probable, that the later-acquired emotions and cognitions will be represented in the cortex by the grey matter in the bottom of the fissures, and by the grey matter of the convolutions of the orbital surface which adjoins the anterior perforated space and which are situated close to the root of the anterior cerebral artery. Pathological observation bears out the idea that disease of the cortex of the orbital surface produces much less mental disturbance than disease of the superior convex surface of the præ-frontal area. And this is only what might be expected if the former is developed at a later period than the latter. The convolutions of the orbital surface would then represent the later-acquired cognitions and emotions, and abolition of them would cause less mental disturbance than abolition of those which are earlier acquired but more fundamental. A man, for instance, may live what is regarded as a respectable life when he is destitute of all reverence, and is wholly incapable of doing an unselfish action, while the only self-restraint he places over his appetites and passions is that which the most calculating selfishness suggests. Yet reverential feeling, unselfishness in action, and self-restraint are the latest acquisitions in the development of the human mind. If, however, a man, instead of being lacking in reverential feeling, becomes openly profane, and instead of not being unselfish he commits deeds of violence in order to deprive others of their rightful property, and if instead of curbing his passions even by a calculating selfishness he gratifies them without shame and regardless of consequences, it is evident that a lower stratum of mental degradation has been reached, and the portion of the cortex now diseased is a more fundamental one, which must have been developed at an earlier period than that which was diseased in the first instance.

CHAPTER II.

MORBID ANATOMY AND CLASSIFICATION OF THE DISEASES OF THE ENCEPHALON.

(I.)—MORBID ANATOMY OF THE ENCEPHALON.

THE operation of the law of evolution having been already traced in the construction of the brain, we must now endeavour to trace the action of the law of dissolution in the breaking down of structure the result of disease.

§ 691. *Histological Morbid Changes.*—The histological changes which occur in the tissues of the brain during diseased processes are essentially the same as those which have already been described in the case of the spinal cord (§ 387), and it is, therefore, unnecessary to repeat the description.

§ 692. *Morbid Alterations of the Circulation within the Cranium.*—It was first pointed out by the second Monroe that the circulation within the cranium differs from that of other parts of the body. The cranium forms a bony case, capable of resisting the atmospheric pressure, and no substance can be dislodged from it without some equivalent taking its place; while, on the other hand, no substance can be added to the contents of the cranium without dislodging an equivalent bulk of some other substance. This opinion was experimentally tested by Kellie, and defended by Abercrombie, Reid, and Watson. Dr. Burrows endeavoured to combat this opinion, but he only showed, what was never denied, that the quantity of blood in the brain could be increased or diminished by various circumstances. The doctrine of Monroe simply asserts that if the quantity of blood in the cavity of the cranium be increased,

some other fluid must be dislodged; while if the quantity of blood be diminished, some other fluid must fill up the vacant space. The quantity of blood in the brain can undoubtedly be increased or diminished, but this can only take place by a corresponding diminution or increase in the quantity of the cerebro-spinal fluid and of the fluid contained in the perivascular lymph spaces. When, however, the intracranial pressure is rendered still greater, as by extravasation of blood from rupture of a blood-vessel, room is made for the substance superadded to the contents of the cranium by a certain quantity of blood being squeezed out of the intracranial arteries, veins, and sinuses, in addition to the displacement of the cerebro-spinal fluid. The circulation within the cranium is liable to be disordered by occlusion or rupture of one or more of the intracephalic vessels, but the reader is referred to the sections on embolism, thrombosis, and cerebral hæmorrhage for detailed descriptions of these processes.

Tumours.—The growth of intracranial tumours of all kinds must necessarily be attended by great disturbance of the cerebral circulation. In order to make room for the increasing bulk of the tumours the cerebro-spinal fluid, as well as the fluid of the perivascular lymph spaces, is first squeezed out, the blood is then compressed from the intracranial blood-vessels, so that the whole brain is rendered anæmic.

§ 693. *Secondary Degenerations.*—Long-standing hæmorrhagic and other diseased foci give rise to various *secondary changes* not only in the surrounding tissues, but also in distant parts. These changes are of two kinds: firstly, those which involve the entire mass of the brain; and secondly, those limited to certain tracts of conducting fibres which are interrupted in their course by the hæmorrhage.

(1) *General Atrophy.*—With regard to the former of these kinds, it is found that the brain frequently undergoes, even after an insignificant hæmorrhage, a slow and general atrophy which occasionally affects both hemispheres. This condition is especially common after extravasations into the cortex. A persistent alteration of one hemisphere of the brain may after a time induce atrophy of the opposite hemisphere of the cerebellum.

(2) *Systemic Degeneration.*—As has been frequently stated, whenever the fibres of the pyramidal tract are injured in any part of their course from their origin in the cortex of the brain down to their termination in the spinal cord, the portions below the seat of injury undergo descending degeneration.

A focal lesion, limited to the middle third of the posterior segment of the internal capsule (*Fig.* 237, F), is followed by descending degeneration of the fibres of the middle third of the crusta (*Fig.* 238, *L*), and of a portion of the longitudinal fibres of the pons and anterior pyramid of the medulla.

In the lower end of the medulla the greater part of the degenerated fibres cross over to the lateral column of the opposite side of the cord (*Fig.* 239, A), while some of them pass down the column of Türck of the same side (*Fig.* 239, B). The course pursued by the secondary degeneration in the case

FIG. 237.

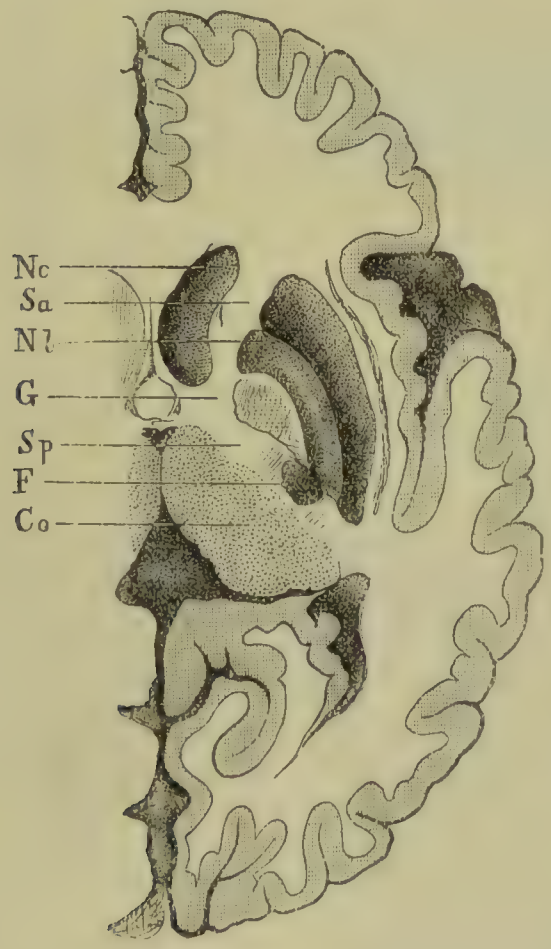


FIG. 237 (Charcot). *Horizontal Section of the Right Hemisphere parallel with the Fissure of Sylvius.*—Nc, Caudate nucleus; Sa, Anterior segment of the internal capsule; Nl, Lenticular nucleus; G, Knee of the internal capsule; Sp, Posterior segment of the internal capsule; Co, Optic thalamus; F, A focal lesion in the middle third of the posterior part of the internal capsule.

FIG. 238.

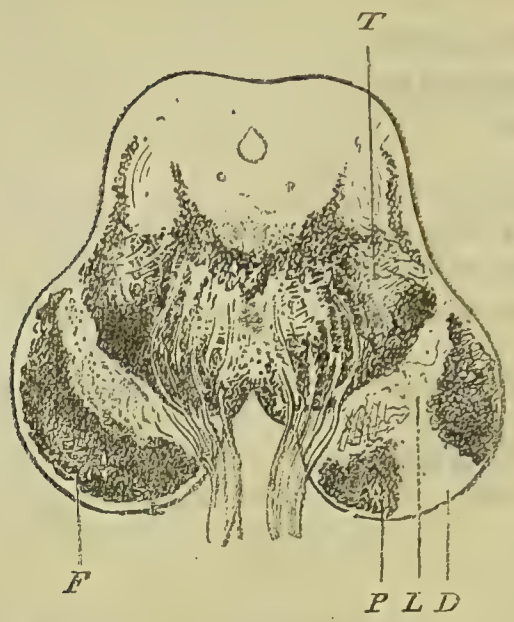


FIG. 238 (Charcot). *Horizontal Section of the Crura Cerebri in a case of Secondary Degeneration.*—*T*, Tegmentum; *F*, Crusta of the healthy side; *L*, Locus niger; *D*, The degenerated fibres, occupying about the middle third of the crusta; *P*, The fibres which undergo secondary degeneration only when the fibres of the anterior segment and the knee of the internal capsule are diseased.

FIG. 239.

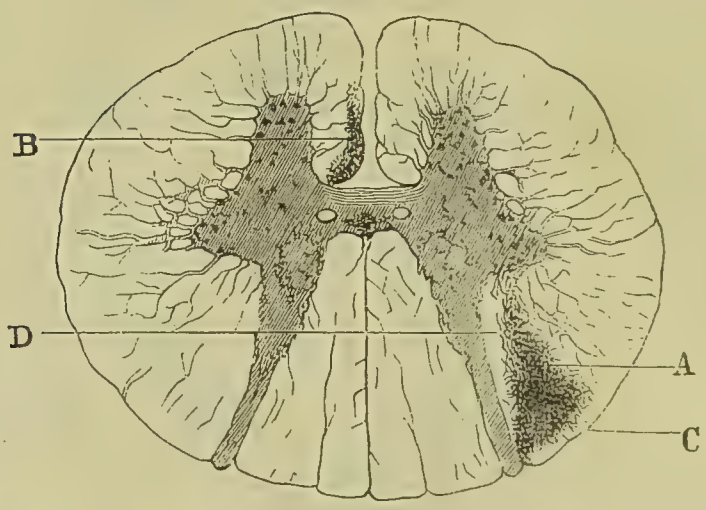


FIG. 239 (Charcot). *Transverse Section of the Cervical Region of the Spinal Cord, from a case of lesion of the motor area of the cortex of the opposite hemisphere.*—A, Degeneration of the pyramidal tract; B, Degeneration of the direct fibres; C, Direct cerebellar tract; D, Intermediate region between the posterior grey horn and the pyramidal tract, the fibres of which do not undergo descending degeneration.

just described corresponds to that of the fundamental fibres of the pyramidal tract during their development (*Figs.* 223 and 224). A case has been described by Brissaud in which, along with extensive recent softening of one hemisphere, an old focus of softening was observed limited exactly to the knee of the internal capsule (*Fig.* 240, D). A streak of degeneration

FIG. 240.

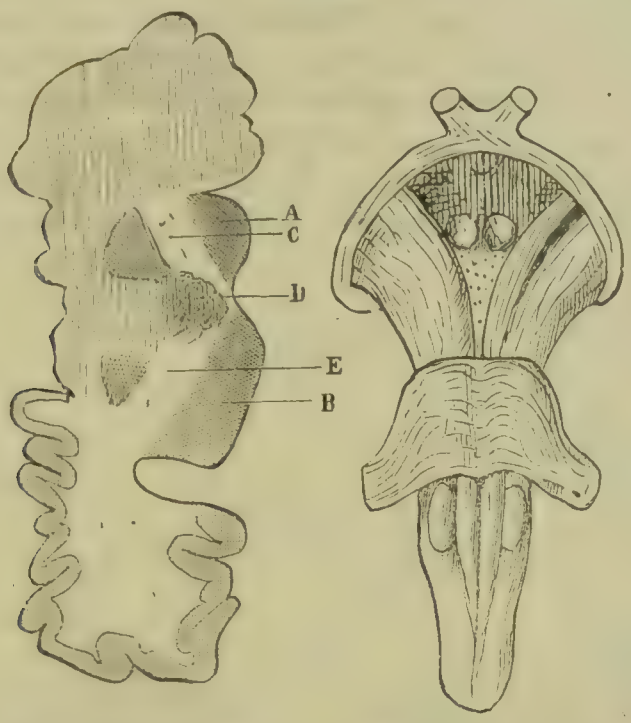


FIG. 240 (Brissaud). *Recent Softening of the Frontal Lobe, the Island of Reil, and Middle Third of the Lenticular Nucleus.*—D, Old focus of softening occupying the knee of the internal capsule; A, Caudate nucleus; B, Optic thalamus; C, Anterior, and E, Posterior division of the internal capsule.

was observed lying between the internal and middle thirds of the crusta, being the anterior portion of the area which has already been described as the mixed area of medullated and non-medullated fibres in a nine months embryo (*Fig.* 223). According to Brissaud degeneration occurs in the knee of the internal capsule in cases of long-standing aphasia.

Another important case has been observed by Brissaud in which an old focus of softening was found in the anterior half of the lenticular nucleus, destroying also the anterior segment of the internal capsule (*Fig.* 241, F). A streak of degeneration was observed in the internal third of the crusta (*Fig.* 241, P), but all the fibres of this area were not implicated in the degeneration, a small bundle of the innermost fibres remaining normal. The degenerated fibres in this case corresponded very nearly to those which we have described as the accessory fibres of the tract. Degeneration of the internal tract of the crusta, according to Brissaud, appears to be always connected with intellectual disorders.

The following bundles of fibres may, therefore, be distinguished in the internal capsule (Brissaud) :—

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

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

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

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

FIG. 241.

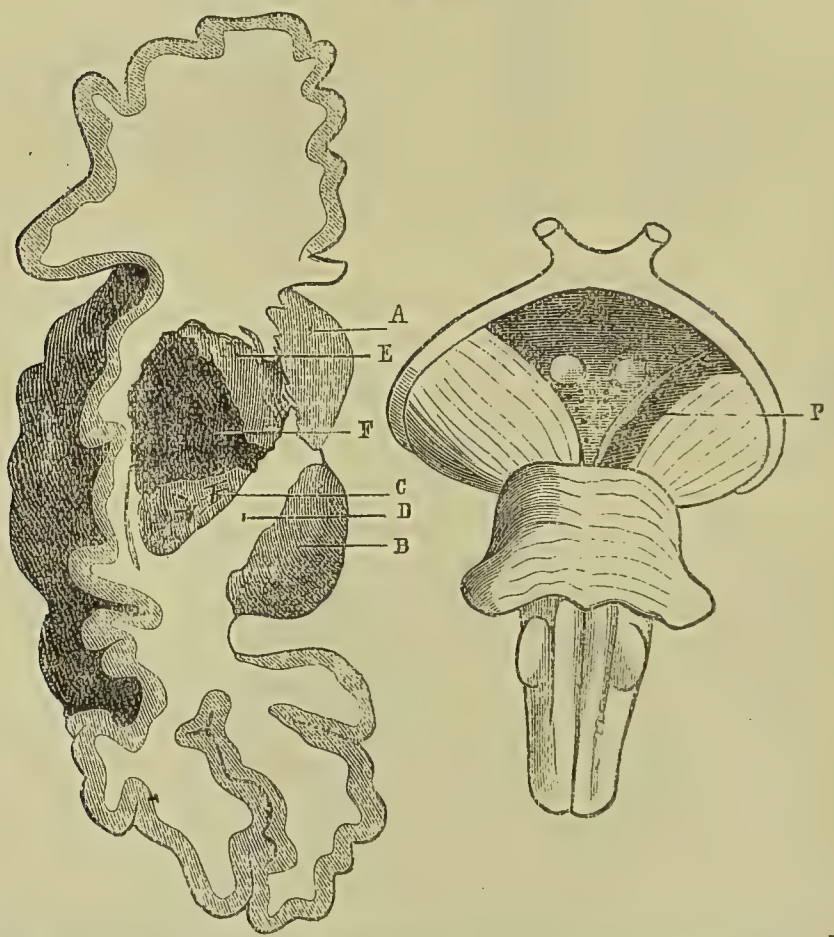


FIG. 241 (Brissaud).—A, Caudate nucleus; B, Optic thalamus; C, Posterior and healthy part of the lenticular nucleus; D, Posterior segment of the internal capsule; E, Lesion of the anterior segment of the capsule; F, Cyst presenting the form of the lenticular nucleus; P, Degeneration of the internal fibres of the crusta.

§ 694. *Congenital Malformations of the Skull and Brain.*

(1) *Anencephalia.*—In this condition the upper portion of the skull and brain is entirely absent. This condition is sometimes associated with amyelia, a condition in which the vertebral column remains unclosed, and the spinal cord is wanting (Förster).

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

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

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

(5) *Hydrencephalocele.*—In this condition the bones of the skull are deficient, an opening being left, generally at one of the fontanelles, through which a soft, fluctuating tumour projects. The tumour contains fluid, and can generally be emptied by steady pressure. The walls of the tumour consist of the soft coverings of the skull, and the distended membranes of the brain. The tumour communicates with the general ventricular cavity of the brain.

(6) *Encephalocele.*—The bones of the skull are again deficient at some part in this condition, but through the opening formed a portion of the brain itself projects, forming a broad, flat, solid tumour. The tumour frequently occupies the forehead, orbit, or side of the nose.

§ 695. *The Law of Dissolution.*—Although the law of dissolution is probably destined at some future time to throw more light on the morbid anatomy of the brain than on that of any other organ of the body, yet it must be admitted that up to the present very little progress has been made in this direction. The law has been applied by Dr. Hughlings-Jackson with much ingenuity and success to the interpretation of disordered cerebral functions, but it has yet to be applied to the elucidation of morbid cerebral structures. The histological elements of the brain doubtless conform to this law in their degenerations in a manner similar to that already described with regard to the histological elements of the spinal cord. And even when the disease is diffused in the neuroglia the small cells and thin fibres of the accessory portion of the brain must suffer injury more readily than the large cells and thick fibres of the fundamental portion. This *a priori* necessity has not, however, been verified as yet by *a posteriori* observations. Even in such a coarse lesion as that caused by occlusion of one of the cerebral arteries—say the inferior frontal branch of the

left Sylvian artery—the operation of this law may probably be traced. If, as we have already endeavoured to prove, the later-formed cells and fibres of Broca's convolution lie near the root of this artery, while the earlier-formed cells and fibres are thrust upwards and forwards towards the terminal twigs of the vessel, it is manifest that the earlier is in a much more favourable position than the later developed portion to obtain nourishment from the neighbouring vascular territories. But this conclusion, although there is much probability in its favour, has not yet been confirmed by careful dissection. These brief and imperfect remarks are all that we feel justified in making, in the present state of our knowledge, with regard to the applicability of the law of dissolution to the structural alterations produced by disease in the brain.

(II.)—CLASSIFICATION OF THE DISEASES OF THE ENCEPHALON.

It is impossible to give a full and scientific classification of the diseases of the encephalon, inasmuch as a large proportion of them are beyond the scope of this work. It is not our intention to enter upon the discussion of the wide class of diseases of the brain comprised under the general term insanity, and yet no classification of the diseases of the encephalon can be considered satisfactory which does not comprise them. In endeavouring to classify the diseases which remain, we shall proceed on the principle of considering first those which give rise to the fewest, and leaving to the last those that occasion the most numerous and complicated symptoms. Now as disease of the membranes can hardly ever exist without producing secondary disease over a large area of the surface of the brain, the symptoms characterising the former may be expected to be on the whole more complicated than those of the latter, and consequently the diseases of the encephalon will be considered prior to those of the membranes. Of the diseases of the encephalon, the lesions which give rise to the least complicated symptoms are the *focal*, and those which give rise to the most complicated symptoms are the *diffused lesions*. It is known that a focal lesion, as a tumour, may by increasing the intracranial pressure and in other ways give rise to diffused effects,

and that a diffused disease like encephalitis may by terminating in abscess occasion local symptoms. The division into *focal* and *diffused* lesions is, therefore, not a scientific but a practical distinction, and must be judged entirely from a practical standpoint. The symptoms caused by focal diseases depend partly upon the *nature* and partly upon the *locality* of the lesion. Cerebral hæmorrhage, for instance, occasions a grouping of symptoms which enables the affection to be readily distinguished from the symptoms produced by the slow growth of an intracranial tumour; yet the symptoms caused by destruction of a certain portion of the internal capsule, for instance, is the same whether the injury be caused by hæmorrhage or by the growth of a tumour. The focal diseases, therefore, admit of consideration under two aspects: (I.) according to the symptoms common to the pathological state in general; and (II.), according to the special functions of the region affected. With these few remarks the following classification may be allowed to explain itself:—

A. *Diseases of the Encephalon.*

I. Focal diseases.

(I.) General consideration of focal diseases, according to the *nature* of the lesion.

1. Occlusion of intracranial vessels.
2. Intracranial hæmorrhage.
3. Intracranial tumours.

(II.) Special consideration of focal diseases, according to the *localisation* of the lesion.

1. Affections of peduncular fibres and internal capsule.
   a. Affections of the pyramidal tract.
      (i.) Hemiplegia.
      (ii.) Hemispasm.
   b. Affections of the sensory peduncular fibres and optic radiations of Gratiolet.
      (i.) Hemianæsthesia.
2. Lesions of the cortex of the brain and of the subjacent portion of the centrum ovale.
   a. Lesions in the area of the middle cerebral artery.
      (i.) Unilateral convulsions, and monospasms.
      (ii.) Monoplegiæ.
      (iii.) Cortical affections of speech.

b. Lesions in the area of the posterior cerebral artery.
c. Lesions in the area of the anterior cerebral artery.
3. Lesions in the basal ganglia, external capsule, claustrum, and base of the brain.
4. Lesions localised in the structures situated below the tentorium.
a. Lesions in the pons and peduncles of the cerebrum.
b. Lesions in the peduncles of the cerebellum.
c. Lesions in the cerebellum.

II. Diffused diseases of the encephalon.

(I.) Anæmia and hyperæmia of the brain.
(II.) Atrophy and hypertrophy of the brain.
(III.) Shock, and concussion of the brain.
(IV.) Encephalitis.

1. General encephalitis.
2. Partial encephalitis.
a. Acute encephalitis, complicating affections of the petrous portion of the temporal and other bones of the skull.
b. Acute pyæmic encephalitis.
c. Encephalitis secondary to other cerebral lesions.
d. Chronic abscess of the brain.

B. *Diseases of the Membranes of the Brain.*

I. Diseases of the dura mater.

(I.) External pachymeningitis.
(II.) Internal pachymeningitis.

II Diseases of the pia mater.

Acute inflammation of the pia mater.

1. Leptomeningitis Infantum.
2. Tubercular meningitis.
3. Meningitis of the base of the brain.
4. Meningitis of the convexity of the brain.
5. Metastatic meningitis.
6. Traumatic meningitis.

CHAPTER III.

(I.) GENERAL CONSIDERATION OF FOCAL DISEASES, ACCORDING TO THE NATURE OF THE LESION.

1. OCCLUSION OF THE INTRACRANIAL VESSELS.

THE arteries, veins, sinuses, and capillaries of the brain are liable to become occluded, a circumstance which gives rise to various pathological changes. The occluding body may be formed on the spot, constituting thrombosis; or may be carried from distant parts, constituting embolism.

(*a*) *Occlusion of the Cerebral Arteries.*

§ 696. *Etiology.—Emboli* consist of fibrinous masses washed away from the left cavities of the heart, the aortic and mitral valves, the arch of the aorta, and occasionally from the pulmonary veins. In cases of carcinoma of the lungs, a cancerous mass may possibly be carried from the pulmonary veins and pass into the cerebral vessels.

Arterial thrombosis is determined partly by general causes tending to diminish the force of the heart's action and to alter the quality of the blood, and in part by local degenerations of the coats of the vessels themselves. During convalescence from acute diseases and other exhausting processes, the heart is weak, and the blood becomes so altered in quality that it is specially prone to coagulate. This process is, of course, much favoured if the heart have undergone dilatation without proportionate hypertrophy. The local causes of thrombosis are alterations of the walls of the arteries, whereby their lining membrane is roughened and their calibre is narrowed. The coagulation of fibrine is favoured by changes in the walls and internal surface of the vessel, such as those due to atheromatous and calcareous

degenerations. Thrombosis is particularly liable to occur when general causes and local degeneration act together.

Thrombosis may take place in any of the arteries at the base of the brain, or in several of them at the same time. A thrombus not unfrequently forms in one of the internal carotid arteries, and the clot then often extends into the middle and anterior cerebral arteries of the same side, and sometimes even into the posterior communicating and the posterior cerebral artery.

Of the predisposing causes of cerebral thrombosis age is probably the most important. Thrombosis occurs more frequently in advanced age, owing to the degenerative changes in the vessels, although it may occur at all ages. Embolism, on the other hand, is met with in relatively young persons, although it may also occur in persons of advanced years.

§ 697. *Symptoms.*—The symptoms which characterise the later stages of thrombosis and embolism are the same, but those of the early period differ considerably.

The *symptoms of embolism* are very sudden in their onset, and are not preceded by any premonitory signs. The patient is suddenly attacked with dizziness, utters an involuntary cry, or complains momentarily of headache, and then almost immediately loses consciousness. The symptoms occasioned by embolism of a cerebral artery are in their general characters and mode of onset almost identical with those of cerebral hæmorrhage, but the unconsciousness caused by embolism is, as a rule, more transient than that produced by hæmorrhage.

In many cases there is no coma, but only some dizziness or slight confusion of mind for a minute or two, along with the sudden advent of paralysis. The attack is sometimes ushered in by epileptiform convulsions, which may sometimes be general like an ordinary epileptic attack, but are at other times limited to one-half of the body, to one extremity, or one-half of the face.

When general convulsions are present they occur simultaneously with the loss of consciousness, and are followed by paralysis immediately, while unilateral and partial convulsions may recur repeatedly before paralysis is fully established. In some cases the attack is accompanied by vomiting, and Hammond reports a case in which active delirium, and another in which

hallucinations and delusions were present for some hours after a sudden attack of hemiplegia. The presence of disease of the aortic and mitral valves in Hammond's cases rendered the diagnosis of embolism very probable.

In many cases sudden speechlessness, a condition which will be subsequently described as *aphasia*, constitutes the only symptom of the affection, and in these cases the symptom may disappear in a few days when collateral circulation is established. As a rule, however, the aphasia is associated with right-sided hemiplegia, which possesses the same general characters as that which results from hæmorrhage into the lenticular nucleus and neighbouring parts. The right side is more frequently affected with paralysis than the left, owing to the greater liability of the left middle cerebral artery to be affected by embolism. The state of the pupils during the onset of the attack has been variously described, and it probably varies in different cases. Erlenmeyer states that the pupils remain sensitive, being neither contracted nor dilated, while Hammond has found dilatation, contraction, or irregularity.

The *symptoms of thrombosis* are, as a rule, more gradual in their development than those of embolus. The more usual premonitory symptoms of thrombosis of a cerebral vessel consist of headache, which may be diffused through the entire head or referred to the neighbourhood of the morbid process (Hammond), dizziness, and a sense of general confusion. The patient may complain of numbness, coldness, or formication in one extremity or throughout the distribution of one nerve or in the entire half of the body. At times there may be considerable mental disturbances, and failure of memory is often a marked symptom. Motor disturbances are usually of the nature of more or less extensive paresis, but occasionally the loss of motor power is preceded by slight convulsive movements. Paralysis may occasionally supervene suddenly, but, as a rule, its development is slow and gradual, its progress being marked by successive remissions and exacerbations. This mode of development appears to be due to the fact that thrombosis has a tendency to extend backwards and to implicate more and more of the arterial branch, in whose ultimate twigs the process may have first begun. The duration of the prodromal stage

may vary from a few hours to several months, and occasionally apoplectic symptoms may come on suddenly, as in embolism. When once the vessel has become completely occluded, the further progress of thrombosis is like that of embolism in the same situation.

When softening occurs the temperature, according to Bourneville, rises on the second or third day after the attack, and in two or three days may be as high as 40° C. (104° F.). In a few days longer the temperature sinks rapidly, its decline being more rapid than that which takes place after the period of reaction in cases of hæmorrhage. When once softening has become thoroughly established the symptoms are those which result from localised cerebral disease generally, and indeed the symptoms of softening and of hæmorrhage when regarded as localised diseases are often identical.

Contractures of the paralysed limbs are not so common in cases of occlusion of vessels as in cases of hæmorrhage, but they occur sufficiently often to make their presence or absence destitute of diagnostic significance.

The sensory, trophic, and vaso-motor disturbances, as well as the affections of the special senses, with the exception of vision, are the same as those which occur in connection with cerebral hæmorrhages. In some cases of embolism the ophthalmic artery has become occluded, giving rise to sudden amaurosis. On ophthalmoscopic examination the arteries of the retina are seen to be empty; they appear like fine threads, but still retain their red colour. The veins are not much diminished in size, and are filled with dark blood. The retina presents a greyish or white opacity which is most marked around the macula lutea, but the fovea centralis remains of a bright red colour, forming a marked contrast with the pallor of the surrounding portion of the retina.

Various disturbances of the mental faculties may result from embolic softening. Probably the most interesting of these is aphasia, which results from embolism of the middle cerebral artery generally of the left side, but this condition will be discussed at greater length in a future chapter.

In some cases of occlusion of cerebral arteries the symptoms begin to improve at an early period, and the patient may

ultimately recover completely. In these cases it is evident that the collateral circulation has been established before softening has commenced. In other cases the patient, after partial or complete recovery, is attacked again with embolism, and there may be a second recovery. In some cases of thrombosis the first symptoms may be of moderate severity, and may afterwards become by sudden accessions more and more severe.

In some few cases death may follow immediately, but as a rule it is not so sudden as in hæmorrhage.

Diseases of the mitral or aortic valves, aortic aneurism, ulcerative endocarditis, and inflammatory or syphilitic affections of the muscular substance of the heart are the complications usually met with. In cases of thrombosis evidences of degeneration of the vascular system can usually be detected in the radial and other arteries. Important symptoms may arise from embolism in the spleen, the kidneys, and the arteries of the extremities.

§ 698. *Diagnosis.*—The problem of diagnosis is to distinguish cerebral embolism, thrombosis, and hæmorrhage from one another. This must be done, not so much by means of the cerebral as of the associated symptoms.

When sudden hemiplegia occurs in a young or middle-aged person who is suffering from valvular disease of the heart or aneurism, the symptoms are in all probability due to embolism. The probability of embolism of a cerebral artery is rendered still greater if there be a history of previous seizures in the brain or other organs. Right-sided hemiplegia, with aphasia, results more frequently from embolism of the left middle cerebral artery than from any other cause, and consequently in such cases the presumption is always in favour of occlusion of the vessel rather than hæmorrhage, provided there be the necessary conditions for its occurrence. There are no absolute means of distinguishing between hæmorrhage and thrombosis, and it is needless to discuss the various diagnostic signs which have from time to time been proposed.

§ 699. *Morbid Anatomy.*—Embolism affects certain vessels with special frequency. The mode of origin of the left carotid

directly from the arch of the aorta, and the angle at which it leaves the arch, very much favour emboli being carried into it. These emboli usually pass the circle of Willis and make their way into the left middle cerebral artery, which is the direct continuation of the internal carotid, and, consequently, this artery is more frequently occluded by an embolus than any other vessel of the brain.

Thrombosis does not appear to have a special preference for any one artery. The middle and posterior cerebral, and vertebral arteries are equally liable to be occluded by thrombosis.

When one of the cerebral arteries—the left middle cerebral artery, for example—is obstructed close to the circle of Willis, the circulation through the nutrient arteries supplied by it to the basal ganglia is arrested, and as these are terminal arteries rapid softening occurs. When one of the vessels of the brain is obstructed on the cardiac side of the circle of Willis, the free anastomosis of the latter re-establishes the circulation so quickly that no pathological changes occur in the brain. If, again, the embolus be carried forwards past the basal portion to the arterial system of the cortex, it is quite possible that the free anastomosis of the latter may prevent decided pathological changes from taking place. In many cases, however, a certain amount of softening does occur under such circumstances, because the anastomosis is not always so free as to compensate for the blocking up of a large branch of the artery. When the embolus is lodged in one of the terminal arteries of the basal arterial system softening always occurs, owing to the absence of anastomosis with neighbouring arteries.

The first effect produced by occlusion of a terminal artery is œdema of the part supplied by it. The venules and arterioles of the part are imperfectly nourished so that their walls dilate and frequently rupture, giving rise to hyperæmia attended by œdematous swelling and hæmorrhage. The tissues, not being supplied with nourishment, break down and undergo softening. When the softened tissues become mixed with extravasated blood, they give rise to *red softening*. The hyperæmia and hemorrhage may fail to occur, and then simple necrobiosis results from the occlusion of the vessel, giving rise to a softened mass of a yellowish-white or white colour. These changes generally begin in the course of the second twenty-four hours after the obstruction has occurred, although cases are reported in which the consistence of the brain tissue was normal after the lapse of two days.

Microscopic examination reveals the presence of a large number of red blood corpuscles, which is the only abnormal appearance observed during the first twenty-four hours. At a later period the nerve elements undergo gradual degeneration. The most prominent microscopic peculiarity consists of granular corpuscles, which are probably derived from degeneration of neuroglia and ganglion cells of the grey substance, and various other sources.

Experimental Investigations.—The first experimental researches with respect to the embolic process was undertaken by Virchow, and great additional light has been thrown upon the subject by the important experimental and microscopic investigations of Cohnheim. Panum studied experimentally the results of occlusion of cerebral vessels with the view of determining the manner in which death is caused. B. Cohn investigated experimentally various clinical and anatomical points; Feltz studied the results of capillary embolism; while Prévost and Cotard made a series of experiments with the view of determining the relation of occlusion of cerebral vessels to softening.

§ 700. *Morbid Physiology.*—The most difficult problem to solve with respect to the morbid physiology of the affection is how occlusion of only one of the cerebral arteries produces loss of consciousness. Brown-Séquard has recently dwelt upon the fact that local lesions exert an influence over remote parts of the nervous system, and the sudden arrest in the circulation in one of the arteries of the brain is likely to produce widely-spread effects. Heubner and Duret have shown that although the abundant anastomoses between the arteries of the cortex after a time establish a collateral circulation, yet at the moment of obstruction great disturbances of the circulation and marked changes in pressure may occur in and around the implicated region. We have already seen that sudden deprivation of nourishment increases the irritability of nerve fibres, and it is probable that the abrupt arrest of the arterial circulation induces a powerful outgoing discharge from the cortex. That this occurs in certain cases is undoubted, inasmuch as the onset of the attack is marked by general convulsions. A powerful discharge of this kind would be followed by exhaustion, and temporary loss of function, or in other words the attack would be characterised by loss of consciousness. In those cases in which there is an absence of convulsions the cortical discharges may be supposed to neutralise one another in the nervous system without producing their usual visible effects.

§ 701. *Prognosis.* — Both embolism and thrombosis are always serious affections. When embolism occurs in a young person recovery from the immediate effects may be rapid and complete, but the underlying affection to which the attack was due will still be present and may cause a similar attack in the future or give rise to other grave symptoms. Thrombosis is usually associated with advanced age, enfeeblement of the heart's action, and degeneration of arteries, and during the attack there is great danger, however slight the symptoms may at first appear, that the occlusion will become more and more extensive.

§ 702. *Treatment.* — Prophylactic measures can only be adopted when premonitory symptoms are present for a long time in connection with a slowly-forming thrombosis. In such cases the heart should, according to theory, be stimulated by digitalis, ammonia, and alcoholic stimulants; but since it is impossible to diagnose this condition during life from hæmorrhage, it will be better to be content with adopting the same treatment as that recommended for hæmorrhage. During the stage of coma also the same means should be used as in hæmorrhage.

(*b*) *Thrombosis of the Cerebral Sinuses.*

§ 703. *History.*—Special attention was first directed to the subject of thrombosis of the cerebral sinuses by the observations of Tonnelé. Many valuable clinical observations with regard to the disease were made by Puchelt, and the attention of Lebert was also directed to it. The treatises of Von Dusch, B. Cohn, and of Lancereaux helped greatly to extend and to systematise our knowledge with respect to this thrombosis; and in more recent times our knowledge has been further increased by the labours of Gerhardt, Griesinger, Corazza, Heubner, and Hugnénin.

§ 704. *Etiology.*—Thrombosis of the sinuses may be divided into two groups: the first comprising the cases which arise in the absence of any affection of the walls of the veins, and the second those which originate from phlebitis.

The cases of the first group arise in conditions of *marasmus*, in which the quality of the blood is altered and the circulation enfeebled. Under such circumstances coagulation of the blood is specially prone to occur in the sinuses, inasmuch as they are

rigid tubes and incapable of collapsing; they are also destitute of muscular walls, and are traversed by bands of connective tissue.

Thrombosis of the sinuses from marasmus is particularly apt to occur in children, especially during the first six months of life, when they are liable to suffer from collapse induced by severe diarrhœa. It also occurs in adults, in consequence of profuse suppuration, cancer, senile marasmus, and other conditions of debility. This form of thrombosis occurs with special frequency in the longitudinal and lateral sinuses. Obstruction to the return of the venous blood towards the heart increases the liability to the formation of thrombosis of the sinuses, but it is not likely that venous stasis can give rise to it in the absence of other favouring conditions.

The second group of thromboses is caused by inflammation of the sinuses, the result generally, probably always, of disease or injury of the cranial bones. Caries of the petrous portion of the temporal bone is by far the most common cause of inflammation of the sinuses; the lateral and petrosal sinuses, which lie in the vicinity of the temporal bone, are then particularly liable to be affected, although the process may implicate the circular and cavernous sinuses as well as the upper part of the internal jugular vein. In most cases a real phlebitis is induced, followed by the formation of purulent thrombi. Thrombosis of the sinuses also frequently follows blows on the head, or inflammatory conditions of the scalp and cranial bones. Erysipelas of the head and face, and furunculus of the face, especially of the upper lip and forehead, not unfrequently give rise to thrombosis of the sinuses. Cohn observed a case in which suppurative phlebitis of the cavernous sinuses occurred in connection with purulent inflammation of the deep muscles of the neck.

§ 705. *Symptoms.*—The symptoms of thrombosis of the cerebral sinuses are generally marked by complicating diseases, so that it is rarely possible to diagnose the affection during life. The symptoms also vary greatly, both according to the seat of the occlusion and according as the thrombosis is or is not the result of phlebitis.

Thrombosis of the sinuses in children almost always arises during the marasmus, caused by exhausting diarrhœa, and the symptoms produced are the same as those of cerebral anæmia, being such as Dr. Marshall Hall described under the name of hydrencephaloid disease. In addition to the collapse, somnolence, and coma of pure cerebral anæmia, motor disorders, as convulsions or paralysis, are generally present. Rigidity of the muscles of the neck, sometimes also of those of the back and even of the limbs, occasionally nystagmus, strabismus, ptosis, and paresis of the facial muscles have been observed.

Thrombosis of the sinuses resulting from marasmus in adults gives rise to very various and indefinite symptoms, and at times a slight degree of apathy and general depression are the only symptoms observed. The patient at the outset may complain of headache, nausea, and vomiting, but these soon give place to coma, while in a few cases loss of consciousness may be preceded by delirium, which may assume a maniacal character. The condition of the pupils is variable.

Motor disturbances are usually present, the most usual being strabismus, trismus, contractures which may involve one-half of the body, or both legs and both arms, tremors, and epileptiform convulsions, either limited to one or involving the four extremities. The motor disorders may assume the form of paresis or paralysis, which may be limited to the facial nerve or to the motor oculi, or may involve one-half or both sides of the body. At other times both paralysis and convulsions may be associated, one extremity being the seat of contracture and the other of paralysis. These symptoms may, however, be present in cases of cerebral anæmia or of venous hyperæmia of the brain.

A valuable sign of the disease is sometimes afforded by swelling of the veins outside the skull which are in communication with the obstructed sinus. The superior longitudinal sinus, for instance, communicates directly with the veins of the nasal cavities and with those on the upper surface of the skull. The occurrence of epistaxis, therefore, favours the idea of obstruction of this sinus, and in children the presence of distended vessels running to the anterior fontanelle from the neighbourhood of the temples and ears on both sides of the

head also favours the same view. Cyanosis of the face limited to the part supplied by the anterior facial veins is also, according to Gerhardt, of diagnostic significance.

The lateral sinus communicates with a small vein which traverses the mastoid process, and in thrombosis of the sinus localised œdema behind the ear may make its appearance. This sign is occasionally valuable, but is rarely met with. Simultaneous occlusion of both lateral sinuses gives rise to the same symptoms as occlusion of the superior longitudinal sinus.

The cavernous sinus communicates with the ophthalmic veins, and in thrombosis of this sinus venous hyperæmia of the fundus oculi has been observed, as well as œdema of the eyelids and conjunctiva and prominence of the eyeballs, due to congestion of the retrobulbar veins and of the frontal vein. Paralysis of the motor nerves of the eye, trigeminal neuralgia, and neuroparalytic ophthalmia may also be present, owing to the disturbance in the nutrition of the nerves which pass along the side of the cavernous sinus.

In thrombosis of the sinuses in infants the fontanelle is depressed, and at times the edges of the bones pushed one over the other; but during the progress of the disease the fontanelle may again become tense and prominent, and the cranial bones pressed apart (Gerhardt). This increase of the contents of the skull is caused either by effusion of serum from the tense veins giving rise to a species of hydrocephalus or to extensive meningeal or intra-cerebral hemorrhage resulting from thrombosis of the sinuses.

The *phlebitic variety*, as already remarked, is generally caused by otitis interna or injuries to the head. These affections also give rise to meningitis and cerebral abscesses as well as to purulent thrombosis, and inasmuch as these pathological conditions are frequently combined, it is very difficult to distinguish clinically between them. In a few reported cases, however, suppurative thrombosis was alone present uncomplicated by meningitis or by lesions of the cerebral substance. The affection sometimes pursues a latent course, and is only discovered after death. The symptoms are usually similar to those observed in cases of septicæmia with specially prominent cerebral symptoms. The attack frequently begins

with chilliness, which generally recurs repeatedly during the course of the disease, and the patient has a characteristic typhoid look, with dry tongue, loss of appetite, and mental confusion. After a time the patient falls into a somnolent condition, which gives place to complete coma, terminating in death. Mild delirium is present in a few cases, and more rarely the delirium assumes an active form.

Suppurative thrombosis is frequently associated with motor and sensory disturbances caused by the accompanying meningitis. These consist of pain in the head, hyperalgesia, paresis, paralysis, and convulsions.

§ 706. *Diagnosis.*—When a patient, suffering from caries of the internal ear, furunculus in the face, or who has received an injury to the head, develops symptoms like those of pyæmia, with marked disturbance of the cerebral functions, purulent thrombosis of the sinuses may be suspected. The diagnosis will be further corroborated by the disturbances of the circulation, which have already been described from the thrombosis.

§ 707. *Course and Prognosis.*—The duration of the disease is difficult to determine, and it may probably extend occasionally over several weeks, although usually terminating in a much shorter time.

The prognosis is very unfavourable, but recovery is said occasionally to take place (Sédillot, Lebert, and Griesinger).

§ 708. *Morbid Anatomy.*—Any sinus may become the seat of thrombosis, but some of them are much more liable to be affected than others. The superior longitudinal sinus is the one which is usually implicated in cases of thrombosis from marasmus, and the sinuses in the neighbourhood of the petrous bone in the phlebitic variety. The veins which empty themselves into the sinuses become enlarged and gorged with blood, and are often filled with thrombotic masses, so that they look like large earthworms when lying on the surface of the brain. Ruptures of the vessels not unfrequently occur, causing meningeal hæmorrhage, but sometimes consists only of small hæmorrhagic spots, while at other times may amount to pro-

fuse hæmorrhage. The cortex of the brain is also frequently the seat of capillary hæmorrhages, and Lancereux has described small spots of softening. The phlebitic variety is frequently accompanied by meningitis, caused by the primary lesion.

§ 709. *Treatment.*—No treatment has hitherto been found of any avail.

(c) *Occlusion of the Cerebral Capillaries.*

§ 710. Experimental investigations have shown that marked disturbances of the cerebral functions may be caused by occlusion of the cerebral capillaries, and clinical records also point to the same conclusion.

§ 711. *Etiology.*—In severe cases of malarial and intermittent fever the cerebral capillaries are liable to be obstructed by dark masses, a condition which has been called *pigment embolism.* The cerebral capillaries may also be obstructed by drops of fat. The fat is usually swept into the blood current by the breaking up of atheromatous formations in the interior of the larger blood-vessels. In cases of injury to bone the fatty tissue of the marrow may be carried into the blood-vessels, giving rise to emboli in the lungs and possibly in the brain.

Chorea has been supposed to be due to capillary embolism, but the subject will be subsequently discussed. The cerebral capillaries are said to be occluded by lime becoming deposited in their walls, a process named by Virchow *lime metastasis.* Some disease of bone is usually associated with this condition, and Virchow thinks that the lime is first absorbed from the diseased bone, and afterwards deposited in the vessels.

§ 712. *Symptoms.*—The experiments of Feltz, and of Prévost and Cotard show that extensive embolism of very fine particles may rapidly induce death in animals by causing diffuse anæmia of the brain. Nothing analogous to this is known to take place in diseased conditions. If the embolic masses are few the symptoms which they give rise to are so slight as not to be recognisable during life. Such is known to be the case in certain instances of fat embolism. In other cases a considerable

territory of the brain may be suddenly deprived of its nutriment, and apoplectic symptoms may then be produced, followed by the usual symptoms of a localised cerebral disease.

The symptoms, however, are usually such as arise from diffused cerebral disease, the more common of them being dizziness, headache, nausea, trembling, and weakness in the extremities, and mental disturbance, as marked loss of memory and other signs of mental decay.

§ 713. *Morbid Anatomy.*—Capillary occlusions are, of course, only to be detected with the microscope. Delacour says that in cases of lime metastasis a resistance is felt to the knife in cutting through the brain, and rough prominences may be felt on the surface with the finger.

The nature of the secondary changes in the brain varies according to the number of the vessels obstructed, and it is only when a large number are occluded that disturbance of the circulation will not be compensated, structural changes then occurring analogous to those following obstruction of the large arteries. Experimental investigation has shown that the first effect of the occlusion is to cause anæmia, and in the further progress of the affection the various stages of necrobiosis may supervene, ending in complete softening. The centres of softening are often of small size, but several are usually present.

§ 714. The *course* and *prognosis* depend upon the extent and nature of the occlusion. Isolated capillary embolisms are of no significance; but if they are numerous the resulting disturbances are in every respect similar to the corresponding secondary effects of the occlusion of the larger arteries.

§ 715. *Treatment.*—The treatment must be conducted on general principles.

CHAPTER IV.

(I.) GENERAL CONSIDERATION OF FOCAL DISEASES, ACCORDING TO THE NATURE OF THE LESION (CONTINUED).

2. INTRACRANIAL HÆMORRHAGE.

INTRACRANIAL hæmorrhage may be divided into (*a*) *cerebral*, and (*b*) *meningeal* hæmorrhage.

(*a*) *Cerebral Hæmorrhage.*

§ 716. *Definition.*—By cerebral hæmorrhage is here meant an extravasation of blood into the substance of the encephalon or into the ventricles of the brain.

§ 717. *History.*—Hæmorrhage into the substance of the encephalon is frequently termed apoplexy. The word ἀποπλήσσω means "I strike down," and a person who had suddenly fallen down insensible was said to be in a condition of ἀποπλήξια. It was pointed out by Wepfer that this condition was frequently caused by cerebral hæmorrhage, and after a time the name of the group of symptoms which signified sudden unconsciousness was transferred to the anatomical condition which was the most frequent cause of that occurrence. The process did not stop here; during the course of investigation it was seen that hæmorrhage into the substance of other organs was not uncommon, and after a time the meaning of the term was extended so as to include these hæmorrhages also. The term therefore having come to signify conditions so different, it will be well to avoid its use as much as possible.

§ 718. *Etiology.*—The circumstances which predispose to cerebral hæmorrhage are—(1) Disease of the vessels, (2) Increase of the arterial tension, (3) Disease of the tissues surrounding the vessels, and (4) Certain diseases of the blood itself.

(1) *Disease of the Vessels.*—The great majority of massive hæmorrhages into the substance of the brain are due to fatty degeneration of branches of the Sylvian artery, which pass through the anterior perforated space to reach the corpus striatum. Fatty degeneration of arteries may be primary or secondary. Primary fatty degeneration is a passive process, not being preceded by any increased nutritive activity of the affected parts, but the secondary form of the process is preceded by an inflammatory cellular infiltration of the sub-endothelial connective tissue of the vessels, and constitutes atheroma. It was formerly believed that when the arteries at the base of the brain were found in a condition of atheromatous degeneration the existence of a similar condition of the vessels in the interior of the brain might be inferred, and that intracerebral hæmorrhages might in most instances be attributed to the brittleness of the vessels. The belief is now growing that the influence of atheromatous disease in the causation of cerebral hæmorrhage is indirect rather than direct. Atheroma of the vessels may occasionally lead to aneurisms of the larger vessels at the base of the brain, but they are not often the cause of hæmorrhage. Besides, rupture of an aneurism of one of the larger vessels would give rise to hæmorrhage between the meninges, and not into the substance of the brain. Atheromatous degeneration may, however, cause hæmorrhage indirectly by rendering the walls of the larger vessels rigid, so that the pulse wave reaches the arterioles without being modified by the normal elasticity of the arteries.

By far the most frequent cause of intracerebral hæmorrhage is that condition of the arterioles which has been described by Charcot and Bouchard as *miliary aneurisms.* These aneurisms are situated on the arterioles, are of a reddish colour, and vary in size from that of a millet-seed to a pin's head. Sometimes a few only are found in the vicinity of the ruptured vessel, while at other times they are scattered in large numbers throughout the whole brain. The parts of the brain in which they are situated, taken in the order of their decreasing frequency, are the lenticular nucleus, the optic thalami, the pons, the convolutions, the caudate nucleus, the cerebellum, the medulla oblongata, the middle peduncles of the cerebellum, and the centrum ovale.

Miliary aneurisms occur rarely before the fortieth year, but are found with increasing frequency after that age. They result, according to Charcot and Bouchard, from a kind of arterial sclerosis of the nature of a chronic periarteritis. This alteration consists in multiplication of the nuclei of the lymph-sheaths and adventitia, a process which is generally accompanied by atrophy of the muscular coat. When atrophy of the latter occurs without a compensatory thickening of the adventitia, rupture of these aneurisms very readily takes place.

The part which primary fatty degeneration of the vessels plays in the causation of cerebral hæmorrhage has been insisted upon by Paget. This condition of the vessels is found at all ages, and in cachectic children even

more frequently than among aged persons, so that care must be taken not to over-estimate its influence in the production of hæmorrhage. Billroth has also shown that in a large number of cases this form of fatty degeneration of the small vessels is secondary to disease of the nervous tissues. Even after making these deductions from its importance as a predisposing cause, there can be no doubt that this condition does increase the liability to cerebral hæmorrhage.

(2) *Vascular Tension.*—It is very doubtful whether increase of the arterial tension ever gives rise to cerebral hæmorrhage without disease of the vascular walls; but when the latter are degenerated then sudden increase of tension becomes a powerful predisposing cause of hæmorrhage. Sudden exposure to cold may increase the arterial tension by inducing extensive contraction of the cutaneous arteries. During the winter months it is very common for individuals to be found in an apoplectic condition on the streets, and taken up by the police supposed to be drunk. Such cases occur usually in persons beyond middle age, their breath may smell of alcohol, and they may even be known to have been drinking during the evening. The evening has been spent in a heated apartment, where, under the conjoined influence of a high temperature, alcohol, and emotional excitement of various kinds, the cutaneous vessels have become dilated, the skin bathed in perspiration, and the cardiac action increased. On going out into the cold air the surface becomes suddenly chilled, the cutaneous vessels contract, the arterial tension becomes immediately greatly increased, the internal organs become gorged with blood, and if, as is frequently the case, the walls of the cerebral vessels are weakened by disease, rupture takes place (Fothergill).

The hypertrophy of the left ventricle which is associated with contracted kidney takes a more active part in the production of cerebral hæmorrhage. Avoiding as much as possible controversial points, it is beyond question that contracted kidney is associated with a general condition in which the walls of the arterioles of the entire body become thickened, inelastic, and the lumina of the vessels themselves much diminished in size. This condition greatly obstructs the flow of blood from the heart towards the capillaries, and the left ventricle becomes the subject of compensatory hypertrophy, with the effect of producing a permanent increase in the arterial tension. And whatever may be the nature of the primary change in the arteries, whether a hypertrophy of the muscular coat, or sclerosis of the external coat, or both combined, the walls of the vessels undergo in long-standing cases degenerative changes which render them brittle and easily ruptured.

Obstruction to the return of the venous blood from the brain probably also predisposes to hæmorrhage, but its direct effect must be small. The obstruction may be temporary or permanent. Temporary in such actions as coughing, sneezing, laughing, or straining at stool, and permanent in affections of the mitral and tricuspid valves, obliteration and compression of the cerebral sinuses, compression of the jugulars and

superior vena cava, and affections of the lungs as emphysema and fibroid phthisis.

(3) *Condition of the Tissues.*—Rochoux advanced the theory that spontaneous hæmorrhage is generally preceded by a process of softening of the cerebral tissue, to which he gave to this process the name of *ramollissement hémorrhagipare.* In consequence of the change of consistence of the nervous tissue, the small vessels lose their natural support, and become unable to resist the pressure of the blood. It is now generally believed that the softening is a secondary process, the result partly of the imbibition of blood serum, and partly of inflammation excited by the extravasation in the surrounding tissues. Hæmorrhage may, however, occur as a result of softening of the tissues in cases of embolism and thrombosis, but this condition will be noticed hereafter. Some authors think that hæmorrhage is due occasionally to atrophy of the cerebral substance, and believe that the vessels then rupture in consequence of their becoming dilated in order to fill the vacuum. But the reduction in the size of the brain proceeds far too slowly for much dilatation of the vessels to result from it; and the atrophy is compensated to some extent by thickening of the skull and increase in the size of the frontal sinuses, but chiefly by increase of the cerebro-spinal fluid.

(4) *State of the Blood.*—Various diseases, the essential condition of which appears to be caused by some change in the composition of the blood, occasionally lead to cerebral hæmorrhage. Cerebral hæmorrhages have been observed in pyæmia, in the typhoid state, scorbutus, purpura, chlorosis, leucocythæmia, pernicious anæmia, and icterus, but are exceptionally met with in these diseases.

§ 719. *Other Predisposing Causes.*—Some families exhibit a predisposition to cerebral hæmorrhage, hence it has been assumed that the disease is hereditary. The action of heredity in predisposing to hæmorrhage is, however, only an indirect result of the inherited tendency to arterial degeneration. It was formerly believed that some individuals inherited an apoplectic constitution. This was supposed to be characterised by broad chest, short neck, large abdomen, powerful muscular system, and florid complexion. Exact statistics, however, prove that cerebral hæmorrhage does not spare any constitution, and that poorly-nourished, thin persons are as frequently attacked as the plethoric.

One of the most important predisposing causes of the disease is *age.* Cerebral hæmorrhage is rare before the fortieth year, relatively frequent afterwards. It must not be forgotten that the disease attacks young persons, and it has been observed in

infants and even at birth. Meningeal hæmorrhage is relatively common in early childhood.

Sex undoubtedly exercises a certain degree of influence in predisposing to cerebral hæmorrhage, probably owing to the fact that men are more frequently exposed to the exciting causes of the disease. The proportionate frequency with which men and women are attacked has been variously estimated by different authors, but it may safely be asserted that the ratio of 2·1 rather under than overstates the proportion.

The influence of *occupation* in predisposing to cerebral hæmorrhage has not yet been satisfactorily determined, and the same may be said with regard to the influence of *climate*, since the immunity from the disease once attributed to warm climates has recently been called in question. In Europe the disease is most common in winter, then in autumn and spring, and least so in summer. *Altitude* appears to exert some influence in the production of the affection, since it is very common in the elevated regions of Mexico, of the Cordilleras, and the Andes. Certain substances, as alcohol, predispose to hæmorrhage by inducing fatty degeneration of the vessels.

§ 720. *Symptoms.*—The symptoms vary greatly according to the situation and extent of the lesion; but the mode of onset being sudden and the lesion of a destructive character, the initial group of symptoms bear a general similarity to each other in all cases.

1. *Premonitory Symptoms.* — The attack is frequently ushered in without any premonitory symptoms, and in no instance can any symptom be relied upon as an invariable antecedent of hæmorrhage. Premonitory symptoms may, however, manifest themselves days and even weeks before the actual onset of the attack, and these are no doubt frequently caused by rupture of minute vessels prior to the graver event which ushers in the apoplectic condition. The usual forerunners of the apoplectic attack are dizziness, headache, ringing in the ears, muscæ volitantes, numbness in the hand or foot, muscular twitchings of the face or of some portion of the upper or lower limbs, especially of the fingers or toes, mistakes in talking or writing, vomiting, mental irritability and drowsi-

ness. These symptoms may appear separately or variously combined, and although all of them may occur without being followed by an apoplectic attack, yet in the old and middle aged, especially when the arteries are degenerated, they should be regarded as warnings.

2. *Modes of Onset.*—For facility of description the mode of onset of cerebral hæmorrhage may be divided into three principal classes: (i.) The apoplectiform onset; (ii.) the epileptiform onset; and (iii.) the simple mode of onset (Bastian).

(i.) *Apoplectiform Onset.*—This mode of onset is characterised by sudden loss of consciousness with resolution of the limbs, and what is popularly termed "apoplexy." In a small number of cases the onset may be instantaneous. In the midst of apparent health the patient may fall insensible to the ground. In such cases the lesion, which need not necessarily be large, is usually found in the pons or medulla. The attack usually begins more gradually. The patient suffers from dizziness, abnormal sensations or pain in the head, mental confusion, difficulty of speech, drowsiness, nausea and vomiting, or a sense of great exhaustion; and after some of these symptoms have continued for a few minutes or longer the stage of unconsciousness comes on.

When the apoplectic attack is well marked the patient lies in a state of profound coma, and is insensible to all kinds of stimuli. The face is usually flushed and swollen, though occasionally it may be pale and clammy, the lips are livid, the head and neck feel warm and are bathed in perspiration, the carotids and other arteries throb violently, the eyelids are closed, the conjunctivæ injected, the eyeballs fixed, the pupils sluggish to light, the respiration is usually deep, with or without stertorous inspiration and protrusion of the cheeks during expiration, the pulse is generally full and slow, and there is either complete muscular resolution, so that the limbs when raised drop like inert bodies, or the resolution is more marked on one side of the body than on the other. In the severest cases there is not only complete absence of voluntary motion, but all the reflex movements are abolished, with the exception of the cardiac and respiratory movements, and those concerned in

deglutition, the latter being generally retained as regards the pharynx and œsophagus.

When this condition has been brought about by a severe lesion of the brain, the patient may die after a few minutes, a few hours, or a few days. In the slighter forms, however, the apoplectic state may last only a short time, and then gradually give place to other related symptoms. When the coma is not very profound, powerful irritations cause reflex movements, and in the lesser degrees of the apoplectic state, the patient, when loudly spoken to, raises his eyelids for a moment or two, and may even reply in a monosyllable when loudly pressed with any question. In such cases a difference can be detected between the two halves of the body; the extremities of one side offer a certain resistance to passive motion, while those of the other sink, when unsupported, like inert masses; the corner of the mouth on one side is lower than on the other, and the opposite naso-labial fold is strongly marked.

(ii.) *The Epileptiform Onset.*—The epileptiform is a mere variety of the apoplectiform mode of onset. The patient, either with or without prodromata, drops down insensible in a kind of epileptic fit, and after a time it is discovered that the patient is paralysed on one side of the body. Temporary hemiplegia may follow severe attacks of unilateral convulsions due to a molecular lesion of the cortex, but in the cases under present consideration, the hæmorrhage destroys a certain portion of the brain, and the paralysis initiated is more or less persistent. Although prodromata may be absent altogether, yet the epileptic attack is very frequently preceded either by pains in the head or by muscular twitchings, or the initial attack may be characterised by unilateral convulsions, and in these cases the half of the body convulsed corresponds with that which is subsequently paralysed. But when convulsions occur after paralysis has become established, it usually happens that the non-paralysed side is the one which is affected with clonic spasms, and in these cases there is probably co-existing, but unequal, damage to both hemispheres of the brain.

Some of the patients whose hemiplegic condition is ushered in by convulsions speedily die, whilst others remain liable to

a recurrence of epileptiform attacks at variable intervals; in many cases no subsequent attack occurs, even though the patient live for many years. Another remarkable peculiarity with respect to cases initiated by convulsions is, that the period of stupor or partial unconsciousness may be prolonged for three, four, five, or even six weeks, and yet the patient may recover. In these cases, however, the patient is not deeply comatose, but lies in a somewhat lethargic condition, with eyes closed or only half open, and takes no notice of anything that is going on around him. A patient, on the other hand, who continues deeply comatose for forty-eight hours very rarely recovers.

(iii.) *Simple Onset.*—In the simple mode of onset the patient may suddenly fall owing to paralysis of an inferior extremity, but the fall is not accompanied by any loss of consciousness. The patient may experience no pain, but usually complains of a feeling of "numbness" in the paralysed side of the body. This mode of invasion is very frequent in the slighter forms of hemiplegia.

Temperature.—The variations of the temperature of the body in cases of apoplexy have been studied with great care by Bourneville. The temperature is at first lowered in all cases, sometimes reaching 96·5° F., and in the fulminating form of the disease it remains low until death. If life continue for ten or twenty hours the initial sinking gives place to a marked elevation of temperature. If the primary depression is followed by a steady and continuous rise of temperature it is a very unfavourable sign, and in these cases the pyrexia may reach 108° F. before death. In the more favourable cases the initial lowering is followed by a stationary period, during which the temperature varies between 99° F. and 100·5° F., and continues to oscillate rather irregularly for from two to four days. If the case be going to terminate in recovery, and supposing there be no inflammatory complications of other organs, the temperature gradually falls to the normal standard, and there remains. When, however, the case is to terminate fatally in the course of a day or two more, the stationary period is followed by a rapid and continuous rise of temperature, which is a not less unfavourable sign than when the same occurs after the period of initial lowering.

Conjugated deviation of the eyes, with rotation of the head away from the paralysed side and towards the hemisphere which is the seat of disease, usually occurs as a temporary symptom in all cases of severe cerebral hæmorrhage (§ 90). The eyes are usually fixed, but occasionally exhibit slight nystagmus. When the disease is situated in the posterior half of the pons the rotation is directed towards the paralysed side (Prévost, Grasset). The rotation may completely disappear when the patient falls asleep.

3. *Permanent Symptoms.*—The permanent symptoms caused by cerebral hæmorrhage consist of paralysis of voluntary motion, generally limited to one side of the body (hemiplegia); various tonic or clonic spasmodic affections, also generally limited to one side of the body (hemispasm); and unilateral sensory disturbances, including affections of the special senses (hemianæsthesia). As these affections are not, however, peculiar to hæmorrhage, we reserve consideration of them for the present.

§ 721. *Disturbances of the Mental Functions.*—The majority of those who have been attacked with cerebral hæmorrhage do not regain their full mental vigour. Memory usually fails, more especially for recent events. In the daily affairs of life the judgment of the patients may not appear to have suffered, but they are unequal to any unusual intellectual effort; and at times the intellect may progressively decline, reducing the patient to a state of childishness or pronounced dementia. At other times they become peevish, whimsical, irritable, or give way to outbursts of passion. The mental affections connected with disturbance of speech (aphasia) will be hereafter considered.

Trophic and Vaso-motor Disturbances.—Immediately after an attack the paralysed limbs of a hemiplegic patient are frequently redder and warmer than the corresponding healthy limbs. The difference in temperature may vary from a fraction of a degree to as much as two degrees. These symptoms are no doubt due to paralysis of the sympathetic. About twenty-four hours after the beginning of an attack the paralysed limbs may become swelled owing to a certain amount of subcutaneous œdema. The temperature of the paralysed limbs gradually

decreases, and is eventually lower than that of the sound side. When no œdema exists the skin may be dry and scaly.

Acute Bed-sore.—This is an acute process of sloughing, which occasionally occurs over the centre of the gluteal region on the paralysed side, after cerebral hæmorrhage or softening. The affection has already been sufficiently described (§ 114).

Congestions and Hæmorrhages.—Congestions and actual hæmorrhages into the substance of the lungs, extravasations in or beneath the pleura, endocardium, and the mucous membrane of the stomach, as well as into the substance of the supra-renal capsules and kidneys, frequently accompany cerebral hæmorrhage. Schiff and Brown-Séquard produced experimentally hyperæmia, or hæmorrhage of the pleura and lungs, by certain lesions of the pons, middle cerebellar peduncles, and the optic thalami and corpora striata. These hyperæmic conditions and hæmorrhages, whether in the lower animals or in man, are sometimes confined to the paralysed side of the body. The diminution of the contractile power of the walls of the arterioles on the paralysed side often gives rise to a perceptible difference between the radial pulses of the two sides.

Inflammation of the Joints.—Some of the joints of the paralysed side of the body may become the subjects of a subacute inflammation, which usually begins from the third to the sixth week after the hemiplegia, although sometimes the joints inflame at a still later period after the beginning of the attack, and occasionally the affection shows itself as early as the fifteenth day. There are the two varieties of this articular inflammation, the one acute and the other chronic. In the first variety the joint becomes red, hot, swollen, and, after death, acute synovitis, frequently with considerable exudation, is discovered. This form almost exclusively attacks the larger joints. A chronic joint affection has been described by Hitzig which seems to be peculiar to the shoulder. The joint is almost immovable, painful on pressure, and, owing to paralysis of the muscles, the humerus semi-dislocated.

Changes in Nerve Trunks.—Cornil has shown that in a certain number of cases there is a sub-inflammatory hypertrophy of the nerves or of their sheaths, and in such cases there is pain on pressure of the paralysed limb, especially marked along the course of the principal nerve-trunks. At other times the whole paralysed side may be generally tender, without any special limitation of the tenderness to the joints and large nerves.

Muscular Atrophy.—In some rare cases an early and rapid wasting takes place in the muscles of one or both limbs a few weeks after the onset of the paralysis, but in these cases there is reason to believe that the fibres of the pyramidal tract have undergone secondary degeneration, and that the motor cells of the anterior horns of the cord have become implicated in the process.

Arrest or Retardation of Growth in Paralysed Limbs.—When hemiplegia occurs in childhood, the arm and leg, or the arm only, on the para-

lysed side grow more slowly than on the sound side, so that as growth advances the limbs of the paralysed remain permanently smaller than those of the opposite side. The arm is more frequently affected than the leg, and there is always a certain amount of muscular rigidity of the affected extremity.

Skin, Hair, and Nails.—The skin of the paralysed side sometimes undergoes trophic changes, which involve the cutis and subcutaneous tissue, so that a fold pinched up by the fingers feels thicker than normal. The hair grows better on the affected side, and the nails become yellowish, marked with ridges, brittle, and curved.

§ 722. *Morbid Anatomy.*—Morbid anatomists usually divide cerebral hæmorrhage into two varieties, named respectively *punctiform* and *massive* hæmorrhages.

Punctiform hæmorrhages occur in the form of a number of minute points of the size of a pin's head, or even smaller. They result from rupture of capillary vessels, and are invariably multiple. Capillary hæmorrhages are observed in the tissues surrounding massive hæmorrhages, or in parts which are the seat of softening, and they are met with in considerable numbers in the cortex of the brain in consequence of thrombosis of the venous sinuses. At other times extravasations of blood are found in the lymph sheaths of the vessels, and they must then be regarded as minor degrees of the massive hæmorrhages.

Massive hæmorrhages may be of various sizes, being sometimes as small as a pea, at other times large enough to destroy almost an entire hemisphere. The hemorrhage may either separate the nerve fibres of the white substance or rupture them, the latter event being by far the more frequent. When the nerve fibres are pushed aside by the hæmorrhage without rupture the form assumed by the clot will be determined by the direction of the fibres, but when the fibres are ruptured the clot is round or oval. In the cortex the form assumed by the hæmorrhage is largely determined by the disposition of the convolutions and membranes, so the effusion usually spreads out laterally and assumes an irregular form. Massive hæmorrhages are, as a rule, single, although several foci may occasionally be observed, and it is not unusual to find traces of many extravasations of various ages in the same brain.

Hæmorrhagic foci may occupy any part of the brain, but

they are much more frequent in certain parts. The favourite seats are the caudate and lenticular nuclei, and the optic thalami.

Recent Focus.—In the recent condition the apoplectic focus forms a dark red clot, which is soft and uniform in character throughout. It is frequently mixed with the *débris* of the substance of the brain. The internal surface of the cavity is irregular and consists of torn shreds of cerebral tissue. This is surrounded by a zone of variable thickness, averaging a few lines in depth and gradually merging into the healthy tissues, composed of softened tissue saturated with blood serum, and frequently the seat of punctiform hæmorrhages. If the nerve fibres have been simply separated from one another without rupture, then the detritus of cerebral tissue in the internal surface of the walls of the cavity is absent, and the softening and punctiform hæmorrhages of the surrounding tissues are much less marked. If the clot be floated out under water, it is sometimes possible to detect the miliary aneurism from which the primary extravasation took place.

Period of Absorption and Repair.—If the hæmorrhage does not end fatally after a few hours structural changes take place, both in the clot and surrounding tissues, which lead to the absorption of the former and to a certain amount of repair in the latter. The blood-clot after coagulation parts with its serum, and the injured tissues surrounding the clot become softened, partly by imbibition of serum, but chiefly owing to a retrograde fatty metamorphosis of the torn fragments of brain tissue. The softened tissues become mixed with the clot so as to form a dark, chocolate-coloured mass, of the consistence of gruel, the more fluid constituents of which are soon absorbed. The hæmatine is dissolved, and soaks into the tissue round the clot to a considerable distance, until it is absorbed. As a result of this process the pulpy material filling the cavity passes from its first dark red to a brighter red, and finally to a saffron colour. A reparative process now begins, by means of which the hæmorrhagic focus is converted into a *cyst*. The first step in the reparative process is the formation of a fibrinous capsule round the entire periphery of the clot. It is at first a line or more in thickness, soft as jelly, and of a trans-

lucent yellowish tint. At a later period this capsule becomes converted into a much thinner but stronger layer of fibrillar connective tissue, which permanently shuts off the apoplectic deposit from the surrounding substance of the brain. The fluid contained in the cyst is at first turbid, but after a time becomes transparent and limpid. These cysts, however, contain not fluid merely but also a loose spongy connective tissue, which is suspended in the fluid like a film. But the reparative process does not always end here; the whole of the fluid may become gradually absorbed, and the opposite walls of the cavity may ultimately come into contact, and adhere to one another by a connective tissue, which usually contains a considerable amount of pigment. This constitutes the hæmorrhagic or apoplectic cicatrix, which consists merely of a thin strip of connective tissue. Superficial foci in the cortex pass through similar phases, and after cicatrisation they appear as yellow indurated spots which have been taken for vestiges of encephalitis.

Duration of the Reparative Process.—The clot is soft and homogeneous during the first three or four days. At this time the process of softening and separation of the internal surface of the cavity, and the absorption of the fluid contents, reach their maximum activity at the eleventh or twelfth day. The reparative process which leads to the formation of the capsule begins usually from the seventh to the ninth, the cyst is complete about the twentieth, and the lining membrane is organised from the thirtieth to the fortieth day.

Circumstances which prevent the Reparative Process.—Various circumstances delay or entirely prevent the reparative process. The principal of these are, a too extensive sero-sanguineous infiltration of the surrounding tissues followed by a co-extensive area of softening, an excess of the irritative process necessary to repair, which gives rise to *secondary encephalitis*, a fresh hæmorrhage and dropsy of the cyst, leading to distention and consequent pressure on the surrounding tissues. Repair of injury to the brain from hæmorrhage may be prevented, like repair of injuries of every part of the body, by the general state of the health in various conditions of debility.

§ 723. *Prognosis.*—The prognosis in any given case depends

upon the opinion formed of the extent and situation of the lesion taken in conjunction with the age and previous state of health of the patient. Death not unfrequently takes place during the apoplectic condition. If the patient cannot be roused at all, if there be no signs of reflex activity when the conjunctiva is touched, while there is involuntary passage of fæces and urine, and well-marked stertor, the patient may die rapidly within a few hours, or even a few minutes; and the persistence of a slighter degree of these symptoms without abatement is a sign of great gravity. Laboured respiration and quickness with marked irregularity of the pulse, are also unfavourable signs. A marked and persistent depression of the temperature is regarded by Charcot as an almost certainly fatal sign. If the patient has recovered from the apoplectic condition, then the prognosis will greatly depend upon the age and general condition. Granular disease of the kidneys, a general state of malnutrition, or evidences of senile degeneration of the arterial system, will render the ultimate prognosis grave in cases where the extent and situation of the hæmorrhage itself would cause no danger to life. A sudden rise of temperature in cases of cerebral hæmorrhage is a very grave indication, unless some inflammatory complication be present to account for it. A sudden depression of temperature, with increase or renewal of a pre-existing comatose condition, indicating as it does the occurrence of a fresh hæmorrhage, is also of serious import.

Acute sloughing of the buttock on the paralysed side, commencing within a few days after the onset of the apoplectic attack is, according to M. Charcot, of fatal significance. Decided difficulty of deglutition and articulation is also a serious symptom, being indicative of marked interference with the functional activity of the medulla and pons. When the patient has outlived the apoplectic attack, the period of reactive inflammation brings new dangers, when death may result.

When the inflammatory period is passed there is comparatively little reason to expect a fatal result from the brain lesion itself or from its more immediate complications. In middle-aged and old people, however, there is a constant danger of a recurrence of the hæmorrhage. The dangers of the apoplectic

attack having been surmounted, the point which has to be determined is the degree of improvement likely to take place in the patient's mental faculties, in his power of articulation and speaking, and as regards the probability of restoration of motor power to his paralysed limbs.

In the majority of instances when the first loss of consciousness has passed away, the patient is left free from any very decided mental defect, except a certain amount of mental weakness and a tendency to emotional displays. In rare cases the hemiplegic attack is followed by a chronic maniacal condition, which may pass into a state of complete dementia. This condition is apt to follow limited cortical hæmorrhage of the occipital lobes, especially in elderly people, but the hæmorrhage may itself be only an effect of previously-existing degenerative changes.

Large lesions occurring in infancy or at the time of birth, either in the substance or on the surface of the brain, often induce a semi-idiotic condition.

§ 724. *Treatment.*—The aims of treatment are (1) to avert a threatened attack; (2) to treat the apoplectic condition; (3) to allay excitement during the stage of inflammatory reaction; and (4) to restore power to the paralysed limbs, and to improve the other morbid conditions which accompany the hemiplegic state.

(1) *Prophylaxis.*—In devising measures to prevent a threatened attack, each case must be made the subject of special study; and much depends for the success of these on the age, general state of health, and hereditary tendencies of the patient. Bodily and mental rest are absolutely necessary. The patient ought to be kept cool, with his head and shoulders well raised. If the patient be beyond middle age, with signs of arterial degeneration and a weak intermittent action of the heart, stimulants, cardiac tonics, and the frequent administration of easily-assimilated fluid nutriment is necessary. In the presence of a moderate amount of granular disease of the kidneys with cardiac hypertrophy and high arterial tension, saline purgatives are indicated.

(2) Within the last few years our treatment of the apoplectic condition has undergone a great change. Bleeding was regarded

as the great remedy for the apoplectic condition from the time of Hippocrates down to within a few years ago, when the teachings of Todd and Trousseau produced a reaction in the opposite direction. When, however, hæmorrhage takes place in a case associated with high arterial tension, a small bleeding may, by lowering the blood pressure and thus diminishing the intracranial pressure, avert for a time threatening symptoms. If the heart be feeble, with compressible pulse, then bleeding is entirely inadmissible.

If there be much heat of the head, with violent throbbing of vessels, pounded ice in a bladder or india-rubber bag, or evaporating lotions should be applied while the head and shoulders are raised, and everything about the neck loosened. In the present day it is superfluous to condemn the barbarous practice of applying mustard plasters to the calves of the legs.

A stimulating treatment is required when the heart's action is feeble and the respiratory centre is threatened. In such a case the patient's face is cold and clammy, the pulse feeble, and the respiration hesitating and intermittent, or it may be assuming the Cheyne-Stokes character.

If the disease be characterised by recurring epileptiform attacks, bromide of potassium may be administered, and if there be a restless condition, with more or less of delirious wandering, the same drug or bromide of camphor may be useful. If the bowels be constipated, an enema containing castor oil or castor oil and turpentine may be administered, or two drops of croton oil may be given. The state of the bladder must also be attended to, and a catheter used if necessary. In many cases no drugs are required during the apoplectic stage, and purgatives should not be resorted to on all occasions as a routine treatment irrespective of the nature of the case.

(3) If the patient survive the first shock of the apoplectic attack the less we interfere during the first few days the better. He must be kept as quiet as possible both in body and mind, and his diet and secretions must be carefully regulated. When the reactive febrile symptoms appear cold should be applied to the head, but the old practice of bleeding at this stage is to be strongly condemned. If headache be present along with persistent wakefulness or delirium, it may be

necessary to administer a full dose of bromide of potassium or even an opiate or chloral. During this time great care must be taken to prevent bed-sores on the paralysed side, by paying constant attention to the state of the bedding and securing extreme cleanliness. In severe cases the patient should be placed on a water bed from the first where this is possible.

(4) The most efficient means of promoting the improvement of the condition of the paralysed nerves and muscles is a thorough attention to the general health of the patient. The treatment which it will be necessary to adopt will depend on the age, habits, and constitution of the patient, and on the presence or absence of any special concomitant disease. The general principles of treatment, however, are to take care that the patient has easily-digestible and nutritious food; that all circumstances which might cause mental excitement are avoided; and that the patient has a due amount of repose and sleep. In the hemiplegias of elderly people, which are usually associated with miliary aneurisms, great care must be taken that the circulation is not subjected to any sudden strain, and with this object it is necessary to take care that the bowels do not become constipated, lest the straining at stool should induce another attack. Iodide of potassium is often beneficial. The patient should also take open-air exercise in a chair or carriage whenever the weather is suitable; and much good may be done at a later period of the disease by sponging with salt water, either tepid or cold, or even by shower baths. When there is advanced degeneration of the arteries or high arterial tension, great caution is necessary in the use of cold sponging and shower baths, since the sudden impression on the cutaneous surface will be followed by contraction of the arterioles distributed to the surface of the body, and this will be followed by sudden increase of the arterial tension, and consequent risk of the rupture of another vessel. It may indeed be laid down as a rule that hemiplegic patients should only use baths of moderate temperature.

These general measures should after a time be followed by local treatment of the paralysed limbs. The first local measures to be resorted to are passive movements of the paralysed limbs, and friction of the skin by means of a

flesh brush, flannel, or the palm of the hand. When a paralysed limb is painful, gentle rubbing is very soothing and grateful to the patient. The patient may be directed to make voluntary efforts to move the limbs. Electricity is one of the most valuable agents we possess in the treatment of paralysed limbs. Both the faradic and galvanic currents have been employed, but the latter appears to be the more generally useful. The constant current has been employed in three different ways. According to one method the current is passed through the brain, in a second it is passed through the cervical sympathetic, while in a third it is directly applied to the paralysed limbs.

The practical rules which must be observed in carrying out the treatment are the following :—

(*a*) This method of treatment should not be adopted in the early stage of hemiplegia, as injury may be done by over-stimulation of the brain.

(*b*) The duration of each application through the brain ought to be short, not exceeding three minutes.

(*c*) The current should be weak, more especially in the case of elderly people—such, for instance, as that derived from five to ten or at most fifteen Leclanché's cells.

(*d*) The electrodes are to be placed on the mastoid processes, or one on the mastoid process and the other on the back of the neck.

(*e*) The electrodes should be placed in position when the index is at zero, and the current is then gradually increased and, after two or three minutes' application, gradually diminished before the electrodes are removed. Sudden interruptions and rapid reversals of the current ought to be avoided.

In the second method the current is passed through the cervical sympathetic. In this method the electrodes are placed over the course of the sympathetic in the neck, and it appears to be indifferent whether the anode is above and the cathode below or the reverse. The currents employed may be stronger than when the brain was directly acted upon. From fifteen to twenty-five Leclanché's cells may be used.

In the third method the electrodes are used along the course of the nerves, the negative pole being placed near the plexus to which the affected nerve belongs, or over the corresponding part of the vertebral column, and the positive pole over the trunks of the nerves. Some, however, recommend descending instead of ascending currents, but it does not appear to be of much consequence which is used. The current from thirty Leclanché's cells may be used for about eight minutes, and in order to increase its stimulating action the intensity may be alternately increased and diminished, while the circuit is kept closed. Interruptions and reversal of the

current should only be used for the purposes of diagnosis. This mode of applying galvanism to the paralysed limbs does good in cases of clonic spasm after hemiplegia, and in some cases of "late rigidity;" but when the contracture has become permanent, so that it does not intermit during sleep, it is hopeless to expect any benefit from treatment.

Faradic currents have been employed in contractures for the purpose of acting, not on the contracted muscles, but upon their antagonists, but it does not appear that much benefit has ever resulted from this treatment. The disturbances of sensibility on the paralysed side do not usually require any special treatment, since the measures which are directed to mitigate the motor paralysis exercise a favourable influence on any existing sensory impairment. If there be hemianæsthesia, metallo-therapeutics, as employed by Charcot, which will be described in the section on hysterical hemianæsthesia, may be adopted, but our knowledge of this subject is too recent and too imperfect to enable us to form a definite opinion of its merits.

(b) Meningeal Hæmorrhage.

Definition.—By meningeal hæmorrhage is here meant an extravasation of blood between the membranes or on the surface of the brain.

§ 725. *Etiology.*—The most frequent causes of meningeal apoplexy are injuries of the skull, by means of which the main meningeal arteries, the sinuses, or the vessels of the pia mater are ruptured, but this subject belongs to surgery.

Aneurisms of the arteries at the base of the skull may by rupture give rise to meningeal hæmorrhage. In a case which came under my observation, a large meningeal hæmorrhage was caused by rupture of an aneurism, about the size of a pea on the left Sylvian artery, about an inch from its origin. Another aneurism unruptured, symmetrical with it in size and position, was found on the right Sylvian artery. Next to the middle cerebral, the basilar artery is most frequently affected with aneurism. Hæmorrhage may also take place from the veins, and large meningeal hæmorrhage may result from thrombosis of the sinuses, especially the superior longitudinal sinus. Blood may make its way from the substance of the brain into the meninges through rupture of the cortex. Meningeal

hæmorrhage may result in the course of infectious diseases, and chronic dyscrasiæ, and frequently occurs in the course of the chronic degeneration of the cortex of the brain, which underlies progressive paralysis of the insane.

The *meningeal apoplexy of new-born children* is caused by certain accidents attending childbirth.

§ 726. *Symptoms.*—It will suffice if we point out here the differences which exist between the symptoms of cerebral and meningeal hæmorrhages. The clinical history of meningeal hæmorrhages of traumatic origin is usually complicated with other cerebral symptoms directly resulting from the injury, such as concussion, and the same may be said with regard to the cases where an intracerebral hæmorrhage has made its way to the surface of the brain, as well as with regard to the hæmorrhage which accompanies general paralysis. Hæmorrhage caused by rupture of an aneurism forms the least complicated class of cases.

In severe cases the patient becomes suddenly apoplectic without any warning, or with only slight premonitory symptoms, such as headache, dizziness, and vomiting. The paralysis is commonly general, affecting all four extremities uniformly, and only in rare cases is hemiplegia met with. Epileptiform convulsions are also frequent in meningeal hæmorrhage, and vomiting is another sign often observed. These cases are accompanied by profound coma, and death results in a few hours, or at most a few days.

In less severe cases the patient may partially recover after a few hours from the apoplectic state, and then may complain of headache, be delirious or somnolent, until he becomes finally comatose.

In other cases the patients do not become immediately apoplectic, but complain of headache, dizziness, weakness or numbness of the extremities, on one or on both sides; there is also more or less stupor, but the fatal coma may not supervene for a long time. In these cases the hæmorrhage appears to be small at first and gradually to increase.

If an aneurism of considerable size have existed for some time before the occurrence of hæmorrhage, the apoplectic

attack may be preceded by some of the symptoms which indicate the existence of a cerebral tumour. The more usual of these symptoms are headache, double optic neuritis, paralysis of the facial nerve in aneurism of the internal carotid, of the third nerve in aneurism of the posterior communicating artery, and vomiting, epileptiform convulsions, and disorders of deglutition, speech, and respiration in aneurism of the basilar artery.

In the meningeal hæmorrhages of the new-born, the children are either born dead or in a condition of asphyxia, and die soon afterwards. If respiration be established the infant remains weak, somnolent, or comatose, and dies after a few days from convulsions. Sometimes the children are weak and somnolent at birth, and remain in this condition from one to three weeks, when vomiting, dyspnœa, convulsions, and coma supervene and soon prove fatal.

§ 727. *Morbid Anatomy.*—The blood may make its way into the arachnoid space in consequence of injury to the dura mater, or from the vessels of the pia mater, or from the cerebral vessels and subsequent rupture of the pia mater. When the extravasation is large the hæmorrhage spreads extensively through the arachnoid space, so that an entire hemisphere, or exceptionally the surfaces of both hemispheres, may be covered with a thick layer of blood. When a large collection of blood has formed at the base and around the pons varolii, it may make its way into the ventricles through the great transverse fissure, and pass down through the aqueduct of Sylvius to the fourth ventricle. The quantity of the effused blood may vary from a few drops to half a litre or more. The pigmented spots sometimes found on the meninges and surface of the brain seems to indicate that small meningeal hæmorrhages may be absorbed, but large hæmorrhages invariably prove fatal.

The appearances presented by the brain vary greatly, according to the amount and seat of the hæmorrhage and the time at which death takes place. Hæmorrhage from the dura mater, if large, compresses without rupturing the brain. In such a case the gyri are found flattened and the substance of the brain pale. Hæmorrhage from rupture of the vessels of the pia mater or of the brain itself, and especially rupture of an aneurism

of the larger sized arteries at the base of the brain, may cause considerable destruction of cerebral tissue.

§ 728. The *prognosis* is more unfavourable in meningeal than intra-cerebral hæmorrhage, inasmuch as it is apt to be more copious, but the treatment of the two affections is similar. Trephining may possibly be of use in some cases of meningeal hæmorrhage.

CHAPTER V.

(I.) GENERAL CONSIDERATION OF FOCAL DISEASES, ACCORDING TO THE NATURE OF THE LESION (CONTINUED).

3. INTRACRANIAL TUMOURS.

§ 729. *Definition.*—Intracranial tumours consist of circumscribed pathological growths situated within the cavity of the skull.

§ 730. *Etiology.*—Tumours of the brain arise from similar causes to those which give origin to tumours in other localities. For the sake of convenience, cerebral tumours may be divided into (*a*) New formations; (*b*) Vascular tumours; (*c*) Parasites. Hereditary predisposition plays an important part in the production of new formations. Cancerous and tubercular tumours and syphilitic gummata depend upon a general constitutional taint, and it is also probable that glioma, sarcoma, and other tumours are more liable to arise in some families than in others. Cancer is one of the most common tumours of the brain, and is generally primary. When secondary it often follows cancer of the orbit. It is a disease of adult and advanced age, the largest number of cases being found between the ages of thirty and sixty years. Tubercle on the other hand is rarely primary, but is generally associated with tubercle of the lungs or cheesy glands; it is essentially a disease of youth, being most common between the ages of three and thirty years. It is probably the most frequent of all cerebral tumours. Syphilitic gummata may be met with at every period of life.

Cerebral tumours are more frequent in men than in women. Out of 329 cases of cerebral tumours of all cases collected by

Ladame 208 were male, 95 female, and in 26 the sex was not stated, so that, according to this computation, the proportion is rather more than two to one. Injuries of the skull act as exciting causes in the production of cerebral tumours. Several cases have come under my own observation in which the disease dated from a blow on the head, and the tumour in these cases frequently grew at a place corresponding to the seat of injury.

Vascular tumours consist of *aneurisms* of the cerebral arteries and *erectile* tumours. Aneurisms are observed at all ages, but they are more common between the ages of forty and sixty years, when the vessels begin to undergo atheromatous degeneration ; the causes of erectile tumours are unknown.

The parasites met with in the brain are the cysticercus and echinococcus.

§ 731. *Symptoms.—Headache* is one of the earliest and most striking of the initial symptoms of intracranial tumours. Ladame found this symptom in two-thirds of the cases collected by him. Headache is more violent in intracranial tumour than in any other disease except meningitis and the uræmia of chronic Bright's disease; it consists of an acute lancinating or severe boring pain, which may continue many weeks without intermission, and is aggravated by impressions of light, noises, and all movements of the head. The pain sometimes occupies the occipital and at other times the frontal or temporal regions; but its seat has no necessary relation to the situation of the tumour, although constant occipital pain is often associated with cerebellar tumour. Neuralgic headache from irritation of the fifth may be associated with the more profound headache of general pressure. Tenderness on percussing the skull may sometimes be observed at a point corresponding to the situation of the tumour (Ferrier).

Dizziness is a frequent initial symptom, and it may be present with or without cephalalgia. Paroxysms of headache and dizziness may be the only symptoms present for months, and the patient may feel well in other respects. Dizziness is probably caused by alterations in the circulation of the brain induced by the growth of the tumour; but the insecurity on assuming the erect posture, which is one of the

main elements of vertigo, is frequently caused by pressure on the labyrinthine fibres of the auditory nerve.

Sensory disturbances are generally ushered in by hyperæsthesia or some other irritative phenomena, which are after a time followed by anæsthesia. Wandering pains, formication, tingling, and numbness alternate with one another before there is a distinct diminution of sensation, and these symptoms do not entirely cease until complete anæsthesia is established. Ladame found cutaneous anæsthesia in one-seventh of his cases.

Neuralgia of the trifacial nerve arises from a variety of causes, but when all the three divisions of the nerve are simultaneously affected the presence of intracranial tumour is to be suspected. The pain occurs in paroxysms, and is usually associated with numbness, formication, itching, and the feeling of the part being swollen. When sensation is diminished on the painful side the pressure of a tumour on the nerve may be suspected, and the diagnosis is rendered more certain if disturbances of other cranial nerves are present.

Motor disturbances are generally ushered in by phenomena of irritation, to be followed by paralysis. The irritative symptoms are cramps of various parts or tremor of one of the extremities or of half the body. The cramps may vary from slight spasmodic twitches of the muscles of the face or of other special groups of muscles, to persistent tonic, clonic, or choreiform muscular spasms in the extremities; or there may be epileptiform convulsions, accompanied by unconsciousness. After a longer or shorter duration the irritative motor symptoms give place to paralysis, which creeps on gradually, and does not become complete for a comparatively long period. Hemiplegia is the more frequent form of paralysis, being present in a third of Ladame's cases. Permanent contractions of the paralysed extremities occur when the pyramidal tract is pressed upon or otherwise injured, but the spastic condition of the limbs is seldom so pronounced in intracranial tumours as in other focal diseases of the brain. The paralysed muscles atrophy apparently simply from disuse, and retain for a long time their electric excitability.

Affections of the Special Senses.—With respect to the affec-

tions of the special senses, those of sight are by far the most important. Calmeil found amblyopia in two-fifths of his cases, and Ladame found amaurosis in one-fifth. The optic disc may present the appearance known as "choked disc" (Stauungspapilla), or there may be neuritis (§ 207). The former is by far the most important sign of cerebral tumour, as it is generally present whenever there is increased intracranial pressure; and although this condition is said occasionally to accompany fluid effusion, yet the usual cause is a solid growth.

It is of the utmost importance for regional diagnosis to examine carefully for contractions of the field of vision, and for the different varieties of hemiopia. Diplopia is also a frequent symptom of tumours at the base of the brain, caused by an affection at the origin or pressure in the course of the third, fourth, or sixth cranial nerves.

The pupils vary; they may occasionally be contracted or unequal, but when by the growth of the tumour the intracranial pressure becomes great, they are always dilated and react feebly to light.

The sense of *hearing* is also frequently affected. Calmeil found some disturbances of hearing in one-ninth of his cases; Ladame says that the sense of hearing is affected only one half as often as the sense of vision. The auditory disturbances usually consist of dulness of hearing and rushing noises, but complete deafness is sometimes observed.

The injection experiments of E. Weber have shown that there is a communication between the arachnoid cavity and the labyrinth by means of the aqueduct of the cochlea, and consequently increased intracranial pressure may produce an affection of the auditory apparatus similar to that which occurs in the eyes under the same circumstances. Alterations of hearing may likewise be caused by pressure on the trunk of the auditory nerve or on its nuclei of origin in the medulla and pons. Pressure on the labyrinthine fibres of the auditory nerve may occasion vertigo and disorders of motor co-ordination similar to those observed in Menière's disease.

The *sense of smell* is relatively seldom affected in cases of tumour of the brain. The number mentioned in literature, how-

ever, is not a true criterion of the real number affected, since the patient is very apt not to mention the loss of smell unless it be entirely lost, and the physician is apt not to make any special investigation of it. Ladame found the sense of smell distinctly diminished or entirely lost in ten only of his collected cases, and never present as the only symptom.

The *sense of taste* is likewise only rarely affected. In Ladame's collected cases mention is made of alterations of this function only seven times, once the affection was unilateral, and the sense was only rarely completely lost. There are good grounds, however, for believing that if taste were carefully tested in all cases of cerebral tumours, alterations would be more frequently found.

The *organic functions* always become more or less injured in intracranial tumour. The intense cephalalgia alone prevents the patient from sleeping, and the continual wakefulness reacts on the general health.

Vomiting is frequently associated with paroxysms of headache and vertigo, but it may occur independently of these. It is often extremely obstinate and may continue for hours, and when it recurs frequently the general nutrition suffers greatly. Constipation is usually present, but in some cases it may alternate with diarrhœa. Irregularity of the heart's action and slowness of the pulse have been frequently observed, probably from irritation of the vagus. Towards the end, however, the pulse becomes very frequent.

The *respiratory function* is not often disturbed, but the rhythm may be quickened by irritation, and rendered slower by pressure, of the brain. Vierordt and Hegelmaier, by recording the movements of the superior abdominal region of rabbits on the drum of the kymograph, found that a moderate artificial pressure on the brain diminished the respirations by one-half, while they were increased in number by a stronger pressure. With moderate pressure the inspirations were fewer and the expirations longer.

Polyphagia is an occasional symptom of cerebral tumour, but it does not prevent the progressive emaciation. Rosenthal mentions a case where the polyphagia was accompanied with diabetes mellitus.

Polyuria and saccharine urine, either separately or combined, are frequently met with. In these cases it is almost certain that there must be irritation of the floor of the fourth ventricle, but the irritation need not be direct. Rosenthal relates the history of a case where diabetes was caused by tumour of the pituitary body, and I have seen a case where polyuria was occasioned by a tumour situated at the base of the skull over the right cavernous sinus.

Fever is not a usual symptom, but it is sometimes present during an attack of cerebritis, these complications being most frequently observed in the incipient stage of tubercular tumour.

The *nutritive* disturbances do not maintain a due proportion to the gravity of the cerebral phenomena, nor does the nature of the tumour appear to exert a marked influence on the general health. Cases have been observed in which cancer of the brain had existed for some months without producing a perceptible influence on the nutrition of the body, and those suffering from sarcoma may even manifest a tendency to obesity. As a rule, however, the subjects of tubercle and cancer sooner or later exhibit traces of cachexia. Tumour of the brain may act injuriously on nutrition in several ways. A state of great marasmus is sometimes induced by frequently recurring vomiting, while at other times the vital powers of the patient become exhausted by incessant headache and sleeplessness.

Psychical disturbances are frequently observed in cerebral tumour, but the statements of authors differ considerably with respect to the relative frequency of the symptom. Andral and Durand-Fardel assert that mental disturbances occur very seldom, while Calmeil observed psychical disorders in one-half, Friedreich in 43 per cent, Lebert in one-third, and Ladame in rather more than a third of their cases. Symptoms of mental irritation frequently precede those of depression. The irritative symptoms consist of mental excitement and those emotional disturbances which are usually known as hysterical, ideas of grandeur, with consequent extravagance, hallucinations, delusions, and outbursts of passion which may amount to maniacal fury. The symptoms of depression consist of drowsiness, apathy, loss of speech, and imbecility. The affections of speech which occur

in cerebral tumour are variable in character. Ladame found affections of speech in 45 of his collected cases.

Terminal Phenomena.—As the tumour grows in size the brain becomes compressed to such an extent that its functions become gradually abolished, and the terminal phenomena of the affection are ushered in. These consist of extreme emaciation, widely spread anæsthesia, blindness and diminution or loss of one or more of the other special senses, motor paralysis often implicating all the extremities, imbecility and deep and enduring coma.

§ 732. *Morbid Anatomy.*—The morbid growths which constitute intracranial tumours are very variable, and, regarded from the standpoint of pathological anatomy, have little or no affinity with each other, but are conveniently grouped together for practical purposes on account of their clinical affinities. The brain is surrounded by unyielding osseous walls, and the development of any foreign body within the cranium encroaches upon the space occupied by it, and consequently there is a close similarity in the symptoms caused by intracranial tumours however different in nature.

(*a*) *Varieties of Intracranial New Formations.*

(1) *Glioma.*—The gliomata form tumours which vary in size from a cherry-stone to that of the closed fist; they are vascular, of a white or greyish-red colour, and are never distinctly circumscribed from the tissues of the brain, the grey matter of which they much resemble in consistence and colour. The hemispheres of the brain are the favourite seats of gliomata, although they may appear in any part of the brain or spinal cord. Gliomata are composed of a matrix, which varies in consistence, and an abundant admixture of cells and nuclei. The cells vary in shape and size; they are sometimes round or oval, with granular contents and one or two nuclei; at other times spindle-shaped or stellate, and provided with fine processes, which are continuous with those of adjoining cells. There are two principal varieties of gliomata, the *hard* and the *soft*. In the *hard gliomata* the cells are scanty, and usually contain several nuclei. The matrix is formed of fine fibrillæ, which are more or less parallel to one another, and can sometimes be isolated into long threads. In the hardest forms the matrix is no longer formed of long, separable fibrillæ, but of a finely reticular substance, which can only be separated into short stiff fibres. At times part only of the tumour is hard, and it then contains one or more hard kernels, which may equal in density fibro-cartilaginous

tumours. True cartilaginous structure has, however, never been found in these tumours. The hard gliomata are allied in general characters to the fibromata, and intermediate forms are met with which are termed *fibro-gliomata.*

In the *fibro-gliomata* the matrix consists of fibres forming thick bundles, or exhibiting a stratiform arrangement enclosing here and there nucleated cells.

The *soft gliomata* contain more cells than the hard; the cells vary considerably in size and form, but are generally small and deficient in plasma. The matrix consists of a fibrillary network, in the interstices of which the cells are imbedded.

Transitional forms between the soft gliomata and other tumours are met with. When the number and size of the cells are increased, the tumours are allied to the sarcomata, and are therefore called *glio-sarcomata;* and when the matrix assumes a mucoid character, the tumour resembles the *myxomata.* The gliomata are sometimes richly supplied with relatively large blood-vessels, constituting what Virchow has named *teleangiectatic* glioma. This form is characterised by the tendency to hæmorrhage, which always occurs in the centre of the tumour, and the appearances presented may closely resemble simple apoplexy. Hæmorrhagic glioma usually occurs in the white substance of the hemisphere, where simple apoplexy is rarely seen; and in the former, even when the tumour is largely destroyed by hæmorrhage, a narrow zone surrounds the clot, which is sufficient to reveal the origin of the mischief.

Gliomata grow slowly, and the tumour generally attains a large size. That these tumours undergo retrogressive changes is shown by the frequent occurrence of fatty degeneration in their interior, but the changes instead of leading to a curative process are much more likely to cause hæmorrhage as soon as the absorption of the fatty *débris* lowers and removes the pressure on the vessels in the interior of the tumour. By fatty metamorphosis and softening of the intercellular substance cavities form which may be distinguished from cysts by their irregular and uneven walls. In the vicinity of tumours where the tissues are reddish and softened, fatty granules, cholesterine crystals, neuroglia nuclei, and fragments of axis cylinders may be found.

(2) *Hyperplasia of the pineal gland* is, both in external characters and in the nature of its elements, very similar to glioma. Virchow says that it forms a solid, greyish-red, slightly lobulated, or a smooth round tumour, which may grow to the size of a walnut or even larger. On section it exhibits the well known grey, moist, vascular tissue of the pineal gland, and in old persons a large number of the sand-like bodies are rarely absent. Histologically, the cell elements are somewhat larger and firmer than in the normal gland. These tumours produce pressure on the corpora quadrigemina and venæ magnæ Galeni, and that on the latter in its turn may give rise to secondary hydrocephalus.

(3) *Myxoma* is rarer in the brain than in the spinal cord and peripheral

nerves. It takes its origin, like glioma, from an overgrowth of the neuroglia, and extends uniformly in all directions by infiltration.

(4) *Solitary Tubercle*, which is by far the most common tumour of the brain, is regarded by Rindfleisch as a product of the neuroglia, and as being allied to the *fibromata*. They consist of hard nodules, varying in size from a pea to a pigeon's egg, and sometimes even larger, of grey, yellow, or yellowish-white colour and globular form. On section the interior of the nodule is yellowish and cheesy, while the outer cortex is of a reddish-grey colour, and very vascular. The thickness of the cortical layer is inversely proportional to the size of the tumour; in a tumour the size of a hazel-nut which I saw lately it was a line in thickness, and in another of the size of a walnut it was not much thicker than brown paper. The cortical tissue is continuous with the cheesy nodule on the one side and with the healthy brain matter on the other. These tumours are met with in all parts of the brain and cord, but their favourite seat is the cortical substance of the cerebrum and cerebellum, close upon the cortico-medullary boundary. This tumour is frequently multiple, and then each nodule is usually small; but when there is only one tumour it may attain a considerable size. Rindfleisch distinguishes a tubercular and a non-tubercular variety of the solitary cheesy nodule.

In the non-tubercular variety the cortex of the nodule consists of a round-celled embryonic tissue, in which nothing peculiarly tubercular can be detected. The layer of nervous matter surrounding the nodule is also infiltrated with corpuscular elements, and thus the nodule increases steadily in size. Within the zone of proliferation there is found a large development of fibres between the corpuscular elements of the embryonic tissue, rendering it dense, while the cells are entirely replaced by fibres in the centre.

The small cheesy nodules are usually multiple, and prove on minute examination to be really tuberculous. The grey zone of proliferation which surrounds them is seen with the naked eye to consist of spherical nodules, each of which corresponds in shape and size to a miliary tubercle, while the interior of the nodule consists of tubercles which have undergone the cheesy transformation. The young granules are continually produced at the circumference, and the tumour grows by the constant addition of these. When the nodule has attained a considerable size, the process of growth stops, and a fibrous envelope gradually forms round the mass, so as to completely isolate it from the surrounding brain tissue, and this condition has led some pathologists to believe that all tubercles occur in an encysted condition in the brain. The centre of the nodule sometimes softens, and occasionally the whole contents of an encysted tubercle may undergo this change. Very rarely the tubercular nodule has been found to have undergone a process of cretification. The cortex of the tumour consists of giant-cells, each being surrounded by lymphoid cells imbedded in a fibrillated reticulum.

(5) *Carcinomata.*—Cancer of the brain frequently appears as fungus

hæmatodes of the dura mater. When it originates from the outer surface of the dura mater it forces its way along the vessels into the compact tissue of the bones, and ultimately perforates them, protruding as a fungoid tumour, and pushing the scalp before it.

Simple cancer of the brain generally grows from the under surface of the pia mater, and even such tumours as appear to lie free in the substance of the brain are usually connected at some point or other with the pia mater lining an adjoining sulcus. Isolated tumours, however, do exist, but they are always secondary. Cancer is one of the most frequent of intracranial tumours. It is generally primary, and, as a rule, remains long isolated. According to Lebert, out of 48 cases 45 were primary, and of these 13 exhibited simultaneously carcinoma of other organs. Primary cancer of the substance of the brain is generally single, but occasionally there is a symmetrical appearance of a tumour in corresponding parts on each side of the brain. Several tumours are generally found in the brain in the secondary form, but these are usually small. The smallest cancerous tumours are generally found embedded in the hemispheres of the brain, in the pons, base of the brain, and the medulla oblongata. Cancer rarely occurs in the medulla oblongata, crura, and corpora quadrigemina, relatively more frequent in the optici thalami, corpora striata, and cerebellum.

Cancerous tumours destroy the neighbouring tissues by pressure and infiltration. They are surrounded by a zone of softened tissue of about a line in breadth, in which active growth proceeds. The microscope displays large cells rolled into nests, and crowded together in a matrix of fibres and blood-vessels.

Many cancers, especially those connected with bone, exhibit a calcification of their stroma. The medullary forms undergo a cheesy metamorphosis, which may lead to their being mistaken for tubercle of the brain.

(6) *Cholesteatoma*, or pearl cancer, according to Rindfleisch, "combines the structure of an epithelioma with the harmlessness of a wart or fibroid thickening." It appears to be derived from the pia mater, and is usually situated in some hollow at the base of the brain. It develops from isolated growths of the size of a mustard-seed, which blend to form masses of the size of a walnut. The tumour is enclosed by a delicate, indistinctly fibrous capsule; it has an irregular form, and its surface presents a beautiful mother-of-pearl lustre. The tumour on section is hard, pearly, non-vascular, and composed of epidermic cells arranged in concentric layers, which have undergone partly horny and partly fatty transformation. These tumours grow very slowly, and consequently may remain for a long time without giving rise to symptoms, and they only excite irritation in the neighbouring tissues in the later stages.

(7) *Papilloma* of the pia mater is occasionally met with; and a second variety of this tumour, in which there is an abundant production of mucus from the surface of the papillæ, is said by Rindfleisch to be frequently

mistaken for myxoma, and he proposes to call this variety *papilloma myxomatodes.*

(8) *Syphilomata* are usually found near the surface of the brain and develop from the perivascular sheaths. They may reach the size of a walnut or even a hen's egg. In their interior there are usually several cheesy patches, while the circumference is made up of soft jelly-like and very vascular tissue. Syphilitic gummata are made up of highly cellular embryonic tissue, with an abundant mucoid basis-substance, the cells being concentrically arranged round the vessels. Other signs of the syphilitic dyscrasia are generally found at the autopsy.

(9) *Sarcomata* occur in all varieties in the brain, and grow from the free surfaces of the interstitial spaces. They appear as hard, slightly vascular, round, somewhat nodulated tumours. The soft, cellular sarcomata present many transitions to other forms of tumour indicated by the names glio-sarcoma, myxo-sarcoma, &c. One form of spindle-cell sarcoma grows by preference from the dura mater at the base of the brain, forming tuberculated masses near the sella Turcica, and compressing the adjacent parts of the brain and the cranial nerves at their points of exit. In some sarcomatous tumours the spindle-cells are arranged in concentric layers forming nests. This form has been named "nested sarcoma" by Dr. Gowers.

(10) *Lipoma* has occasionally been met with on the inner surface of the dura mater and on the ventricular ependyma.

(11) *Psammomum* is a tumour with a basis of connective or sometimes of mucoid tissue, distinguished by its containing calcareous concretions. It usually grows from the membranes of the brain, and especially from the choroid plexus, in which situation it often contains numerous cysts. According to the most recent investigations psammomum is to be regarded, not as a distinct kind of tumour, but as a calcareous deposit in tumours of widely different structure (Dreschfeld).

(12) *Osteomata.*—If we exclude the calcifications of other tumours, true formations of bone are the rarest of all intracranial growths. Osseous formations in the dura mater, after injuries, are more common. Syphilitic exostoses, although for the most part arising from the external table, yet sometimes spring from the internal surface of the skull, and cause pressure on the brain like other tumours in the same locality.

(13) *Cystic growths* in the brain are not so common as was formerly supposed. They are most common in the pituitary body.

(14) *Angiomata* generally occur in the brain as a complication of other tumours, such as glioma. The growths on the inner surface of the dura mater, described under the name of pachymeningitis hæmorrhagica bregmatica, belong to this class.

(*b*) *Aneurisms.*

Aneurisms of the cerebral arteries are not rare. They are of various sizes, but only those which arise from the larger vessels, chiefly at the

base of the brain, will come under consideration at present. They generally arise in consequence of atheromatous degeneration of the vessels. The common termination is in rupture.

(c) *Parasites of the Brain.*

The animal parasites which occur in the cranial cavity are (1) Cysticercus cellulosæ, and (2) Echinococcus hominis.

(1) *Cysticercus Cellulosæ.*—Cysticerci of the brain generally occur, according to Rosenthal, in the parts which are richly supplied by vessels, such as the ventricles, the ganglia and their commissures, the pia mater, and the cortex of the brain. They were found 23 times in the meninges, especially the pia mater, 59 times in the cortex, 32 times in the basal ganglia and adjacent commissures, 18 times in the ventricles, 18 times in the cerebellum, 4 times in the pons, and twice in the medulla oblongata (Rosenthal). The parasite is sometimes found in other parts of the body as well as in the brain. Out of 88 cases collected by Kuechenmeister, the cysticerci were found 11 times in other parts of the body. Cerebral cysticerci are usually enclosed in a soft capsule, in which the animal may be seen with the naked eye as a small white tubercle; while its neck, with the characteristic hooklets, may be discovered on microscopic examination. Cerebral cysticerci occur with greatest frequency in places where cows pasture in fields strewn with the excrement collected in cities (Cobbold).

(2) *Echinococcus Hominis.*—Echinococcus cysts often reach a large size. In a case reported by Dr. Morgan, the cyst was as large as a walnut, and weighed 647 grammes. Of forty observations collected by Dr. Morgan, the cyst was situated 10 times in the cerebral lobes, 8 times in the cerebellum, 4 times in the ventricles, twice in the ventricles, and once in the pons. The cysts attain their greatest size in the hemispheres, and in the lateral ventricles, especially in children before the fontanelles are closed. The cyst is composed of an external fibrous membrane which encloses the parasites; its internal surface is lined by small buds, each about the size of a millet seed, and provided with the characteristic ring of hooklets. The cavity of the cyst is usually filled with a liquid, which is either clear or contains floating débris and secondary vesicles, the buds of the latter being destitute of hooks, and called acephalocyst.

§ 733. *Morbid Physiology.*—The only part of the physiology of cerebral tumours with which we are here concerned is to connect the symptoms with the effects produced by the growth upon the nervous tissues. The tumour grows from a minute point, and gradually increases in circumference, so that it is at first almost entirely latent, or only gives rise to indefinite symptoms. As the tumour increases in size it produces progressive general compression of the whole brain. In

order to make room for the increasing size of the tumour, the cerebro-spinal fluid is first removed, the blood is then squeezed out of the vessels, and the whole substance of the brain suffers pressure. It is evident, therefore, that a process of this nature will ultimately lead to gradual abolition of the functions of the brain.

But not only is the brain subjected to general compression, but the tissues surrounding the new growth are liable to special pressure, which soon leads to their destruction. The tumour itself must probably always be regarded as a destroying lesion, and consequently its direct tendency, as a local growth, is to give rise to depressive symptoms. It must, however, be remembered that the abolition of the function of a higher centre may leave the functional activity of a lower centre more unrestrained.

But although the direct tendency of the tumour is to destroy the surrounding tissues, yet its indirect effect is often irritative. The tumour acts as a foreign body, and is liable to cause hyperæmia and inflammation of the surrounding tissues. Intercurrent attacks of irritative symptoms are therefore very liable to take place in the course of cerebral tumour, but they are generally followed by a further extension of those of depression. It must also be remembered that irritation of a higher centre may produce an inhibitory action on a lower centre. But the processes set up in the surrounding tissues are not always of an irritative or inflammatory nature. An artery may be compressed and the tissues to which it is distributed may undergo ischæmic softening. The veins in the vicinity of the tumour may be compressed, giving rise to effusion of serum either into the surrounding tissues or into the ventricles of the brain. Softening as well as œdema of the surrounding tissues, in whatever way produced, must be regarded as a destroying lesion, and the symptoms depend upon the situation and not the nature of the lesion.

§ 734. *Grouping of the Symptoms.*—A review of the symptoms of intracranial tumours shows that, although they are very numerous and variable, they admit for practical purposes of the following arrangement:—(1) General and initial symp-

toms, which may be present in every kind of intracranial tumour, whatever its position; (2) Symptoms caused by the localisation of the lesion; (3) Intercurrent symptoms depending on accessory lesions; (4) Terminal phenomena.

(1) *The general and initial symptoms* consist of headache, dizziness, restlessness, and mental irritability, paræsthesiæ, various disturbances of the special senses, and convulsions. These symptoms may be present individually or in various combinations, and for a long time they may be the only symptoms complained of.

(2) The symptoms which depend upon the localisation of the tumour do not differ essentially from the symptoms caused by other focal diseases of the brain. They result from destruction of the surrounding parts of the brain; they are essentially paralytic in their character, although the loss of function may occasionally be preceded by transitory irritative phenomena. These symptoms will be more fully described hereafter.

(3) The accessory lesions which give rise to intercurrent symptoms are hyperæmia and inflammation of the surrounding tissues. The chief symptoms caused by these lesions are hallucinations, maniacal and convulsive paroxysms, and attacks of apoplexy and meningitis.

(4) The terminal symptoms are caused by gradual and increasing compression of the brain, and consist of the progressive abolition of the mental faculties, and general sensory and motor paralysis, ending in coma. In many cases of cerebral tumours death results from an intercurrent disease, from an attack of cerebral hæmorrhage, or from sudden paralysis of the respiratory centre when the tumour is situated in the pons or upper end of the medulla, or when the ventricles are distended with serum. The intensity of the symptoms is by no means proportional to the size of the tumour, inasmuch as a growth may sometimes attain a large size without giving rise to marked symptoms, while at other times a small-sized tumour may give rise to intense disturbances.

The following are the conditions on which the differences in the intensity of the symptoms appear to depend:—(*a*) Idiosyncrasies of the patient; (*b*) the position of the tumour; (*c*) the nature and rate of growth of the tumour; (*d*) the changes set up in the surrounding tissues; and (*e*) the presence of several tumours, or the existence of complications.

(*a*) *Idiosyncrasies of the Patient.*—It is well known that some men react much more actively than others to the same degree of irritation. A degree of irritation, for instance, which would not produce an appreciable effect on adults may occasion violent convulsions in children.

(*b*) *Position of the Tumour.*—Some parts of the brain are tolerant and others are very intolerant of displacement or any interference from without. The white substance of the hemispheres and the occipital lobes belong to the first category; the medulla, pons, and the internal capsule of the lenticular nucleus to the second.

(*c*) *Nature of the Tumour and its Rate of Growth.*—It may be laid down as a general rule that the intensity of the symptoms is in direct proportion to the rapidity of the growth of the tumour. The slow-growing cholesteatomata, for instance, usually attain a considerable size before giving rise to any distinctive symptoms. When the growth is rapid there is a greater flow of blood to the part, and the surrounding tissues are more liable to undergo irritative changes, while the brain has no time to accommodate itself to the new disturbance. The increased bulk of the tumour is sometimes caused not by growth of its tissue elements, but by œdema or hæmorrhage, and then it produces all the effects of a sudden injury to the brain. Conducting fibres which, if pushed aside by a slow-growing tumour, would maintain for a long time their functional integrity, are now suddenly stretched, ruptured, and irremediably damaged. Retrogressive changes within the tumours may, according to their nature, cause great variations in the symptoms. Sometimes these changes may lead to hæmorrhage and all its consequences; while at other times the tumour may by these changes become diminished in bulk, thus relieving the pressure on the brain and leading to a temporary amelioration of the symptoms.

(*d*) *Morbid Changes in the Surrounding Tissues.*—The changes set up in the tissues surrounding the tumour may either constitute discharging or destroying lesions. It is not possible to draw a clear line of demarcation between these two kinds so far as the symptoms are concerned, since the effect of a destroying lesion in the immediate vicinity of the tumour may be obscured by those of discharging lesions in remote parts. In the early stages of the growth of the tumour the discharging lesions predominate. The tumour acts as a mechanical irritant or foreign body, and it may directly irritate the part in which it is situated, or indirectly irritate remote parts by reflex action, or again its effects may be more or less diffused and general.

It is very important to observe that the symptoms of intracranial tumours frequently intermit in the early stages of the disease, and only become permanent and continuous in the latter stages when the whole brain is subjected to pressure. The reasons for this intermittence of symptoms are not far to seek. A large discharge of nervous energy is followed by exhaustion, so that the discharging lesions caused by the local irritation of the tumours are followed by exhaustion, accompanied by temporary subsidence of the active symptoms. At other times the symptoms may be caused not so much by the size of the tumour as by œdema and inflammation of the surrounding tissues, and when the latter subside the symptoms disappear for a time, although the primary lesion still persists.

(*e*) *The Presence of several Tumours and Complications.*—The variety and complication of symptoms are very much increased when several tumours are present, or when symptoms of tumour are associated with cerebral disturbance caused by an independent affection, such as Bright's disease.

§ 735. *Diagnosis.*—Intracranial tumours may be confounded with other cerebral lesions, and indeed at an early stage it is almost impossible to be sure of the diagnosis. The most important symptom of tumour is to be found in the optic discs. Many cases are recorded in which the presence of double optic neuritis was the only symptom that could lead one to the suspicion of cerebral tumour, and in which the diagnosis was subsequently justified by the progress of the case. Two cases of this kind have come under my own observation, and the occurrence of such cases has led Dr. Hughlings-Jackson to insist on the routine use of the ophthalmoscope in the examination of patients.

In tubercle the disease of the brain is generally associated with tuberculous affections of other organs, and a hereditary predisposition to the disease can usually be ascertained.

Hydrocephalus, in its chronic form, is a frequent accompaniment of tumour, especially when the latter is situated under the tentorium, where the growth is liable to produce pressure on the venæ Galeni magnæ, or to prevent the return of the cerebro-spinal fluid into the spinal canal.

Apoplexy occurs in advanced age, its onset is sudden, and it is usually associated with disease of the heart, atheroma of the vessels, and granular kidney; while the paralysis is sudden, without premonitory symptoms, and frequently followed by late rigidity in the extremities. Tumour, on the other hand, occurs at every time of life without being necessarily associated with other diseases, while the paralysis comes on slowly and increases gradually, and is preceded by other symptoms, such as violent cephalalgia, vomiting, vertigo, and neuralgia, and it is rarely followed by late rigidity. They may be further distinguished from each other by the double optic neuritis of cerebral tumour, in opposition to the rarer unilateral embolic amaurosis of apoplexy. Care, however, must be taken not to confound one form of albuminuric retinitis with the optic neuritis of cerebral tumour.

In *chronic softening* the paroxysms of headache are less frequent and intense than in tumour, while affections of the special senses and anæsthesia of the cephalic nerves occur more frequently in tumour than in softening; on the other hand the

occurrence of sudden and complete hemiplegia and aphasia is more common in softening than in tumour. Alternate and bilateral paralysis occur, according to Hasse, frequently in tumour and only exceptionally in softening.

Abscess of the brain is to some extent similar to tumour in its physical relations, inasmuch as it may produce increase of intracranial pressure, and, like tumour, the tissues surrounding the diseased focus are often affected by inflammatory attacks. Abscess usually occurs as the direct consequence of an injury, such as fractures of the skull and concussions of the brain, or associated with some other disease, such as caries of the petrous portion of the temporal bone, ozæna, foci of suppuration, diseased vessels, or valvular diseases of the heart; while tumour is never more than a remote consequence of an injury. In tumour the cephalalgia is severe, the various symptoms assume a progressive character, and there is usually a gradual extinction of the functions of the brain; or apoplexy may occur, but meningitis is rare.

Atrophy of the brain produces an early destruction of the mental activities which passes gradually into imbecility. The presence of tremors of the lips, tongue, and limbs, of epileptiform convulsions, hemiplegia or paraplegia, and loss of mental power, form a group of symptoms so characteristic that they cannot well be mistaken for those of tumour.

Hypertrophy of the brain of children gives rise to symptoms as cephalalgia and epileptiform convulsions somewhat similar to those of tumour. The large circumference of the great fontanelle, with its strong pulsation, the slow dilatation of the head, the distinct traces of rickets in the skeleton, and spasms of the larynx combine to prevent this disease from being mistaken for cerebral tumour.

Syphilis of the brain may give rise to symptoms closely simulating those of cerebral tumour, and indeed the presence of a distinct gumma induces symptoms which are identical with the symptoms of other forms of tumour. The history of the case, permanent traces of the disease such as cicatrices, the peculiar pains of the nerves and bones, epileptiform convulsions, and evidences of the presence of more than one focus of disease, are amongst the signs to be made use of in forming a diagnosis.

§ 736. *Diagnosis of the nature of the Tumour.*—It is not always possible to diagnose the nature of the tumour, although this may be done sometimes with a considerable degree of certainty. The development of *glioma* is frequently preceded by an injury to the skull, the progress of the symptoms is slow, and the illness is consequently of comparatively long duration. Hæmorrhage not unfrequently occurs into the substance of the tumour or into the surrounding tissues, and the patient is, therefore, liable to suffer from intercurrent attacks of apoplexy.

Turbercular tumour may be suspected when the symptoms of intracranial tumour occur in childhood, and when a hereditary predisposition to tubercle can be traced. The diagnosis is rendered more certain when evidence of tuberculosis in other organs or cheesy degeneration of the glands can be detected. The tumour is also more likely to be of a tubercular nature when the symptoms indicate that it is situated in the cerebellum, or that multiple lesions are present. Tubercular tumour often begins after an acute febrile disease, as measles or scarlet fever, while its progress is frequently complicated by slight attacks of meningitis.

Carcinoma of the brain is characterised by the rapid progress of the symptoms, and the presence of the cancerous cachexia or evidence of the deposition of cancer in other organs.

Sarcomata are not easily diagnosticated during life, but when the most prominent symptoms are afforded by compression of the nerves at the base of the brain sarcoma may be suspected.

Syphilomata of the brain will be subsequently described in detail.

Cysticercus cellulosæ, when situated in the brain, often remains latent for a comparatively long period. The more usual symptoms of the affection are headache and vertigo, followed by muscular spasms, epileptiform convulsions, and various mental disturbances, but distinct paralysis is rare. The convulsions caused by the presence of the parasite may at first be similar in every respect to those of idiopathic epilepsy, but in the terminal period the attacks increase in number and violence, as many as 80 to 100 daily having been known to occur during the week previous to death (Rosenthal). The psychical disturbances consist at first of illusions, delirium,

maniacal attacks, followed by melancholy, somnolency, and stupor. The diagnosis of the presence of cysticerci is rendered more probable if, in addition to the symptoms just described, the history of the case show that the patient had previously suffered from tænia, or if the patient be a butcher or pork dealer.

Echinococcus hominis, when found in the brain, does not give rise to characteristic symptoms. The most constant symptoms are headache, vertigo, vomiting, tremors, epileptiform attacks, and the usual evidences of the presence of an intracranial tumour in the optic discs. In the cases collected by Dr. Morgan the duration of the symptoms averaged one and a half years. The tumour may sometimes make its way through the cranial bones. In Reeb's case it made its way through the parietal bone, while in a case observed by Westphal two openings were found in the frontal bone through which the tumour projected; an incision having been made 90 vesicles flowed through the opening, and the case terminated in recovery. Westphal states that the diagnosis of the presence of echinococci in the brain must be made from the general symptoms of intracranial tumour appearing and disappearing alternately, œdema of the eyelids, an opening in the cranial bones through which a fluctuating tumour projects, or exploratory puncture.

Aneurism of the cerebral arteries gives rise to symptoms like those of other tumours situated at the base of the brain, nor are there any sure signs by means of which the former may be distinguished from the latter. Even auscultation of the skull has not hitherto proved of much use in the diagnosis of intracranial aneurism. If aneurism of any of the other vessels of the body co-exist with the symptoms of tumour situated at the base of the skull, then aneurism of one of the cerebral vessels may be suspected. It is probable that aneurism gives rise to more pronounced symptoms of irritation, such as intense cephalalgia, paroxysms of severe and intractable trifacial neuralgia, attacks of mania and other grave psychical disorders, than solid growths. If a patient, who has been suffering from the symptoms of tumour situated at the base of the brain, die suddenly from an attack of ingravescent apoplexy, it may be conjectured that the tumour was an aneurism rather than a

new formation. If a case, in which the patient has suffered from the symptoms of tumour situated in the anterior fossa of the skull, terminate fatally from a copious hæmorrhage from the nose, it may be assumed with considerable probability that an aneurism of the anterior cerebral artery has perforated the cribriform plate of the ethnoid bone. If pulsation and a murmur on auscultation be observed in the orbit immediately after an injury to the skull, it is probable that a communication has been established between the internal carotid artery and the cavernous sinus (Lebert).

§ 737. *Prognosis.*—With the exception of syphilitic cases, death is the usual consequence of cerebral tumours. Even a syphilitic tumour may not be amenable to treatment if it be of long standing, since irreparable mischief to the brain may have already been caused by it. Cases of cerebral tumour may sometimes terminate in sudden death through an attack of apoplexy or of convulsions, or occasionally without evident cause. In other cases the symptoms may become quiescent, the vomiting cease, the amaurosis even disappear, and the patient regard himself cured. After a time, however, the symptoms usually recur with increased intensity, and lead to a fatal termination.

§ 738. *Treatment.*—In the large majority of cases very little can be done by treatment, but even in these unpromising cases curative efforts should not be abandoned. In the earlier stages of cerebral tumours the symptoms are generally those of irritation and of local congestion, and these must be treated by cold to the head, purgatives, and occasionally by the use of flying blisters.

The cephalalgia may be combated by ice to the head, and if no relief be afforded, narcotics are to be cautiously resorted to. Subcutaneous injections of morphia will be found the most useful and reliable remedy, although small doses of belladonna have occasionally been attended with benefit. The chloride of ammonium may occasionally be found useful.

When convulsions are a prominent symptom, doses of from half a drachm to a drachm of the bromide of potassium may be useful.

With the view of promoting absorption of the morbid growth, iodide of potassium has been administered in large doses and with apparent benefit. For adults half-drachm doses may be given to begin with, and increased until a drachm is taken three times a day. Of course if there be evidence of syphilis, energetic anti-syphilitic treatment by means of mercury and iodide of potassium is indicated.

CHAPTER VI.

(II.) SPECIAL CONSIDERATION OF FOCAL DISEASES, ACCORDING TO THE LOCALISATION OF THE LESION.

1. AFFECTIONS OF THE PEDUNCULAR FIBRES AND INTERNAL CAPSULE.

a. Affections of the Pyramidal Tract.

(i.) Hemiplegia.

§ 739. Hemiplegia consists of paralysis of one-half of the body, although many of the muscles are either not implicated or only temporarily weakened. The paralysis is, as a rule, limited to the arm, leg, and part of the face.

In facial paralysis of cerebral origin the cheek on the affected side looks flat, the corresponding naso-labial fold is obliterated, the upper lip is less arched, and the angle of the mouth is lowered on the affected side, the distortion becoming more marked when the facial muscles of the healthy side contract. Paralysis of the orbicularis oris interferes with the pronunciation of the labials and with such actions as whistling and blowing out a candle. The patient can frown as usual, raise his eyebrow and eyelid and close his eye on the paralysed almost as well as on the healthy side, but is unable to perform a unilateral action like winking on the affected side. The facial paralysis begins usually to disappear in a few weeks, and sometimes in a few days, while it may persist for months. The muscles chiefly affected in facial paralysis of cerebral origin are the buccinator, orbicularis oris, and the straight muscles which pass to the angle of the mouth and to the nose on the paralysed side; while the occipito-frontalis, corrugator super-

cilii, and orbicularis oculi remain almost entirely unaffected. In facial paralysis of peripheral origin all the muscles supplied by the facial nerve below the lesion are equally paralysed.

The hypoglossal nerve is affected in most cases of apoplexy, as shown by a certain degree of difficulty in executing the movements of the tongue. On protrusion its point deviates more or less to the paralysed side, the base being dragged further forwards on the healthy side. The affection of the tongue, as a rule, disappears in a short time, but is occasionally permanent.

Some observers state that the muscles of the trunk are unaffected in hemiplegia, but the inspiratory muscles undoubtedly act less freely on the paralysed side for the first few days in severe cases.

(ii.) Hemispasm.

§ 740. The spasms which occur in connection with focal cerebral lesions are of three kinds: (*a*) *Tonic*, (*b*) *combined tonic and clonic*, and (*c*) *clonic spasms*.

(*a*) *Tonic Spasms.*—The tonic contractions which occur in connection with focal lesions of the brain may be divided into two classes: (i.) *Early* and (ii.) *late* rigidity.

(i.) *Early Rigidity.*—The contractions which occur in early rigidity may be subdivided into those which immediately accompany the hæmorrhage, and those which occur a few days after the attack. The contractions of the first kind are probably produced by irritation of the fibres of the pyramidal tract, occasioned either by rupture or partial injury. The second form of early rigidity appears in the paralysed parts a few days after the occurrence of hæmorrhage, and during the time inflammatory changes are taking place in the tissues surrounding the clot. These contractions, therefore, are probably also the result of irritation of the fibres of the pyramidal tract. Early rigidity may be so slight as only to be manifest when passive movement of the paralysed extremity is made. When the arm is flexed, for instance, if an attempt be made to straighten it, the biceps offers resistance to the movement; while at other times resistance is offered to flexion by contraction of the triceps.

The rigidity may sometimes be limited to the fingers, while at other times the arm is drawn to the side of the chest, the elbow and wrist are firmly bent, the fingers are flexed upon the palm, and all attempts to extend the limb increase the contractions, and cause pain as well as some amount of tremor or slight clonic spasm. The resistance yields occasionally under steady pressure. This form of rigidity may affect the leg as well as the arm, and then the thigh becomes flexed on the trunk, and the leg on the thigh, so that the heel is brought up to the buttock. Early rigidity generally disappears soon, but may persist for weeks or months. The affected muscles do not undergo atrophy, their faradic and reflex excitability is increased, and they become completely relaxed during sleep, although the spasm recurs immediately on the patient awaking. The appearance of early rigidity diminishes the chances of the patient's recovery, and when it continues for a long time changes take place in the muscles, tendons, and joints of the affected extremities, which ultimately leave them permanently contracted and useless.

(ii.) *Late Rigidity.*—This form of contracture is caused by descending degeneration of the fibres of the pyramidal tract, and corresponds in its essential character to the spasmodic rigidity of primary lateral sclerosis. Its most characteristic feature is the exaggeration of the tendinous and periosteal reflexes. When the lower extremity is affected the patellar-reflex is in excess, and ankle-clonus is readily elicited, and corresponding phenomena may be obtained in the upper extremity when it becomes the subject of contracture. When the loss of voluntary power is complete, the rigidity is more or less constant, although it is in most cases diminished during sleep and increased during voluntary efforts and emotional disturbances.

The attitudes assumed by the limbs affected with late rigidity differ considerably in different cases, but on the whole they conform to the rule observed in almost all spasmodic affections, namely, that flexion predominates in the upper, and extension in the lower extremity. In the most usual attitude of the upper extremity the arm is drawn towards the trunk by contraction of the pectoralis major. The forearm is semi-flexed

PLATE VI.

on the arm and pronated, the hand is slightly flexed on the forearm, and the fingers are closed. In some cases the forearm, instead of being semi-flexed and pronated, is semi-flexed and supinated. In a few rare cases the forearm is extended upon the arm, and then the forearm may either be in a state of supination or pronation (Charcot). Probably the most frequent attitude of the hand is that in which the fingers are extended at the metacarpo-phalangeal and flexed at the phalangeal joints (Gowers). The inferior extremity is, as a rule, maintained in a state of rigid extension, the foot being in the position of talipes equino-varus. In some few cases flexion predominates over extension in the lower extremity, and then the thigh becomes flexed on the trunk, and the legs on the thigh, so that the heel touches the buttock. In these cases the contracture is apt to extend to the opposite extremity, and then station and locomotion are impossible. In some cases the contracture extends to the inferior muscles of the face. The contracture is at first transitory, and only manifested when the patient laughs or cries, but after a time it becomes permanent. The angle of the mouth on the affected side is then elevated, the naso-labial fold is increased in depth, and even the eye of the corresponding side may be smaller than the healthy eye (Plate VI., 2, 3, and 4).

After a time, however, the muscles may undergo progressive atrophy, and the contractures almost entirely disappear, although the bones and ligaments having become adapted to the form in which the limb has so long been maintained the deformity persists. In these cases it is probable that the descending degeneration of the lateral column of the spinal cord has extended to the ganglion cells of the anterior grey horns. The muscles which do not suffer at all, or suffer least, from late rigidity are those that are bilaterally associated in their actions, while those acting independently of the corresponding muscles of the other side are most affected. In accordance with this rule, the muscles of the trunk remain unaffected, and the muscles of the lower extremity are less frequently and less profoundly affected than those of the upper; the superior muscles of the face generally escape, while the inferior facial muscles are occasionally attacked. The rigidity, however, is not always so fixed and

unvarying as that just described. It may never become fully established, or after having become established may undergo a considerable amount of improvement. When the rigidity has never been fully established, it may be observed that the tension becomes less when the limb is warm and greater when it is cold; that it can be diminished by gently rubbing the muscles; and that it disappears almost, if not entirely, during sleep. On the other hand, the rigidity is increased during voluntary efforts to move the limb, this effect being more marked when the patient is under observation.

Although rigidity may have become fully established, at the end of some months it gradually diminishes to such a degree that Brissaud proposes to call the condition *latent* contracture. The patient may perform all the simple movements of the limb, and probably with undiminished power, but whenever his attention is specially directed to the movements, as when he wishes to perform any manual operation requiring a little dexterity, the muscles instantly become rigid, the fingers are flexed on the palm, and the deformity which was present during the period of fixed contracture reappears. It may also be shown that the tendon reflexes continue exaggerated, although the muscular tension has in great part disappeared. It is not yet ascertained whether the disappearance of the muscular tension is due to a corresponding repair of the fibres of the injured pyramidal tract on the opposite side, or to the establishment of new connections with the cortex of the brain on the same side through commissural fibres in the cord.

(*b*) *Combined Tonic and Clonic Spasms.*—The cases just described, in which a slight degree of muscular tension permanently present in the affected extremity is associated with marked spasm on a voluntary effort being made to move the limb, form a fitting transition to those cases in which a fixed tonic contraction of some of the muscles is associated with clonic contractions of others. In the combined tonic and clonic varieties of post-hemiplegic motor disorders, the muscular contractions are at first entirely like those which occur in late rigidity, but after a time some of the muscles implicated become the subjects of clonic spasm.

Varieties.—The combined tonic and clonic spasms of hemi-

plegic limbs consist of the following varieties :—(i.) Intermittent tremor, and (ii.) Choreiform movements.

(i.) *Intermittent Tremor.*—The most usual form of tremor observed in hemiplegic limbs corresponds with that which is observed in spastic spinal paralysis. The tendon reflexes are exaggerated, and the tremor is induced when the muscles are put upon the stretch by any attempt at voluntary movement or otherwise. This kind of tremor is therefore similar to that described as "spinal epilepsy" in lateral sclerosis of the spinal cord, and, like the latter, it is associated with descending sclerosis of the pyramidal tract. The tremor is, like that of multiple sclerosis, absent during repose.

The muscles of hemiplegic limbs are liable to be affected with fibrillary contractions similar to those which occur in progressive muscular atrophy and amyotrophic lateral sclerosis. It is probable that muscular atrophy is always associated with these contractions in hemiplegia, and that the descending changes of the pyramidal tract have extended to the ganglion cells of the anterior grey horns of the cord.

(ii.) *Choreiform Movements.*—Clonic choreiform spasms of the extremities may either precede or follow an attack of hemiplegia, the former being named *pre-hemiplegic*, and the latter *post-hemiplegic chorea* (Weir Mitchell, Charcot). In *pre-hemiplegic chorea* the patient complains of a feeling of numbness and feebleness of the extremities of one side, his gait becomes hesitating and irregular, and the upper extremity of the affected side is attaked by choreiform movements. These symptoms may continue for some days, when complete hemiplegia, usually associated with hemianæsthesia, is either suddenly or gradually established. *Post-hemiplegic chorea* occurs in partially but never in completely paralysed limbs, and usually appears simultaneously with a marked diminution of the paralytic symptoms. The clonic spasms as a rule become gradually established as motor power returns, although they sometimes supervene suddenly, and appear to be sometimes induced by a strenuous voluntary effort on the part of the patient to move the paralysed limb. Clonic spasms occur more frequently in the arm than in the leg, and when they exist in both they are more severe in the former, while if the leg be exclusively affected

the arm is usually completely paralysed. The muscles of the face are sometimes affected by those spasms, causing various distortions, which become greatly increased when the patient laughs or cries.

The movements affected by choreiform spasm in the upper extremity are, in decreasing order of frequency, the special movements of the fingers and thumb, flexion and extension of the wrist, pronation and supination of the forearm, extension and flexion at the elbow, and movements at the shoulder-joint. The interossei are particulary liable to be affected by choreiform spasm, and consequently the movements most frequently observed consist of varying degrees of flexion and extension at the metacarpo-phalangeal articulations, associated respectively with extension and flexion at the phalangeal articulations. The movements induced by these spasms are of wider range than those of hemiplegic tremor, resembling in this respect the movements of chorea. They are disorderly and irregular, and may or may not continue during complete repose; they cease during sleep, and become much aggravated during voluntary efforts to perform a definite movement with the affected limb, such as that of raising a glass of water to the mouth. When the lower extremity is affected, the whole body may be thrown into a state of agitation during locomotion.

Two forms of *post-hemiplegic chorea* may be distinguished: (*a*) the post-hemiplegic chorea of adults; and (*β*) the spastic hemiplegic of infancy. The spastic hemiplegia of infancy may consist of a purely tonic spasm of the muscles without any admixture of clonic spasms, although the choreiform variety is probably the more common.

(*a*) *Post-hemiplegic Chorea of Adults.*—The post-hemiplegic chorea of adults and the corresponding affection of infancy differ in various ways. In the former the history of the case shows that the attack of hemiplegia which preceded the appearance of the clonic spasms occurred during adult life, or at any rate not in early infancy. The attack of hemiplegia may have occasionally become gradually established when due to the slow growth of a tumour, but as a rule it has come on suddenly with apoplectic symptoms. An examination of the patient may reveal valvular disease of the heart, or there may be a history

of injury to the head. The post-hemiplegic chorea of adults, apart from the history, differs from that of infancy in the co-existence of hemianæsthesia in the former and its absence in the latter. The anæsthesia extends over the lateral half of the body; and all forms of sensibility, including the special senses, are more or less affected. Three distinct cases of the post-hemiplegic chorea of adults have come under my own observation. All the patients were comparatively young men, their ages ranging from 25 to 33 years. The attack of hemiplegia, which had preceded the choreiform movements, occurred in each several years previously to my seeing them. Two of the patients presented evidence of slight stenosis of the mitral valve, and in the third the apoplectic attack had been induced by a fall on the head. The attitude assumed by the affected arm was very similar in the three cases. There was marked tonic spasm of the posterior third of the deltoid in all of them, so that the elbow was abducted from the trunk to the extent of about two and a half inches, while it was also drawn backwards considerably behind the posterior plane of the body. The forearm was slightly flexed on the arm and strongly pronated, the hand was slightly flexed on the forearm, while the fingers were kept in constant movement by clonic spasms of the interossei muscles. There was also a certain degree of spasmodic pronation and supination of the forearm and flexion and extension of the hand in all; while in one, irregular jerking movements of the forearm, hand, and fingers occurred when the patient attempted to grasp any object with the paralysed hand. A marked feature presented by these cases was the fact that each patient carried the affected hand in the out pocket of his coat, in order to arrest its disorderly movements. In this position the upper arm was directed downwards, outwards, and backwards from the shoulder, the elbow being considerably removed from the trunk and behind its posterior plane, the forearm was slightly bent on the arm, and the back of the hand was pressed closely against the hip.

In the three patients referred to the tactile sensibility of the palm and fingers of the affected hand was remarkably deficient. When the patient was asked to close his hand on a coin placed on the palm, with his eyes closed, he could not say whether he

had or had not the coin in his grasp; and when the coin was withdrawn before the closure of the fingers, it was amusing to observe his puzzled expression on opening his eyes and hand when he found the latter empty. The patients could be pricked with a pin over half the face, trunk, and over extremities on the affected side almost without pain. In one of these cases all forms of cutaneous sensibility, and the muscular sense, were diminished over half of the body on the affected side, the senses of taste and smell were also diminished on the corresponding side, but the senses of hearing and sight were not affected to an appreciable extent.

(β) *Spastic Hemiplegia of Infancy.*—In the spastic hemiplegia of infancy the lesion which determines the paralysis occurs during birth, or in early infancy. The paralysis appears sometimes to have become established before birth, but cases of this kind are exceptional. It is, however, not uncommon to ascertain, on inquiry from the parents, that the patient who is affected with the spastic hemiplegia of infancy suffered from repeated convulsions accompanied by unconsciousness for the first two or three days after birth, although it may not be observed that the child is paralysed on one half of the body till some time subsequently. In the majority of these patients, however, the onset of the disease dates from the age of two to three months to that of four or five years. The most usual history is that after an illness of indefinite character extending over a few days, or without any warning, the child has been taken with convulsions. These convulsions, as a rule, have recurred repeatedly for some hours or days, the child remaining in the meantime in a state of unconsciousness. In many cases this is the only history which can be obtained, but where the parents are intelligent it may be ascertained that the convulsions were limited to the side of the body which had subsequently become paralysed. Many infants doubtless die during these convulsions or a few days after, but in the cases which survive it is soon observed that one half of the body is paralysed. The hemiplegia in these cases pursues the usual course, contractures become established, and choreiform movements may or may not make their appearance during partial recovery, but when once these movements appear they remain

permanent. So far, then, these cases present nothing peculiar except that the disease dates from childhood, that it is ushered in by convulsions and profound unconsciousness, and that the motor paralysis is not accompanied by hemianæsthesia.

In the spastic hemiplegia of childhood, however, it is soon observed that the intellect of the patient, however bright the child may have been previous to the attacks of convulsions which marked the onset of the disease, has become markedly defective. This form of hemiplegia is, indeed, nearly always associated with some degree of idiocy.

Another marked peculiarity of the affection is that at a certain age the hemiplegia becomes associated with epilepsy. The epileptic attacks generally begin when the patient is from seven to fifteen years of age, and at first are usually limited to the paralysed side of the body, and may not be attended by decided loss of consciousness. In the case of a well-developed girl fourteen years of age, under my care, suffering from the spastic hemiplegia of childhood, the epileptic attacks began when she was eight years of age. The right half of the body was paralysed, the arm being more paralysed than the leg, both limbs were somewhat rigid, but neither manifested any choreiform movements. The epileptic attack always began by movements of the paralysed arm; these soon extended to the muscles of the mouth on the same side, and then to the paralysed leg. In most attacks this patient became unconscious for a few moments, and then got up and walked about as if nothing had happened. In some, however, the convulsions were limited to the paralysed arm, with probably a slight extension of them to the angle of the mouth, but the leg remained free, and there was no loss of consciousness. The patient was once reported by the nurse to have walked across the ward during an attack, holding down the convulsed and paralysed arm with the opposite hand. In old-established cases the convulsions may become general, but it may be observed that they retain a unilateral character at the commencement of the attack, and the patient usually describes a unilateral aura.

The aura is often described as a sensation beginning in the paralysed hand, and ascending along the arm to the shoulder and head, when unconsciousness supervenes. At other times

the sensation begins in the paralysed leg, and ascends successively to the arm and head. In several cases under the care of Mr. Hardie, which I examined recently in Crumpsall Workhouse, three of which are represented in *Plate* VI., *Figs.* 2, 3, and 4, the patients could not give any account of an aura; and so far as I could judge from the account given by their attendants, the convulsions did not assume a unilateral character. In all these cases marked idiocy was present, so that the presence of an aura could not be determined from the inability of the patients to describe it. In one case of the kind, with choreiform movements of the paralysed hand, sent to me by Mr. Cullingworth, the patient had an epileptic attack once while I was examining her. I could not observe that the convulsions assumed a pronounced unilateral character at any time during the attack. On cross-examining her with respect to the aura, she positively denied that she had had any warning whatever of impending attacks; but after a time she volunteered the statement, "When the fits began first I used to have a creeping feeling in the leg, which came up to the arm," at the same time pointing successively to the paralysed leg and arm.

These patients also present other phenomena which are worthy of notice, the most remarkable of which is an arrest of development of the paralysed limbs, generally implicating the corresponding side of the face. The circumference of the paralysed extremities is usually less than that of corresponding parts of the opposite limbs, although not always so. Where a limb is subject to violent choreiform movements, the muscles may become hypertrophied so that its circumference exceeds that of the corresponding healthy extremity. But even under these circumstances it may be found that the circumference of the bones on the affected side is less than that of the sound side, and that the enlargement is limited to the muscles. Each of the long bones of the affected extremities may be from $\frac{1}{4}$in. to 1in. shorter than the corresponding bones of the affected side, and even the clavicle of the paralysed side may be from $\frac{1}{4}$in. to $\frac{1}{2}$in. shorter than the opposite clavicle. The diminution of size of half the face may extend to all the features, including the eyebrows, eyelids, half of the nose, the cheek, and half the mouth.

(*c*) *Clonic Spasms.*—The post-hemiplegic motor disorders, which consist of clonic spasms unaccompanied by tonic contractions of the muscles, are (i.) continuous or remittent tremor, (ii.) choreiform movements (athetosis), and (iii.) jerking movements on voluntary effort (hemiataxia).

(i.) *Continuous or Remittent Tremor.*—The tremor which has already been described as occurring in hemiplegic limbs was associated with increased muscular tension, excess of the tendon reflexes, and only occurred when a voluntary movement of the limb was made. In the form of tremor about to be described, muscular tension, if present in excess at all, is not a prominent feature of the case, the tendon reflexes are not exaggerated, the tremor is continuous at least during waking hours, and instead of being exaggerated it may be diminished or arrested by a voluntary effort. We have seen that the first form of tremor is like that which is observed in sclerosis in patches; while the second form is in all essential particulars like the tremors of paralysis agitans. A case of the latter kind has been described by Grasset. The tremors, which continued during repose, were accompanied by sensations of heat like those complained of by patients suffering from paralysis agitans. A case is described by Leyden in which tremors occurred in the right arm, momentarily arrested by a voluntary effort, while there was complete absence of any paralysis or contractures and of sensory disturbances. A round sarcomatous tumour was found in the left optic thalamus. By the courtesy of Dr. Leech, I had an opportunity of showing to the members of the British Medical Association at the Manchester meeting, a case in which one-half of the body presented all the characteristics of a moderately advanced paralysis agitans. The tremors extended to the right foot, leg, and one-half of the trunk; while the attitude of the forearm, fingers, and thumb was quite characteristic. The symptoms supervened nine months previously, and were preceded by a slight attack of confusion, not amounting to unconsciousness, followed by slight paresis of the right side of the body.

(ii.) *Athetosis.*—An affection has been described by Hammond under the name of *athetosis*, in which the patient is unable to maintain the fingers or toes in fixed positions. The

fingers and toes in this affection are maintained in continuous slow movement, and are made to assume various distorted positions. These movements are not always limited to the fingers and toes, but extend to the hand and foot, and occasionally even to the muscles of the neck and face. No motor weakness has been recognised, the movements are only to a slight extent under the control of the will, they usually persist during sleep, and are not accompanied by contractures. Cases of the affection have been described by Allbutt, Currie Ritchie, Fisher (Boston, U.S.), Gairdner, and others, while Claye Shaw and Dreschfeld have drawn attention to the analagous condition sometimes observed in the limbs of imbecile children. Oulmont has written a valuable monograph of the whole subject.

The appearance of the clonic spasm is in almost all cases preceded by a distinct attack of hemiplegia, and when no decided paralysis can be ascertained to have been present the history of the case shows that the patient has suffered from an attack of convulsions and unconsciousness.

Hemianæsthesia is described as being present on the affected side in some of the reported cases, while a certain degree of numbness of the same side is frequently mentioned. In a considerable number of cases the condition of sensibility is not mentioned, and probably no special attention was directed to the point.

The affected extremity usually presents vaso-motor disturbances. It is red or livid, moist, and colder than the corresponding extremity.

The affected hand or foot is also frequently atrophied; although the muscles which are affected by the spasm may undergo a certain amount of hypertrophy. The electric contractility of the affected muscles varies in different cases, being sometimes normal, at other times enfeebled or increased.

Oulmont has observed an unusual degree of relaxation of the ligaments and joints of the affected extremities.

A *bilateral athetosis* has been described by Oulmont. It does not differ essentially from the unilateral affection, except that the muscles of the face appear to be more liable to be implicated to a greater extent in the former. The bilateral affection is generally associated with idiocy, but may occur without

this complication. It is not, according to Oulmont, preceded by apoplexy or hemiplegia, and is unaccompanied by sensory disturbances.

(iii.) *Hemiataxia.*—A case has been described by Dr. Gowers in which there was great inco-ordination of the right arm during voluntary movement, while there was complete absence of permanent rigidity and spontaneous spasm. The patient had suffered from a slight attack of apoplexy followed by hemiplegia a year and a half before he came under observation, but the paralysis had disappeared, a slight weakness of the arm, leg, and face alone remaining. The ataxic movements of the arm became exaggerated on the eyes being closed. Tactile sensibility was diminished in the right arm, but sensibility to pain was normal. In a somewhat similar case recorded by the same observer the autopsy revealed "a puckered cicatrix" passing through the left thalamus from the one side to the other. A case in which ataxic movements occurred in the right hand is also described by Grasset. The patient had a series of apoplectic attacks followed by hemiplegia and a certain embarrassment of speech. The ataxic movements were limited to the right arm, the paralysis being more marked in the face and arm than in the leg. At the autopsy three centres of softening were found in the left hemisphere. The first occupied the region of the lenticulo-striate artery; the second was in the optic thalamus close to its ventricular border; and the third was found in the thalamus close to the posterior portion of the internal capsule.

§ 741. *The Hemiplegic Walk.*—When the muscles of the paralysed lower extremity have acquired a certain degree of rigidity, the patient is able to walk by the aid of a stick, even if the voluntary paralysis of the affected side remain complete. The patient leans towards the healthy side, but is prevented from falling over to that side by the support of the stick; the pelvis and hip-joint of the paralysed side are elevated by contraction of the abductors of the opposite thigh, so that the weight is taken off the paralysed extremity. When the paralysed lower extremity, say the right leg, is the active one, the line of gravity is carried over to a slight extent to that side; but instead of reaching the centre of the paralysed foot,

it remains midway between it and the end of the stick, so that the weight of the body is maintained partly by the paralysed lower extremity and partly by the healthy arm through the stick. The healthy or left lower extremity is now quickly moved forwards a step, an unusual degree of flexion of the thigh upon the body taking place in order to avoid the necessity of carrying the line of gravity too far to the paralysed side. The left leg now becomes active, and the paralysed one must be moved forwards. The manner in which this movement is executed depends upon the degree of paralysis and of muscular rigidity present. If the paralysis be almost complete and the rigidity not great, the extremity is partly swung and partly dragged round mainly by the contraction of the inward rotators of the healthy limb. Contraction of these muscles causes the pelvis to rotate forwards on the hip-joint of the healthy side, and consequently the opposite hip-joint, dragging after it the paralysed leg, is moved forwards. This forward movement is aided by a further elevation of the right hip-joint caused by contraction of the abductors of the opposite thigh, and sometimes by a slight backward inclination of the trunk by means of which the distance between the points of origin and insertion of the flexors of the thigh on the body is increased.

If a high degree of contracture with talipes equinus be present, the paralysed lower extremity is moved forwards much in the same manner as has already been described in the case of primary lateral sclerosis. When once the weight of the body is taken off the paralysed extremity the heel becomes elevated, and the toe during the forward movement, which takes place in a semicircular manner, makes a characteristic scraping noise.

If tremors or choreoid movements be present in the paralysed lower extremity, the hemiplegic walk may become modified in such numerous ways as to render it impossible to comprise the different varieties which may be presented in a single description.

b. Affections of the Sensory Peduncular Tract and Optic Radiations of Gratiolet.

Hemianæsthesia.

§ 742.—In cerebral hemianæsthesia the affection develops suddenly after an attack of apoplexy, or gradually as the result,

for instance, of the progressive growth of a tumour. The sensibility is diminished over the whole of one-half of the body, face, and extremities, including the accessible mucous membranes as well as the skin. The abolition of sensation is sometimes incomplete, and then cutaneous analgesia or thermo-anæsthesia may be present, while tactile sensibility remains unaffected. At other times the anæsthesia of the skin and mucous membranes is complete, and even muscular sensibility and muscular sense are abolished. The patient, for instance, does not feel deep pressure, strong contraction of the muscles may be produced by the faradic current without causing pain, and when his eyes are closed he is unable to describe the position in which the affected extremities may be placed by passive movements, and is not aware when his attempted voluntary movements are forcibly prevented. The patient can walk without difficulty when his eyes are closed, but by slight pressure upon the affected side he may be easily induced to walk in a circle while under the impression that he is walking in a straight line.

One-half of the mucous membrane of the tongue, mouth, and veil of the palate, and the conjunctiva of the same side, are insensitive, but the cornea retains its sensibility.

The affected side is colder, and the prick of a pin does not bleed so readily as on the opposite half of the body.

The cutaneous reflex actions may be abolished on the side affected, while the deep reflexes are retained.

The *senses of taste and smell* are both abolished on the affected side.

The *sense of hearing* is also diminished, and in some cases there may be complete unilateral deafness.

The *sense of sight* is impaired but not abolished, but hemiopia has not been observed when the lesion is limited to the internal capsule. The acuteness of vision may be tested in the usual manner by Snellen's scale. There is also concentric restriction of the field of vision, and the perception of certain colours may entirely cease (dyschromatopsia).

§ 743. *Morbid Anatomy and Physiology.*—It is impossible to separate lesions of the internal capsule and crusta from

those of the ganglia by which they are surrounded. Since the days of Willis and Morgagni up to a few years ago, paralysis of one-half of the body has been associated with disease of the corpus striatum. This doctrine had indeed received a shock upwards of twenty years ago, from the observations of Türck, who showed that hemianæsthesia of the opposite side of the body might result from disease situated in the posterior part of the lenticular nucleus. It was also suggested by Meynert and Broadbent that some of the fibres of the crusta passed upwards to reach the cortex of the brain without being in any way connected with the basal ganglia; and Charcot, with his usual readiness and skill in utilising the details of anatomical research for clinical purposes, suggested, and soon proved by observation and analysis of cases, that both hemiplegia and hemianæsthesia are caused by injury of the direct fibres which lie between the basal ganglia, and not by lesions of the ganglia themselves. We have already seen that the fibres of the posterior third of the posterior segment of the internal capsule are sensory; that those of its middle third connect the mechanisms in the cortex of the brain and spinal cord which regulate the fundamental actions; that those of the anterior third of the posterior division connect the mechanisms which regulate the specialised actions; and that those in the knee and the anterior segment of the capsule connect the mechanisms which regulate the most specialised actions. Speaking broadly, it may be said that the fibres of the middle third of the posterior segment of the capsule are concerned in regulating the actions of the trunk, lower extremities, and probably the general actions of the upper extremities; that the fibres of the anterior third of the posterior segment are concerned in regulating the more special movements of the hand as an organ of prehension, and probably also the movements of rotation of the head and neck, along with the associated ocular movements; and that the fibres of the knee of the capsule and the adjoining part of the anterior segment of the capsule are concerned in the regulation of the movements of facial expression, articulation, and the most special movements of the hand, as those of writing.

Of all the arteries of the brain the lenticulo-striate artery is, according to Charcot, the one which is most liable to rupture.

This artery lies, as we have seen, between the external capsule and the external surface of the third division of the lenticular nucleus. When this vessel ruptures, if the hæmorrhage be small, it may lodge between the external capsule and the lenticular nucleus, and give rise to no symptoms (Charcot). The vessel, however, being a comparatively large one, the hæmorrhage, as a rule, extends beyond these limits. It is sometimes directed upwards between the external capsule and the lenticular nucleus, and may then extend for a considerable distance into the centrum ovale. Under these circumstances the fibres of the internal capsule become ruptured at their point of emergence from between the basal ganglia where they form the foot of the corona radiata. Hæmorrhages in this situation may be so extensive as to extend upwards to the summits of the

FIG. 242.

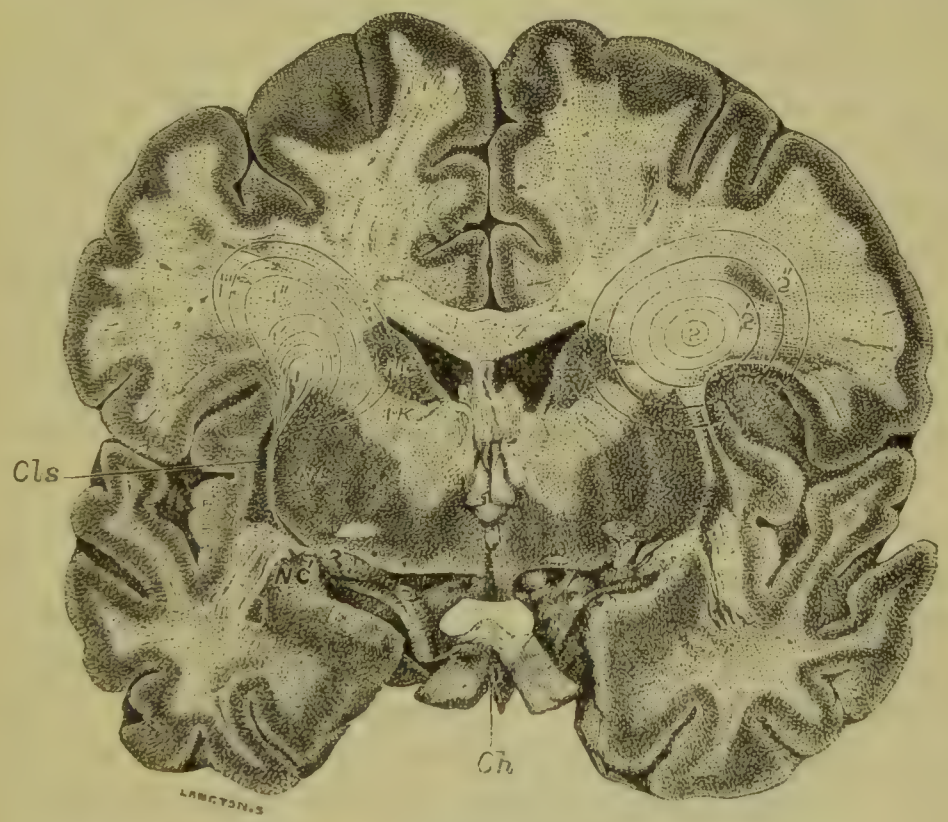


FIG. 242 (Modified from Charcot). *Vertical Section of the Brain a little behind the Knee of the Internal Capsule*, showing the effects of rupture of the lenticulo-striate artery.—*NC*, Head, and *NC'*, Tail of the caudate nucleus; *Ch*, Chiasma; *NL*, Lenticular nucleus; *IK*, Internal capsule; *Cls*, Claustrum; 1, The most frequent position in which the lenticulo-striate artery is ruptured; 1', 1'', 1''', Progressive extension of the hæmorrhage producing compression and rupture of the fibres of the pyramidal tract (hemiplegia); 2, Primary focus in the internal capsule; 2', 2'', 2''', Successive extension of the clot.

ascending frontal and parietal convolutions, while the cortex of the Island of Reil is compressed by the clot, but the external capsule is rarely ruptured. At other times the hæmorrhage is directed inwards through the grey matter of the lenticular nucleus; and if it be large, it must impinge upon and rupture the fibres of the internal capsule, and when these fibres give way the hæmorrhage may make its way into the lateral ventricles, then through the foramen of Monroe into the third, and through the aqueduct of Sylvius into the fourth ventricle.

If the hæmorrhage remain limited to the space between the external capsule and lenticular nucleus, it produces no symptoms during life; but when it makes its way into the substance of the lenticular nucleus, or into the centrum ovale above the nucleus, the fibres of the pyramidal tract are compressed, and hemiplegia of the opposite side of the body results. If the fibres of the pyramidal tract, however, remain intact, the patient will recover more or less completely from the paralysis. A case which came under my observation several years ago was that of an old man who died a few hours after being knocked down by a cab when crossing a street. The left lenticular nucleus was completely destroyed, and its usual position was occupied by a cyst containing serous fluid. No good history of the case was procurable, but he was not supposed to be suffering at the time of the injury from any form of paralysis. A still more striking case will be subsequently described, in which both lenticular nuclei were converted into cysts, the symptoms during life being those of bulbar paralysis without any evidence of paralysis of the extremities. When the hæmorrhage remains limited to the lenticular nucleus, not only does the patient ultimately recover the full use of his limbs, but the apoplectic symptoms during the attack are slight. The patient complains of giddiness, there may be vomiting, and confusion of ideas, but he does not lose consciousness, or the loss is transitory. When, however, some or all of the fibres of the internal capsule rupture, the larger size of the clot produces a more profound immediate effect, while injury to the fibres of the pyramidal tract gives rise to a paralysis which remains permanent. The degree and extent of the paralysis will, of course, depend upon the extent of the injury done to the motor tract. It is

probable that the first form of early rigidity occurs during the time the fibres of the tract are being stretched or ruptured by the hæmorrhage; the second form of early rigidity is again probably caused by irritation of these fibres, caused by inflammatory changes in the tissues surrounding the clot; while late rigidity is caused either directly or indirectly by descending degeneration of the ruptured fibres. But if the hæmorrhage make its way either between the ascending longitudinal fibres of the corona radiata, so that a large clot forms in the centrum ovale, or if it rupture into the lateral ventricle, profound symptoms of coma supervene, and the patient dies in a short time.

We have seen that the comparatively unyielding wall formed by the external capsule directs hæmorrhage from the lenticulostriate artery inwards, and consequently the full force of the blood will impinge against the internal capsule at a point a little behind its knee, or at the point where embryological considera-

FIG. 243.

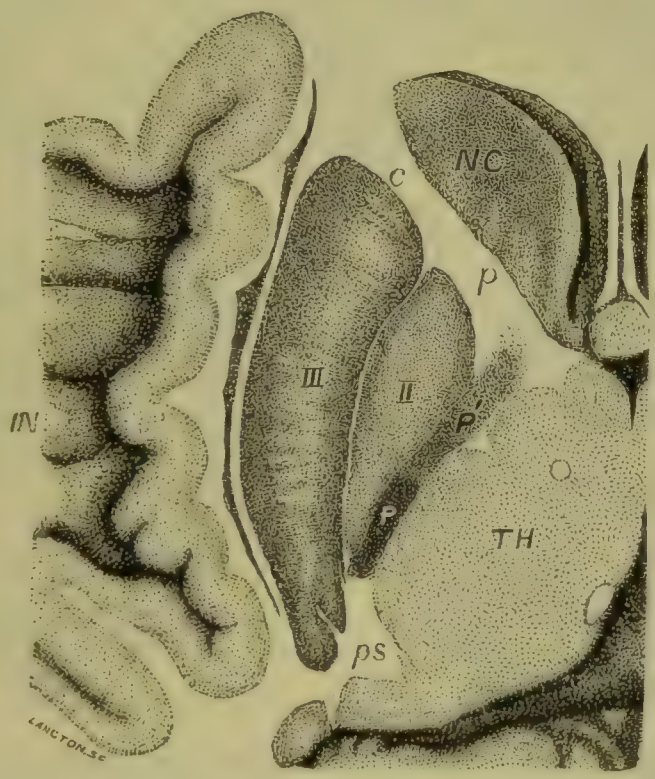


FIG. 243. *Horizontal Section of the Basal Ganglia and Internal Capsule in an embryo of nine months.*—*NC*, Caudate nucleus; *TH*, Optic thalamus; *IN*, Island of Reil; *II*, *III*, Second and third segments of the lenticular nucleus; *ps*, Sensory peduncular tract; *P*, Fundamental, and *P'*, Mixed portion, and *p*, Geniculate fasciculus of the pyramidal tract; *c*, Anterior segment of the internal capsule.

tions had led us to believe those fibres to pass, which connect with each other the nervous mechanisms in the cortex and spinal cord that regulate the movements of the hand. In hæmorrhage from this artery, therefore, the upper extremity is more paralysed than either the lower extremity or face. Rupture of the anterior branches of the artery may injure the anterior segment of the capsule to a greater extent than the posterior segment, and then facial paralysis predominates. The fibres which conduct those impressions from the cortex which cause rotation of the head and eyes to the opposite side probably also pass in the anterior third of the posterior segment of the internal capsule, and on the side of the capsule which adjoins the lenticular nucleus, and they also must be ruptured by a moderately-sized

FIG. 244.

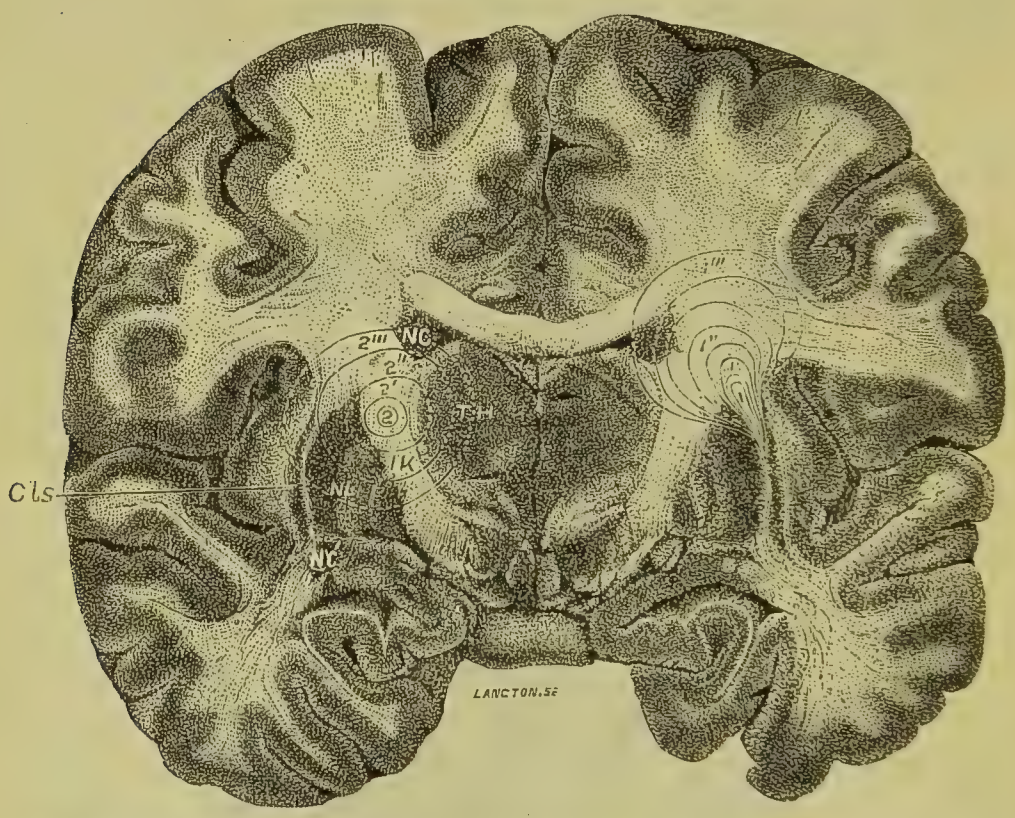


FIG. 244 (Modified from Charcot). *Vertical Section of the Brain on a level with the Posterior Part of the Internal Capsule*, showing the effects of rupture of the lenticulo-optic artery (hemianæsthesia).—*NC*, *NC'*, Tail of the caudate nucleus; *NL*, Lenticular nucleus; *TH*, Optic thalamus; *Cls*, Claustrum; 1, Primary focus in the posterior part of the external capsule (hemianæsthesia); 1′, 1″, 1‴, Progressive extension of the primary focus causing compression or destruction of the internal capsule; 2, Primary focus in the internal capsule (hemianæsthesia); 2′, 2″, 2‴, Successive extension of the focus.

hæmorrhage of the lenticulo-striate artery, but the conjugate deviation which results is as usual only a transitory symptom (§ 90). Hæmorrhage of the lenticulo-optic artery is also directed inwards against the fibres of the internal capsule by the unyielding walls of the external capsule, and its greatest force impinges against the posterior half of the posterior segment of the capsule. It is evident, therefore, that hæmorrhage from this vessel will tend to injure the sensory peduncular fibres and the fibres of the fundamental mechanism, but inasmuch as the muscles of the trunk are bilaterally associated, the paralysis resulting from injury of the latter fibres will be more marked in the leg than in any other part of the body. An analysis of clinical records had led Dr. Hughlings-Jackson long ago to conclude that the form of hemiplegia in which the leg is more profoundly affected than the arm is generally associated with hemianæsthesia. The fibres of Gratiolet are not usually affected in hæmorrhage from the opto-striate artery, and consequently the special senses are not always implicated in the anæsthesia.

The anterior segment of the internal capsule is frequently injured by lesions of the head of the caudate nucleus, the resulting hemiplegia of the opposite side being thus more marked in the face than arm, and in the arm than leg, while sensibility is seldom affected.

Cases are recorded of lesions of old date having been found at the autopsy without paralytic symptoms having been present during life (Nothnagel, Samt). In a case of this kind recently described by Honegger there were no descending changes observed in the crusta, medulla oblongata, or spinal cord, although the fibres of the middle third of the posterior segment of the internal capsule in the left hemisphere appear to have been in great part destroyed.

The internal capsule may be injured by lesions of the optic thalamus. Hæmorrhage from the posterior internal optic artery, if small, does not appear to give rise to any definite symptoms, and certainly not to permanent paralysis. A large hæmorrhage from the vessel generally makes its way into the cavity of the ventricle, and death results in a short time. Lesions in the region of distribution of the posterior external optic artery are liable to implicate the fibres of the external and posterior

extremity of the crusta and their continuations through the internal capsule. The path of least resistance to the passage of hæmorrhage from the vessel appears to be upwards and inwards; and as the internal capsule lies below and to the outside of the thalamus, its fibres are never injured to the same extent by hæmorrhages from this vessel as they are in those which take place into the lenticular nucleus. Hemiplegia is, therefore, not a prominent feature of lesions of the optic thalamus, and when it occurs it is seldom complete. The sensory peduncular fibres, and the optic radiations of Gratiolet, are very liable to be injured by lesions in the region of distribution of the posterior external optic artery, and consequently complete hemianæsthesia with implication of the special senses is a frequent symptom. When the lesion occurs in the pulvinar, the external geniculate body is apt to be implicated, and then bilateral hemianopsia of the opposite side results. When the lesion is situated more anteriorly close to the internal capsule, the fibres of the pyramidal tract suffer injury, and hemiplegia results. The hemiplegia is usually associated with a certain degree of hemianæsthesia, and after a time choreiform movements are apt to become established in the paralysed limbs. In six cases of post-hemiplegic chorea collected by Raymond, in which a post-mortem examination was obtained, the lesion was situated in every instance in the posterior part of the optic thalamus, and involved the fibres of the internal capsule; and in two cases of pre-hemiplegic chorea reported by him, the lesion was situated in the same locality. In a case of pre-hemiplegic chorea reported since then by Grasset, several lesions were found in different regions of the hemispheres, but one of these occupied the external margin of the optic thalamus close to the internal capsule.

The lesions which have been found to give rise most frequently to hemichorea are yellow cicatrices, the remains of old hæmorrhages, or softening from occlusion of the posterior external optic artery, although choreiform movements have occasionally been observed during the growth of tumours in this region. It is evident, therefore, that the symptoms depend, not upon the nature of the lesion, but on its localisation. The symptoms do not appear to depend upon lesion of the optic thalamus

itself, inasmuch as they are never present, unless some of the fibres of the sensory-peduncular and pyramidal tracts are injured, nor does it even appear to be caused by injury of the sensory fibres, since hemianæsthesia with bilateral hemianopsia may be present without being associated with choreiform movements. It would seem, therefore, that injury to some of the fibres which lie in front of the sensory peduncular tract is the cause of hemichorea. That some of the fibres of the pyramidal tract are always injured in these cases can scarcely be doubted, inasmuch as the clonic are always associated with tonic spasms, and exaggeration of the tendon reflexes, the latter symptoms being those which are always associated with disease of the pyramidal tract. Two probable explanations of the clonic spasms present in these cases suggest themselves to my mind. The first is that fibres connecting the cerebrum with the cerebellum are injured by these lesions, so that the normal proportion between the outgoing discharges which regulate the tonic (cerebellar) and the clonic (cerebral) actions of the body is lost. The second is that the injured fibres all belong to the pyramidal tract, and that those which suffer most are related to the more fundamental and not to the more special functions, as in disease of the lenticular nucleus. We have seen that the more fundamental actions are regulated from the convolutions near the longitudinal fissure, while the more special movements are regulated from the convolutions bordering the Sylvian fissure; and it is therefore manifest that the fibres which descend in the corona radiata from the former will pass along the optic thalamus side of the internal capsule, while those which descend from the latter will pass on the side of the capsule next the lenticular nucleus. The effects produced by destructive processes in any structure whatever must differ greatly according as the foundations or the latest-formed portions are the first to be injured. It appears to me, therefore, that partial injury done to the fundamental motor mechanism while the accessory one is left unaffected would be very likely to cause the phenomena of hemichorea. In such an event the usual tonic contractions and exaggerated tendon reflexes would result from injury of the pyramidal tract, while the apparatus of the more voluntary and special actions, although still uninjured, would

act in an irregular manner owing to the damage done to the fundamental apparatus.

The lesions found in cases of unilateral athetosis, although not always strictly limited to the region of the posterior external optic artery, have often been in its vicinity. In three cases of athetosis observed by Charcot the lesion was situated in the posterior extremity of the optic thalamus in one, the posterior part of the caudate nucleus in a second, and the most posterior part of the corona radiata in a third. The lesions of all these cases were situated in such positions that the same system of fibres which are implicated in post-hemiplegic chorea would be likely to suffer damage, and consequently athetosis must generally be regarded as a minor degree of post-hemiplegic chorea. In a case observed by Landouzy an old focus of softening was found in the portion of the lenticular nucleus which adjoins the internal capsule. In another, observed by Gnauck, the co-existence of sensory disturbances in the region of distribution of the fifth nerve on the side opposite to the spasmodic movements rendered it probable that the lesion was situated in the lateral half of the pons. It is, therefore, probable that the lesion in athetosis may occupy different positions in the vicinity of the pyramidal tract. The position occupied by the lesion in all cases rendered it probable that the fibres of the pyramidal tract are never completely ruptured, and consequently there are no descending changes in the cord and no muscular rigidity during life. The fibres of the tract are, however, likely to have suffered partial injury by being involved in a cicatrix or other morbid change, and the impulses which pass through them become consequently irregular.

Direct Cerebral Paralysis.—Although the paralysis of the extremities is usually situated on the side of the body opposite the lesion in the brain, it is occasionally situated on the same side, and is then called direct paralysis. The most reasonable supposition in these cases is that the pyramidal tracts do not decussate as usual in the medulla oblongata. The usual method of crossing is that from 91 to 97 per cent of the fibres cross over to the lateral column of the opposite side of the cord, while from 9 to 3 per cent pass downwards in the columns of Türck of the same side. Flechsig, however, has shown that

the proportion of fibres which decussate is very variable, and he has even found that it occasionally fails altogether. It is, therefore, probable that the decussation may fail in cases of direct paralysis, although this has not yet been proved by dissection.

The lesions observed in the spastic hemiplegia of childhood scarcely belong to the category at present under consideration, inasmuch as they primarily involve the cortex of the brain, while the internal capsule is only secondarily implicated. In infantile hemiplegia the lesion is situated in the convolutions of the motor area of the cortex. The primary lesion, consisting probably of a local encephalitis sometimes following an injury, local softening, or hæmorrhage, gives rise to extensive secondary changes. In some cases a large loss of substance has been observed, causing various deformities of the skull when it occurs in early life, or leading to hydrocephalus in order to fill up the vacant space. At other times a puckered cicatrix may be found at the seat of the primary lesion, while the hemisphere has undergone a diffused consecutive atrophy. The fibres of the pyramidal tract in connection with the diseased focus undergo descending degeneration, and to it the spastic condition of the paralysed extremities is either directly or indirectly due. Bilateral athetosis appears also to be due to partial atrophy of the motor area of the cortex, both hemispheres being probably inplicated. The considerations which favour this opinion are that the affection is either congenital or becomes established in early infancy, that it is associated with some degree of imbecility or idiocy, and that there are no sensory disturbances.

CHAPTER VII.

(II.) SPECIAL CONSIDERATION OF FOCAL DISEASES, ACCORDING TO THE LOCALISATION OF THE LESION (Continued).

2. CORTICAL LESIONS.

a. Lesions in the Area of the Middle Cerebral Artery.

(i.) Monospasms and Unilateral Convulsions.

§ 744. Irritative lesions of the cortex are characterised by unilateral convulsions or monospasms. Lesions of various kinds may cause irritation of the cortex, the most common of these being localised meningo-encephalitis, tubercle, syphilitic gummata and other tumours, cicatrices of wounds and spicula of bone, and of these the syphilitic are by far the most frequent lesions. The tissues in the immediate neighbourhood of the main focus of disease are maintained in a state of irritation, and are consequently supplied by an usually large quantity of blood. The ganglion cells of the grey substance absorb an undue supply of nutriment, so that they discharge themselves in a sudden and explosive manner (Hughlings-Jackson). But we have already seen that explosive discharges of nervous energy are followed by exhaustion and consequent paralysis of the muscles implicated in the convulsion, and accordingly unilateral convulsions are often followed by temporary paralysis of the convulsed limbs. It must be remembered that an irritative lesion is frequently associated with a destroying one. A syphilitic gumma, for instance, destroys the portion of the cortex in which it is situated, while it maintains the surrounding tissues in a state of irritation. It is not, therefore, unusual to find a certain degree of permanent paralysis associated with unilateral convulsions.

Unilateral convulsions were first distinguished clinically and their varieties accurately described by Bravais, although he did not recognise their pathological significance. Similar observations were made by Bright and Wilks, who surmised that these convulsions were due to local disease. The pathology of these spasmodic affections was first clearly recognised by Hughlings-Jackson, and it was in explanation of these convulsions that he first suggested the idea of the existence of motor centres in the cortex, an idea which has been so fruitful to pathology.

In some cases the spasm is limited to one limb or to the side of the head (monospasm); in other cases it begins in one limb (protospasm), and extends to the other or to the head, to the half of the body, or the convulsions may become bilateral and generalised. Another characteristic of these convulsions is that they are either not attended by loss of consciousness or the convulsion begins before the patient becomes unconscious, so that he is afterwards able to describe a motor aura.

§ 745. *Varieties.*—The following are the clinical varieties of unilateral convulsions:—

(*a*) *Crural monospasm or protospasm*, in which the spasms are either limited to the leg, or begin in it, the arm being next attacked and the face last.

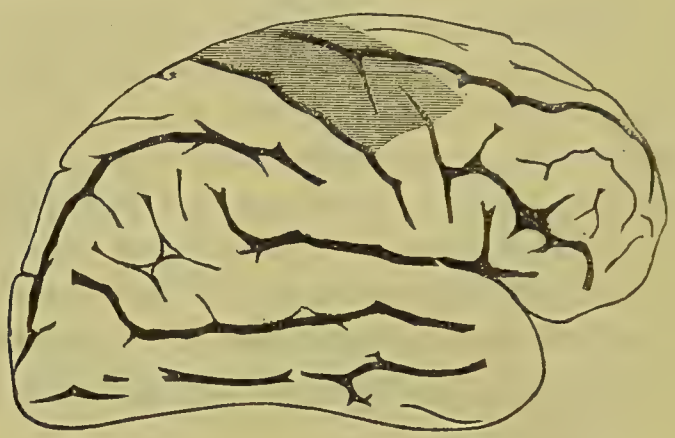
(*b*) *Brachial monospasm or protospasm*, in which the spasms are either limited to the arm, or begin in the arm, the face being next implicated and the leg last.

(*c*) *Facial monospasm or protospasm*, in which the spasms are either limited to the side of the face or begin in the face, the arm being next implicated and the leg last.

(*a*) *Crural Monospasm or Protospasm.*—There are not many uncomplicated cases on record in which the spasms were limited to the leg, or invariably began in the leg, and in which a post-mortem examination was obtained. Ferrier quotes a case recorded by Broca of crural monospasm caused by injury to the left side of the skull, which was cured by trephining, but the exact position on the brain is not mentioned. Charcot and Pitres quote a case from Griesinger of frequently recurring spasm of the leg and arm. Numerous cysticerci were found in the brain, the largest of which occupied the superior part of the ascending parietal convolution of the opposite side. Several small cysts were found on the frontal and parietal surface of the same hemisphere. Hughlings-Jackson reports a case in which the fits were often limited to the leg, and always began there. The leg became gradually weaker after each attack, and

finally became permanently paralysed. A tumour was found at the upper and posterior part of the left frontal lobe, about two inches in diameter, extending from the posterior extremities of the first and second frontal convolutions backwards to the fissure of Rolando. In another case recorded by the same author, the convulsions began in the left great toe, and were often limited to the left leg. A syphilitic lesion was found at the upper part of the ascending parietal convolution and over several of the adjacent convolutions of the parietal lobule. Bourneville describes a case of the hemiplegia of infancy, in which the convulsions began by tremors and twitching in the left or paralysed leg. The cortex of the right hemisphere

Fig. 245.



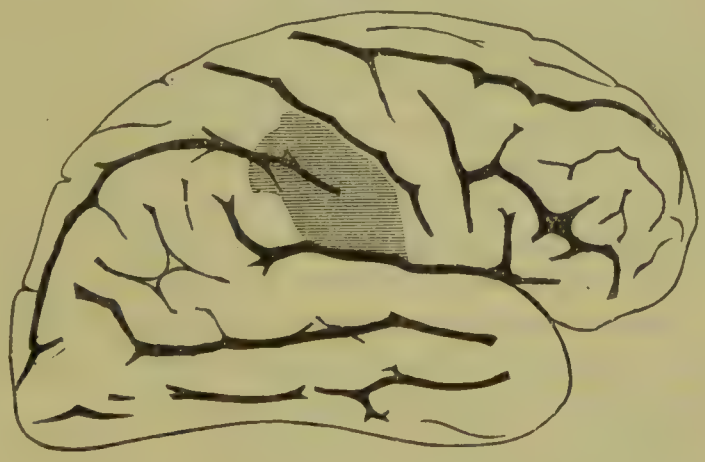
was found atrophied in front of the fissure of Rolando in the superior half of the ascending frontal, the posterior extremities of the first and second frontal (*Fig.* 245), and the whole extent of the paracentral lobule.

(*b*) *Brachial Monospasm or Protospasm.*—Several cases are recorded in which the spasm is either limited to or begins in the arm. Instances of this kind have been recorded by Dr. Hughlings-Jackson. In the case of one man who suffered from repeated convulsions limited to the right arm with subsequent paralysis, a nodule was found situated at the posterior extremity of the first frontal convolution of the left hemisphere. In another case, in which the spasms were almost similar to those observed in the last case, a nodule was found situated at the posterior extremity of the first frontal convolution where it joins the ascending frontal. The spasm in this case began in the shoulder and went down the arm, contrary, Dr. Jackson thinks, to the usual order. In a third case the spasm invariably began in the left thumb, and a tumour of the size of a hazel-nut was found under the grey matter at the posterior extremity of the third frontal convolution of the right hemisphere. In a fourth case the spasms began in the right arm, and occasionally in the right side of the face, and the patient had suffered from a transitory attack of left hemiplegia. In the left hemisphere adhesion was found between the dura mater and the brain, over "the lower part of the ascending frontal and ascending parietal convolutions, to a trifling extent to the hinder part of the third frontal and

several of the convolutions of the upper wall of the fissure of Sylvius behind the ascending parietal." In the right hemisphere, the side opposite the paralysis, a mass was found behind the fissure of Rolando, but has no bearing upon our present subject. In a fifth case temporary right hemiplegia supervened after a unilateral convulsion. Convulsions recurred repeatedly, beginning in the little finger of the right hand, occasionally in the right side of the face, and always followed by slow and hesitating speech. A syphilitic tumour of considerable size was found in the cortex about the junction of the frontal and parietal lobes, surrounded by an area of softening in the posterior extremities of the frontal, ascending frontal and ascending parietal convolutions, and partly of the Island of Reil. A case of partial epilepsy is reported by Ballet and Lalesque in which the spasms began in the right hand. Paresis of the right arm supervened, the right side of the face and tongue being also implicated to a slight degree as the case progressed. Some degree of embarrassment of speech was also present before death. At the autopsy three small hydatid cysts were found in the cortex of the left hemisphere, one being situated about the middle of the ascending frontal convolution, the second at the junction of the middle and lower thirds of the ascending parietal convolution, and the third at the posterior extremity of the second frontal convolution.

A case of brachial protospasm, caused by syphilitic disease, has been recorded by Dr. Dreschfeld, in which I conducted the post-mortem examination, confirming the diagnosis made by Dr. Dreschfeld during life. The attacks began "by sudden clenching of the fist, flexing of the wrist, and

Fig. 246.



pronation of the forearm of the left side, the corresponding angle of the mouth being at the same time drawn downwards. This sudden tonic spasm lasted for several seconds, and was then followed by a few clonic spasms of the same extremity and a slight tremor of the arm, the patient being at the same time agitated and pale, but perfectly conscious." The dura mater was found adherent to the brain on the right side over the greater part of the ascending parietal convolution and the supra-marginal lobule (*Fig.* 246).

The case of a boy, three months old, is reported by Mr. Cullingworth, who developed cerebral symptoms somewhat suddenly nearly four months subsequently to an injury to his head. The symptoms began by screaming and elevation of temperature. A few hours later it was observed that the left arm and hand were flexed and rigid, and this was soon followed by conjugate deviation of the eyes to the right. The dura mater was found thickened and adherent to the bone over a small area of the right hemisphere immediately to the right of the longitudinal fissure. The cortex underlying the adhesions was reddened and softened, the softened part involving the upper portion of the ascending frontal convolution. A layer of pus was found over the whole surface of both hemispheres and the greater portion of the cerebellum.

Charcot and Lépine describe a case of partial epilepsy beginning in the left arm in which after death a hæmorrhagic focus was found situated in the posterior part of the first right frontal convolution. In another case of partial epilepsy beginning in the left arm, described by the same authors, an old focus of softening was found between the first and second frontal convolutions of the right hemisphere where they adjoin the ascending frontal convolution; while in another case described by them, the convulsions began in the right arm, and a small focus of disease was found in the superior part of the ascending parietal convolution of the left hemisphere. A case is described by Glicky, in which the convulsions began in the left arm, but subsequently involved the left half of the body; a glioma was found which had destroyed the two ascending central convolutions and the paracentral lobule on the right side. Mahot reports a case of partial convulsions beginning in the fingers of the left hand, in which a tuberculous mass was observed imbedded in the substance of the right ascending frontal convolution in its middle third. Berger reports the case of a woman who suffered from convulsions of the right arm with subsequent weakness of the same, the convulsions after a time became general, and the right arm was completely paralysed, while there was weakness of the muscles of the leg and face. A sarcoma growing from the dura mater had penetrated into the cortex of the brain over the left ascending frontal convolution, opposite the posterior extremity of the second frontal convolution. Burresi describes a case of partial epilepsy of the left arm followed by paresis, and at last by complete paralysis; a tuberculous mass was found in the fissure of Rolando.

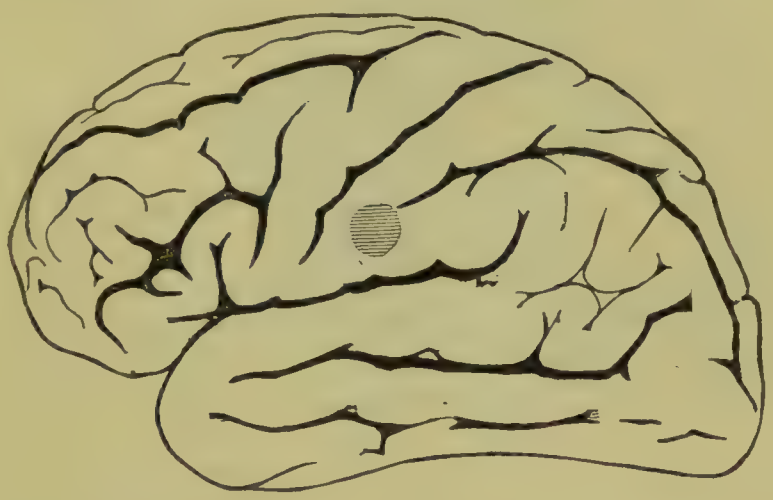
(*c*) *Facial Monospasm or Protospasm.*—The case of a French soldier is described by Hitzig, who, two months after a bullet wound on the right side of the head, suffered from clonic spasms followed by paralysis of the left side of the face and tongue. An abscess was found in the cortex of the right hemisphere situated in the inferior part of the ascending frontal on a level with the third frontal convolution.

Wernher reports a case in which there were convulsions of the muscles of the face, neck, forearm, and of the extensors and flexors of the fingers, all on the right side. The lesion was situated in the cortex of the left

hemisphere in the inferior part of the ascending frontal convolution near the fissure of Sylvius.

The case of a woman is described by Dr. Bramwell, who, after a cranial injury received some years previously, began to have right-sided convulsions. The convulsions always began in the right platysma, and were often almost entirely confined to this muscle. A spiculum of bone was found projecting from the inner table of the skull, and causing a limited lesion of the inferior margin of the ascending parietal convolution (*Fig.* 247).

Fig. 247.



Seeligmuller describes a case of epileptiform convulsions of the right half of the face, followed after a time by facial paralysis. At a somewhat later period the right arm became convulsed, and afterwards paralysed. A sarcomatous tumour was found in the ascending parietal convolution, which probably began to grow at its lower extremity and progressed upwards.

These cases tend to show that convulsions, either limited to or beginning in the face, are caused by a lesion situated in the inferior part of the ascending frontal and parietal convolutions, the portion which adjoins the fissure of Sylvius.

(ii.) Cortical Paralyses and Monoplegiæ.

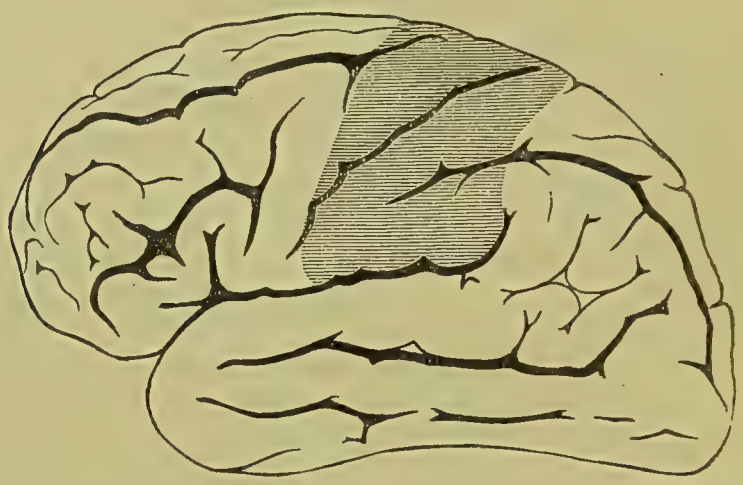
§ 746. It is now well established that destructive lesions of the cortex of the brain cause permanent paralyses. Destroying lesions of the motor area of the cortex may be divided into (1) *General lesions*, extending over the greater part of the area; and (2) *Partial or localised lesions*, limited to small portions of it (Ferrier).

(1) *General or Extensive Lesions (Hemiplegiæ).*—Extensive

lesions of the cortex give rise to complete hemiplegia, similar in all essential particulars to that resulting from disease of the internal capsule.

A case of complete hemiplegia of the right side of six years' duration is described by Lépine in which there was total destruction, caused by yellow softening, of the ascending parietal convolution and partial destruction of the Island of Reil, ascending frontal convolution, and of the anterior part of the superior and inferior parietal lobules of the left

Fig. 248.



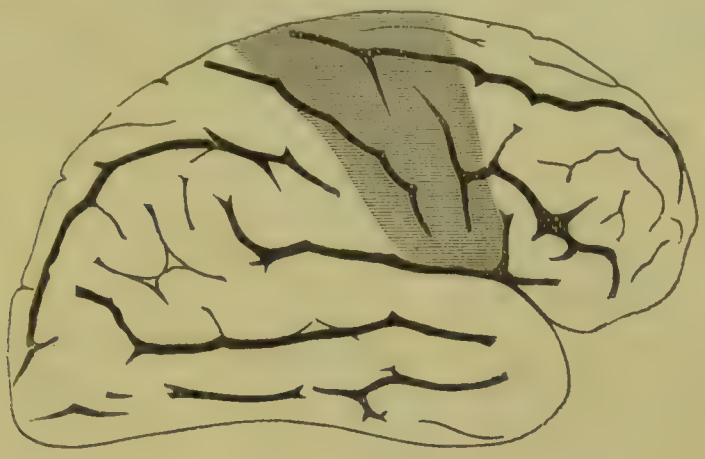
hemisphere (*Fig.* 248). Secondary degeneration was traced in the left half of the pons and in the left pyramid of the medulla. Duret reports a case of complete right hemiplegia supervening in the course of a meningo-encephalitis. A thick fibro-purulent exudation occupied the three frontal convolutions of both sides, but it extended on the left over the ascending frontal and ascending parietal convolutions, the lobule of the *pli courbe*, and the parietal lobule.

A case of right hemiplegia with aphasia of one year's duration, accompanied by late rigidity of the paralysed limbs, is reported by Charcot and Pitres, in which a patch of yellow softening was found, involving the whole of the ascending frontal, the base of the third frontal, and the whole of the ascending parietal convolutions, along with the inferior parietal lobule and the two posterior digitations of the Island of Reil in the left hemisphere. The basal ganglia were normal. Secondary degeneration was observed in the crus, pons, and anterior pyramid on the same side.

A case is quoted by Trousseau which occurred in the *clinique* of Charcot, in which left hemiplegia existed for three months; the ascending frontal and third frontal convolutions, and Island of Reil in the right hemisphere were found softened. Secondary degeneration was traced in the crus, pons, and pyramid of the same side as the lesion, and on the

opposite side of the spinal cord (*Fig.* 249). Cases of this kind might be multiplied, but it would occupy too much space to narrate them. The following cases are examples of hemiplegia, caused by lesions of the centrum ovale.

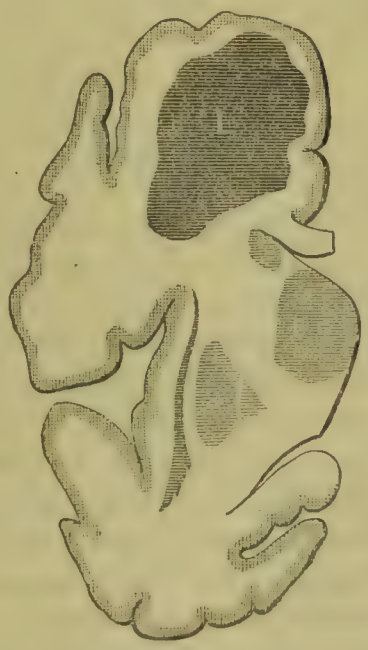
Fig. 249.



A case of right hemiplegia, with late rigidity of the paralysed limbs, is related by Hodgson, in which a cavity of considerable size was found in the centrum ovale of the left hemisphere, situated between the anterior horn of the lateral ventricle and the Island of Reil. The rest of the brain was normal.

Dussaussay describes a case, quoted by Pitres, of right hemiplegia

Fig. 250.

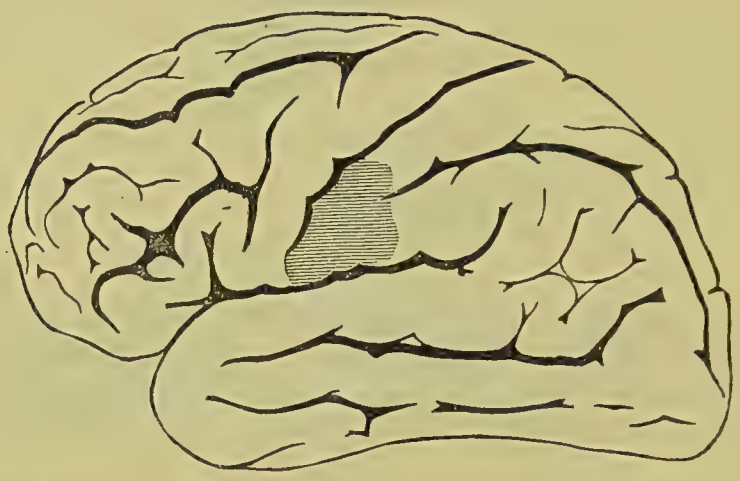


with conjugate deviation of the eyes to the left. A cavity was discovered in the centrum ovale of the left hemisphere, containing dark, coagulated blood (*Fig.* 250). The cavity was limited internally by the grey substance

of the paracentral lobule, superiorly and externally by the grey substance of the ascending frontal and parietal convolutions; in front it extended to the præ-central fissure, and behind to the posterior border of the ascending parietal convolution; while it was separated inferiorly from the corpus striatum by a layer of white substance 1cc. in thickness.

Dr. Ringrose Atkins has recorded a case of right hemiplegia due to embolism, in which, in addition to a patch of softening at the lower extremity of the ascending parietal convolution (*Fig.* 251), there was a

Fig. 251.



focus of softening two inches in diameter in the centrum ovale, extending from a point 2¼ inches behind the apex of the left frontal lobe to a point 3¼ inches outward to the apex of the occipital lobe. The basal ganglia were normal.

(2) *Partial or Localised Lesions of the Motor Area of the Cortex—Monoplegiæ.*

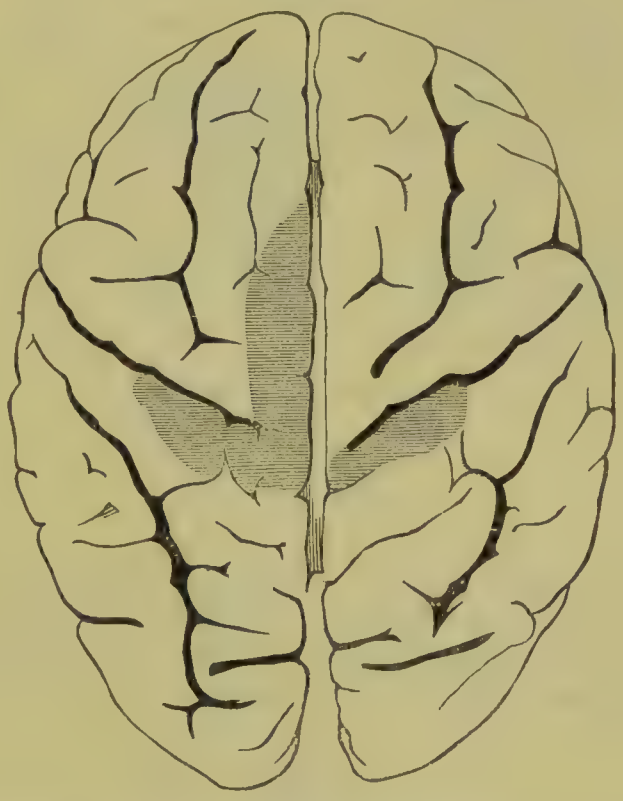
(*a*) *Crural Monoplegia.*—The recorded cases of disease of the cortex in which the paralysis was limited to the leg are not numerous. A sufficient number are reported to render the existence of a cortical centre for the regulation of the movements of the lower extremity more than probable, even from clinical evidence alone and in the absence of the more elaborate proof afforded by experiment on animals.

Löffler describes the case of a Danish corporal, who was struck by a bullet at the superior and posterior extremity of the left parietal bone, close to the sagittal suture. The right leg was immediately paralysed, and the right arm on the seventh day after the accident. On trephining, recovery took place, the arm being first restored and then the leg. In another case reported by the same author, fracture of the summit of the right parietal bone was followed by paralysis of the left leg.

The case of a woman, aged 76 years, is reported by Oudin, in which there was paralysis with contractures and arrest of development of the

right lower extremities, dating from the age of nine and a half years, and following a fall. At the autopsy the median parts of the superior surface of the hemispheres were found to present a remarkable asymmetry. The posterior extremity of the first frontal and superior extremity of the ascending frontal convolution were remarkably atrophied on the left hemisphere, while the corresponding parts of the right hemisphere were normal and of comparatively large size. The superior portions of the ascending parietal convolutions were atrophied on both sides, although the atrophy in the left hemisphere was more pronounced than in the right. The anterior extremity of the superior parietal lobule was also involved in the atrophy on the left side (*Fig.* 252).

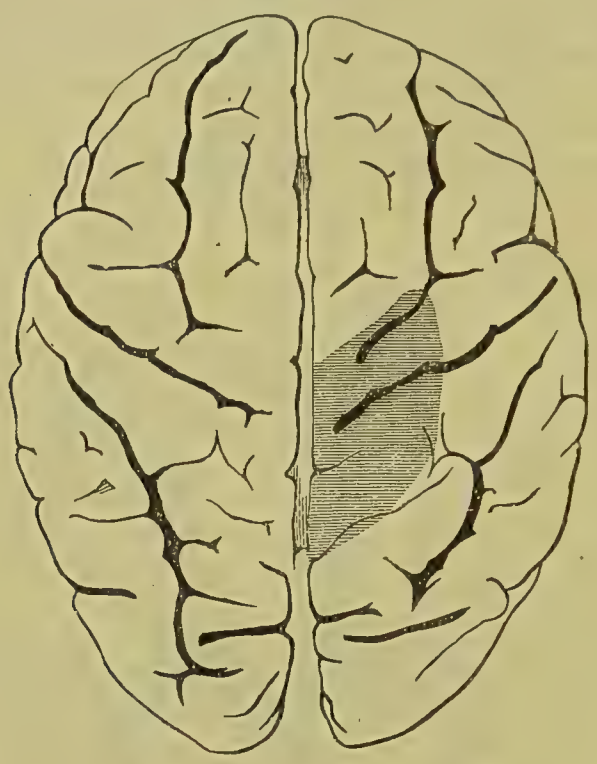
Fig. 252.



Dr. Haddon, of Manchester, records a case in which paralysis remained limited to the left leg for five months, but after a time the left arm also became paralysed. After death a tumour three inches in diameter was found connected with the dura mater, situated to the right of the middle line, compressing the subjacent hemisphere, and destroying the upper extremities of the ascending frontal and parietal convolutions, as well as the postero-parietal and paracentral lobules (*Figs.* 253 and 254). The case of a man, æt. 40 years, is reported by Dr. Ferrier, in which the symptoms of general tuberculosis were complicated by monoplegia of the left lower extremity. The paralysis was strictly limited for four days to the left leg, but subsequently extended to the left arm. The patient died a month subsequently to the appearance of the paralytic symptoms, and at the

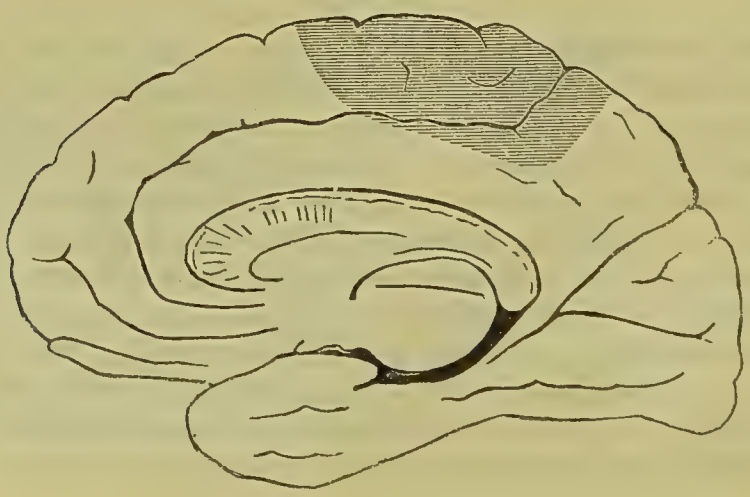
autopsy the pia mater over the upper margin and internal aspect of the right hemisphere on both sides of the fissure of Rolando was merged into a caseous mass, which could not be removed without tearing the cortical substance. The lesion occupied the quadrilateral lobule on the internal

FIG. 253.



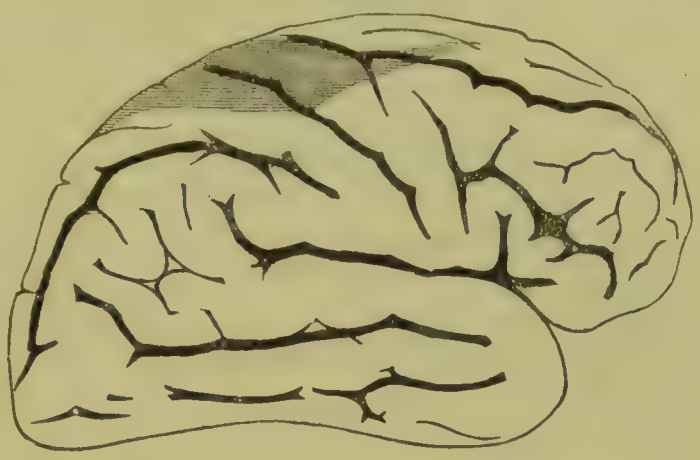
aspect of the hemisphere, and the upper extremities of the ascending parietal and frontal convolutions on its superior and external aspect, the portion of the cortex implicated corresponding to the areas marked 1 and 2 on the monkey's brain (*Figs.* 230 and 231).

FIG. 254.



(*b*) *Brachio-crural Monoplegia.*—Paralysis of the leg and arm are frequently associated in disease of the cortex. Charcot and Pitres describe a case of paralysis with rigidity of the limbs of three years' duration, in which a patch of softening was found at the upper extremity of the fissure of Rolando on the convex surface of the right hemisphere

FIG. 255.



(*Fig.* 255). Hughlings-Jackson reports a case of paralysis of the left extremities caused by a glioma situated in the superior part of the fissure of Rolando, and comprising the ascending parietal convolution and the

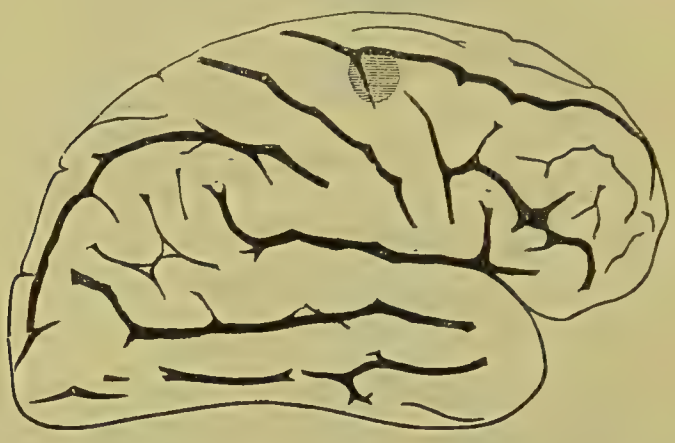
FIG. 256.

parietal lobule. A larger number of examples of brachio-crural monoplegia resulting from cortical disease might be quoted, but these must suffice. Pitres describes a case of paralysis with unilateral convulsions of the

left extremities in which a focus of softening was found, not in the cortex, but in the centrum ovale, immediately beneath the posterior extremity of the first frontal convolution (*Fig.* 256, L), and extending backwards underneath the superior parietal lobule.

(*c*) *Brachial Monoplegia.*—A case of paralysis of the left arm is described by Pierret in which a centre of softening was found in the cortex of the right hemisphere at the point where the second frontal joins the

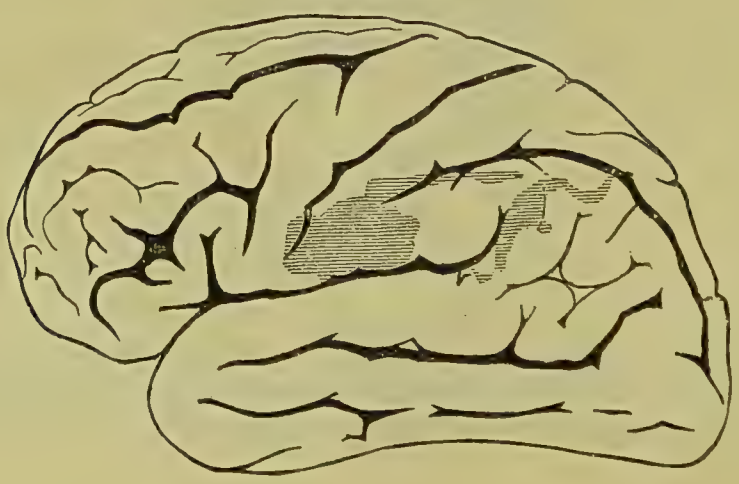
Fig. 257.



ascending frontal convolution (*Fig.* 257). Boyer records a case in which the arm and leg became suddenly paralysed, the paralysis of the arm alone remaining permanent. Death took place five years subsequently to this attack, and a patch of atrophy was found on the right hemisphere in the ascending frontal and parietal convolutions, with an extension of the lesion to the temporo-sphenoidal lobe.

A case of paralysis of the right hand and arm is reported by Ringrose Atkins supervening a few days before death in a patient suffering from general paralysis. The cortex was softened in the middle of the ascending frontal and parietal convolutions, the lesion also extending backwards along the anterior edge of the supra-marginal gyrus as shown in *Fig.* 258.

Fig. 258.

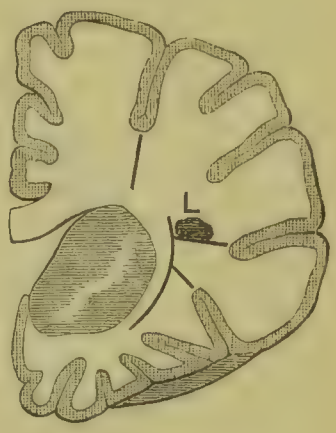


Decaisne has collected a large number of cases of brachial monoplegiæ, but it would occupy too much space to quote more cases at present.

It may be observed in passing that the central convolutions of the opposite hemisphere have been found atrophied in cases of long-standing amputation (Chuquet, Boyer). The results obtained have not, however, been very definite. Dr. Gowers found in a case of congenital absence of the left hand the middle part of the ascending parietal convolutions in the right hemisphere distinctly smaller than the corresponding convolutions in the left, and a somewhat similar case has been recorded by Bastian.

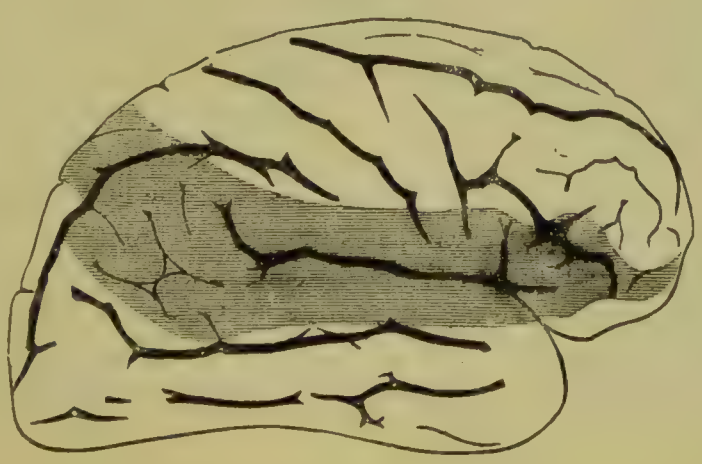
(d) Brachio-facial Monoplegia.—Paralysis of the face and arm are not uncommonly associated. When the left hemisphere is the seat of the lesion, these cases are usually associated with aphasia. Dieulafoy records a case of paralysis of the face and arm in which the autopsy revealed a hæmorrhagic focus, the size of a nut, situated in the ascending frontal convolution on a line with the third frontal convolution. Troisier mentions a case of paralysis of the arm and face in which tubercular granulations and congestion were found immediately posterior to the third frontal convolution. Landouzy describes a case of slight paralysis of the inferior facial muscles and of the arm caused by a spot of tubercular meningitis occupying the inferior part of the fissure of Rolando, and the inferior half of the two ascending convolutions. Pitres quotes from Anton Frey a case in which there was paresis of the left arm and of the left side of the face; the autopsy showed a focus of softening in the medullary fibres at the junction of the middle frontal with the ascending frontal convolutions (*Fig.* 259).

Fig. 259.



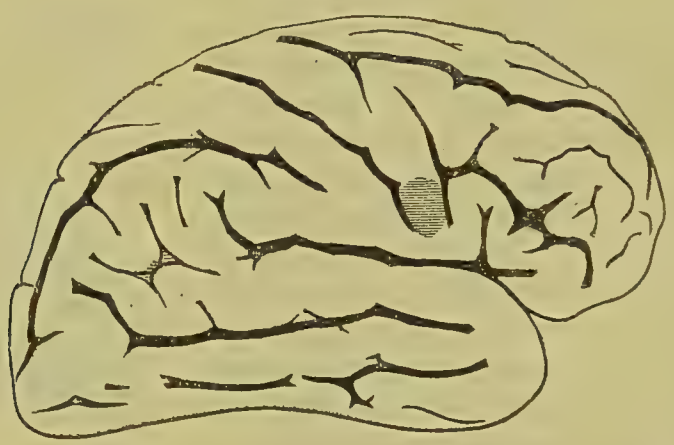
(e) Facial Monoplegia.—Facial paralysis of cerebral origin is generally complicated by aphasia or paralysis of the arm, but a few uncomplicated cases of facial paralysis from diseases of the cortex have been observed.

Fig. 260.



Charcot and Pitres describe a case of apoplexy followed by left hemiplegia and rigidity of the limbs. The rigidity disappeared after a time and the paralysis became limited to the lower facial muscles. An extensive area of softening was found in the cortex of the right hemisphere, invading the third frontal, the lower extremities of the ascending frontal and parietal convolutions, and a large extent of the parietal and temporo-sphenoidal lobes of the Island of Reil (*Fig.* 260). Although the cortical lesion in this case was so extensive, it will be seen that disease of the inferior extremities of the ascending convolutions was the important lesion so far as the motor area is concerned. Hitzig relates the case of a soldier who received a bullet-wound on the right side of the head and became affected two months subsequently with clonic spasms in the left side of the face, followed by paralysis of those muscles and of the left half of the tongue. After death an abscess was found in the ascending frontal convolution between the præ-central fissure and the fissure of Rolando, corresponding to the seat of injury (*Fig.* 261).

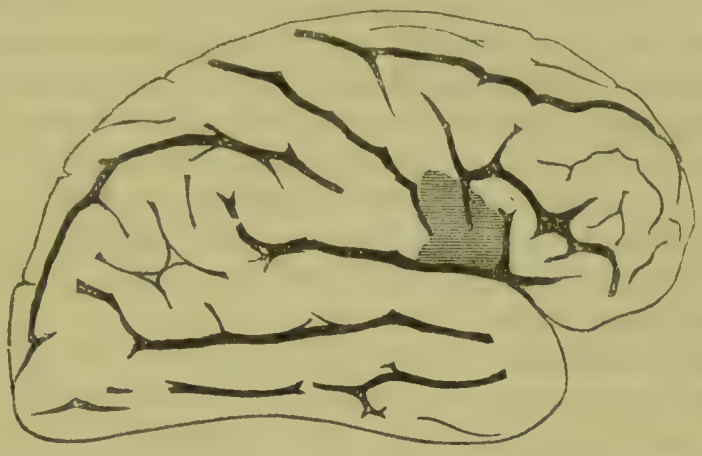
Fig. 261.



A case of left hemiplegia is reported by Dr. Gowers in which gradual recovery took place, with the exception of marked paralysis of the inferior facial muscles. At the autopsy a hæmorrhagic extravasation was found in and beneath the upper half of the præ-central sulcus which had passed into the substance of the adjoining convolutions, consisting of the posterior extremities of the middle and superior frontal and corresponding part of the ascending frontal of the right hemisphere. A large number of cases might be cited in which right facial paralysis existed, associated with aphasia, and in which the lesion was situated at the junction of the third frontal with the ascending frontal convolution of the left hemisphere. The case of a woman, aged 71 years, is reported by Ballet, who had a slight attack of apoplexy without loss of consciousness. The permanent symptoms consisted of paralysis of the left half of the face and of the tongue. There was also slight feebleness of the left upper extremities, but the lower was unaffected. There were no sensory disturbances. Towards the evening of

the same day the head and neck became deviated to the right and the paralysis of the left arm became more marked. Death took place from coma four days subsequently to the beginning of the attack, and at the autopsy a hæmorrhagic focus, of the size of a large nut, was found in the inferior part of the ascending frontal convolution (*Fig.* 262). The inferior frontal and inferior parietal fasciculi of the white tissue were partially destroyed, but the basal ganglia were normal.

FIG. 262.



(*f*) *Unilateral Oculo-motor Monoplegia.*—It has already been mentioned that conjugate deviation of the eyes and rotation of the head and neck are frequent symptoms both of convulsions and of hemiplegia, and that the deviation in the former is directed away from, and in the latter towards the hemisphere in which the lesion is situated. In the brain of the monkey, Ferrier localises a centre (*Fig.* 232, 12) in the posterior extremity of the second frontal extremity, irritation of which causes elevation of the eyelids, dilatation of the pupils, conjugate deviation of the eyes, and turning of the head to the opposite side; while, on the other hand, extensive movements of the eyeballs, along with associated movements of the head and neck, result from irritation of the supra-marginal and angular gyri (*Fig.* 232, 13, 13′).

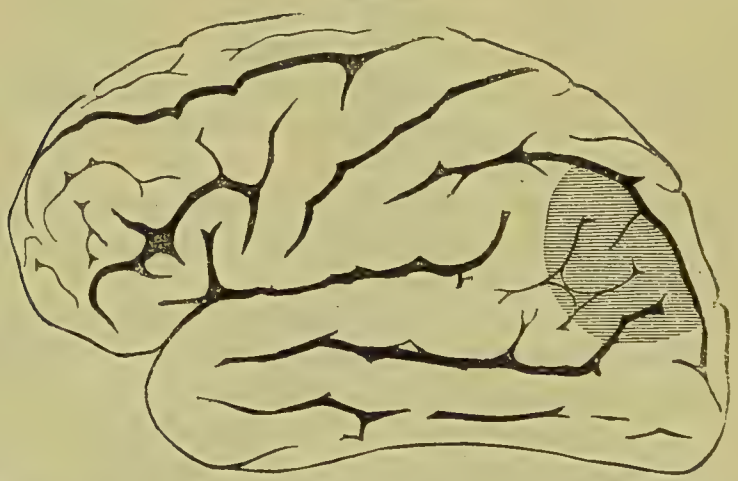
A case is reported by Chouppe which appears to show that the centre for the production of conjugate deviation of the eyes and rotation of the head and neck is situated in the posterior extremity of the second frontal convolution. The case as quoted by Landouzy was that of a young man, 19 years of age, who presented the ordinary symptoms of tubercular meningitis, the most striking being a rotation of the head and eyes to the right without any other paralysis. After death a superficial focus of disease, of the size of a franc piece, was found on the posterior extremity of the middle frontal convolution in the left hemisphere. Other lesions were found in the superior and lateral part of the sphenoidal lobe of the right hemisphere. Landouzy thinks that the deviation of the eyes was caused by an irritative lesion of the posterior extremity of the second frontal convolution, but it must be remembered that the lesion in the

superior part of the sphenoidal lobe was close to the angular gyrus, and it is probable that the deviation was due to a destroying lesion in this area.

The case of a child, aged five months, is mentioned by Ferrier, on the authority of Dr. Carroll, of New York, in which a fracture of the skull was produced by a fall. When Dr. Carroll saw the patient, the head was rotated to the right, its range of motion never extending to the left of the middle line; the eyes, when at rest, were turned to the right, but could be voluntarily moved almost to the middle line; pupils, perhaps, a little dilated, but responsive to light; upper lids elevated. There was a fracture in the right parietal region, and a linear fracture could be detected in the parietal bone, about midway between the squamous and sagittal sutures, and intersecting a vertical line drawn upwards from the auditory meatus. The position of the fracture was, as pointed out by Ferrier, such as might coincide with injury of the posterior extremity of the second frontal convolution, the lesion being doubtless of a paralytic nature. It must, however, be admitted that these two cases are not of themselves sufficient to prove the existence of a centre for the rotation of the eyes situated in the middle frontal convolution.

Strong evidence has indeed been recently brought forward by Grasset to show that when conjugate deviation of the eyes is caused by disease of the cortex, the lesion is situated in the supra-marginal and angular gyri. He reports a case of left hemiplegia with conjugate deviation directed to the right, in which the lesion consisted of disease of the *pli courbe* of the right hemisphere (*Fig.* 263). Liouville describes a case of right unilateral

Fig. 263.



convulsions in which the head was strongly turned towards the right. The lesion, which consisted of tubercular meningitis, was situated on both sides of the horizontal limb of the fissure of Sylvius on the left hemisphere.

Sergiu reports a case of left hemiplegia with contracture of the muscles of the right side (probably paralysis of the muscles of the left side) of the neck. The lesion consisted of a meningo-encephalitis in the right middle

lobe at the level of the superior part of the fissure of Sylvius. Charcot and Pitres mention a case reported by Samt, in which there was right hemiplegia, while the head and eyes were deviated to the left. A focus of softening was found situated upon the parietal lobe, not quite reaching the ascending frontal convolution in front, bounded posteriorly and inferiorly by the posterior extremity of the parallel fissure, and passing beyond the interparietal fissure superiorly, but not quite reaching to the great longitudinal fissure. These cases, although many more might be added, will suffice to show the importance, with regard to conjugate deviation of the eyes, of the convolutions which border the posterior extremities of the Sylvian and parallel fissures.

Many cases are recorded in which conjugate deviation of the eyes was caused by disease of the centrum ovale, and in these the lesion was, as a rule, situated between the internal capsule and the supra-marginal and angular gyri. Prévost reports a case of right hemiplegia with rotation of the head and eyes to the left. A hæmorrhagic focus was found in the posterior part of the parietal lobe of the left hemisphere. In another case reported by the same author right hemiplegia, with rotation of the head and eyes to the left, was caused by a sarcoma, of the size of a pigeon's egg, situated in the centrum ovale behind the fissure of Rolando, and along the longitudinal fissure.

It would appear that disease in the neighbourhood of the angular gyrus and supra-marginal lobule produces at times paralysis of the levator palpebræ superioris of the opposite side, without the other muscles supplied by the third nerve being implicated (Landouzy).

Lesions may occur in the cortex of the brain in the area of distribution of the middle cerebral artery without being attended by paralysis. Boyer maintains that there are two "neutral" zones in the area, the one occupying the superior parietal lobule, and the other the anterior part of the præcuneus and a part of the gyrus fornicatus. A case is reported by Dr. Ringrose Atkins, in which there was a superficial erosion of the cortex on the postero-parietal lobule of the left hemisphere without motor disturbance having been present during life. I would suggest that the neutral zones of Boyer are associated with centrifugal fibres connecting the cortex of the brain with the cerebellum. Other cases are recorded in which the cortical motor centre of the leg was found diseased at the autopsy, yet in which the leg on the opposite side either had never been paralysed or had recovered. It is probable that in such cases the movements of both lower extremities were regulated from one hemisphere, the one on the side opposite the lesion receiving its impulses through commissural fibres in the spinal cord.

The motor area of the cortex may be compressed by very large tumours without paralysis being produced. In the Pathological Museum of the Owens College there is a preparation, presented by Mr. Windsor in 1877, of a sarcomatous tumour, about the size of the closed fist, which grew from the dura mater over the vertex, and near to the falx cerebri. The

underlying hemisphere was compressed and flattened, the motor area of the cortex being involved, but the patient had no paralytic symptoms during life. Two cases of a more or less similar kind have been recently described by Pitres.

Sensory Disturbances.—It has been maintained by Tripier that lesions of the cortical motor area of the brain are sometimes attended by hemianæsthesia as well as paralysis of the opposite side of the body, tactile sensibility being specially affected. He adduces in favour of this opinion some experimental evidence, and reports of seven clinical cases in which more or less of hemiplegia was associated with hemianæsthesia, the lesion in all of them being found limited to the motor area of the cortex of the hemisphere opposite to the side affected. But hemianæsthesia so frequently results from functional disturbances of the brain that it would be somewhat hazardous to conclude from these cases alone that the lesion of the motor area of the cortex was the cause of the loss of sensibility. Several cases are collected by Nothnagel to show that diminution of the muscular sense is not unfrequently associated with motor paralysis from cortical disease. He thinks that the cortical centres of the muscular sense lie near to, although they are not identical with, the motor centres.

Vaso-motor and trophic disturbances, consisting of elevation of the temperature of the paralysed limbs and acute bed-sore, have been observed in cases of disease of the cortex of the brain, but they do not possess any value as localising symptoms.

(iii.) Affections of Speech from Cortical Disease.

§ 747. The disorders of speech which are liable to occur in cortical disease constitute one of the most complicated problems of neurology; and before proceeding further, it is desirable to limit our subject so as to separate disorders of speech due to disease of the cortex of the brain from other affections of the nervous system that may resemble them. Language, taken in its widest sense, consists of the various means by which animals indicate mental states to one another. Mental states may be, as we have seen, divided into feelings, cognitions, and volitions. In one sense language may be said very often, if not always, to indicate volitions; but inasmuch as volitions are practically always determined by what are called motives, or in other words by the feelings and cognitions, the language of volitions merges itself into that of the other two mental states. Language may therefore be divided into that of the feelings or *emotional language*, and that of the cognitions or *intellectual language* or *speech*.

But the division between the language of the emotions and speech is by no means clear and trenchant. When a man delivers an oration, for instance, only a small part of what he utters is speech. All the variations of tone, the melodious voice, the graces of attitude and gesture, the charm of elegant and rhythmical language, and the thousand other ways by which a great orator knows how to sway and influence his audience, belong to emotional and not to intellectual language. Similar remarks apply to written language. The pleasure we derive from looking at a clearly-printed volume, and especially from looking at an illuminated text, the pleasure derived from looking at a well-executed picture rather than at a diagram, the methods, as accent, italics, and notes of exclamation, by which inflection and emphasis and wonder are indicated; the rhythm of metrical language, and the diction and imagery of poetry belong to emotional language. The languages of emotional and of intellectual gesture are also by no means readily separated. The gestures of those who retain the full use of spoken and written language are in great part indicative of the feelings, but that gesture can be made subservient to intellectual expression is shown by the importance it assumes in the intellectual training of the deaf and dumb.

Language is the instrument of the social state, and that it may be the means of intercommunication between animals it possesses to each a *subjective* and an *objective* value, or fulfils an *impressive* and *expressive* function. Each individual of a social community, in order to become an effective member, must be able to feel or comprehend the mental states of the others from watching their gestures and listening to their various vocalisations, and must also be able by his gestures and vocalisations to render his own mental states intelligible to the others.

The *subjective* or *impressive* function of language, or rather of speech, with which we are here more immediately concerned, may be subdivided into *receptive* and *regulative* functions.

The *receptive* department is represented structurally by the various peripheral sense-organs and the centripetal fibres, or cells and fibres, which conduct impressions made upon the former to the cortex. Complete loss of speech from disease of the receptive apparatus is unknown.

The vocal speech of a person born blind is almost entirely unaffected either in its subjective or objective aspects, while the patient may, by the device of raised letters, be taught to understand written language. The deaf mute is taught both to understand and to give expression to a complicated speech by gesture; and in recent times such patients have been taught to use their vocal organs for expression in speech, while they are made to understand the vocal speech of others by closely observing the movements of the muscles of articulation.

The remarkable case of Laura Bridgeman, who became blind and deaf in her second year, while her sense of smell and taste were also very deficient, shows how much careful training may do in developing language and thought through the sense of touch. This girl was taught by Dr. Howe, of Boston, who affixed on a number of common objects labels on which the name of the article was written in raised characters. After she had learnt to associate each label with its object, a number of separate labels were put in her hand, and she was then encouraged to place each label on its corresponding object. After a time the separate letters were placed in her hand, and she was then taught to put them together so as to form the names of common objects. "Up to this," says Dr. Howe, "the proceeding was only a mechanical one, and the result was about as great as if one had taught a number of tricks to a clever dog. The poor child had sat there in mute astonishment, and patiently imitated everything that was performed before her. But now the matter seemed to dawn upon her in its true light, her understanding began to exercise itself, she noticed that she now possessed the means of arranging for herself symbols of something that lay before her mind, and of showing this to another mind; immediately her countenance beamed with human reason; she could no longer be compared to a parrot or dog; the immortal intellect now seized greedily upon this new bond of union with other intellects! I could almost point out the moment at which this truth dawned upon her and poured light over her whole face."

The structural counterpart of the *regulative* function consists of that part of the cortex of the brain in which the centripetal impulses are reduced to such order as is necessary to render them the correlatives of the cognitions. Now, the cognitions, as we have seen, express the relations between our feelings, and all cognitions must be expressed by propositions. The mode of expression may not always assume a distinct propositional form, but it must at least possess a propositional value if it convey distinct knowledge. If I repeat the word "orange" in the hearing of another, it may, or may not, convey to him distinct

information; but if any information be imparted, the word must convey to the listener the idea that the object named "orange" belongs to a class of objects already known to him under that name, and the word in this sense possesses the value of a distinct proposition. If the listener has never had any experience of the object named "orange," it is clear that the utterance of the name will convey no meaning; but if he has had experience of other fruits and of colours, distinct information may be conveyed to him with regard to the object by saying "an orange is a yellow fruit." The listener will be able to associate the general properties of fruit and a distinct colour with the word in future, but the information has been imparted by means of a formal proposition. The activity of the regulative cortical centres of speech have for their functional correlative the arrangement of the presentative and representative cognitions into the form of distinct mental propositions.

The objective or expressive function of speech may be subdivided into *emissive* and *executive* departments.

The *emissive* department is represented structurally by that organisation in the cortex of the brain in which the regulative impulses are finally co-ordinated before being conducted to the executive department.

The *executive* department is represented structurally by groups of nerve cells in the central grey tube, and by the nerves and muscles concerned in vocalisation, articulation, the manual operations of writing, and various gestures. Complete loss of speech from disease in the executive structure is most unusual. The patient, for instance, may lose his voice in different diseases of the larynx, but he can still articulate; he may lose both voice and articulation in bulbar paralysis, but is generally able to make known his wants in writing, and when unable to write from want of previous education he can make his ordinary wants known by gesture.

Our further remarks must be limited to the derangements of speech caused by disease of the cortex of the brain. These consist of disorders of the regulative department of the impressive function, and of the emissive department of the expressive function; and as the latter is probably the simpler of the two, we shall deal with it first.

§ 748. (*a*) *Loss or Impairment of the Emissive department of the Expressive faculty of Speech while the Impressive faculty is unaffected. (Ataxic Aphasia—Agraphia—Amimia.)*

In cases of this kind the patient is unable to communicate his thoughts by words or by writing, while his intellectual pantomime is impaired. He can often utter words, but these may not possess any intellectual value; in the words of Dr. Hughlings-Jackson the patient is *speechless* but not *wordless*. The words which the patient can utter, as a rule, continue the same in the same patient—"recurring utterances." Or the patient may under excitement swear, or even utter a phrase appropriate to the surrounding circumstance, such as "Good-bye," when a friend is leaving. It will be readily seen that the "recurring utterances" such as "Yes" or "No," which are repeated on all occasions whether appropriate or not, do not possess any intellectual value, while of the occasional utterances swearing is a purely emotional expression, and even the phrase "Good-bye" must be regarded as expressing a state of mental regret rather than a purely intellectual appreciation of the surrounding conditions. In some cases, in addition to the usual recurring utterances of "Yes" and "No," the patient repeats such phrases as "Come on to me" (Jackson), or "I want protection" (Paget). The man whose recurring utterance was "Come on to me" was a railway signalman, and had been taken ill on the rails in front of his box, while the man who could only say "I want protection" had his left cerebral hemisphere injured in a brawl. Dr. Hughlings-Jackson makes the very probable supposition that in these cases the recurring utterance constituted the last words spoken or which were in a state of mental preparation for utterance when the damage occurred to the brain. It is not improbable that words uttered or about to be uttered during a period of great excitement might leave permanent traces which would render them liable to be subsequently uttered as interjectional phrases during emotional states. That all these words and phrases must be regarded as expressive of emotional rather than intellectual states is shown by the fact that the patient is frequently unable to repeat his favourite oath or his formula

of leave-taking, or perhaps "Yes" or "No" when asked to do so (Broadbent).

The patient, on the other hand, understands all that is said to him, and remembers what is read to him or what he reads himself. His articulatory actions are well performed, and during eating and swallowing his vocal organs act normally, and he may sing, laugh, smile, and frown as usual. He will point to objects named and recognise drawings of them, provided they were known to him before his illness. He is able to play at cards and other games, and recognises handwriting. The few words which the patient can use, as yes or no, may be uttered with such variations of tone and gesture as to indicate when he is angry or joyful. His use of words is, in accordance with Mr. Herbert Spencer's theory, more akin to song than to speech, and belongs rather to emotional than to intellectual language.

So far we have considered the cases of those patients who are completely deprived of the power of expressing intellectual language while retaining the power of understanding it, but we must now turn our attention to those lesser grades of ataxic aphasia in which the patient still retains the use of a few words or phrases of real speech value. We have seen that most aphasics use words in an interjectional sense, and when excited oaths or phrases as "God bless me" may be uttered, but these also must be regarded as compound interjections and as purely indicative of emotional conditions. Besides the interjectional use of words and phrases, the patient may occasionally utter a word or phrase which is evidently equivalent to a distinct proposition. He may, for instance, retain the full use of the words "yes" and "no," and even when he uses "no" to express assent as well as dissent, he may be able by the aid of pantomime to indicate in which sense he intends the word to be understood. Dr. Hughlings-Jackson mentions the case of a woman who could only utter the phrase "Yes, but you know," who was once heard to say "Take care!" when a child was in danger of falling, but could not repeat the phrase when asked to do. It cannot be denied that this utterance possesses an intellectual element, inasmuch as it is an appropriate admonition to a person in danger of falling. It must,

however, be remembered that the phrase, although appropriate to the occasion, was uttered under circumstances calculated to induce alarm and excitement, and the same words had probably been frequently repeated under similar circumstances. In the slighter defects of speech the patient can talk, but uses a word kindred in its meaning with the one intended, as "worm-powder" for "cough-medicine," or in its sound, as "parasol" for "castor-oil" (Jackson).

(*b*) *Loss or Impairment of the Regulative department of the Impressive faculty of Speech, while the Expressive is either unaffected or only secondarily implicated. (Amnesic Aphasia.)*

(1) *Loss of Memory of Names or Nouns.*—In many cases of loss of memory for words the names of things are forgotten, while the memory for dates, events, and the relations between these may remain good. Dr. Broadbent mentions the following case:—An old gentleman, after very slight right hemiplegia, could give long answers fluently, and volunteer statements, so long as the phrase did not contain a noun.

"Oh, yes; I am much better than when you last saw me." "I shall be 73 on the three—four——," when he confused himself in trying to find the word December. He could not name a hand when told to do so, but in his effort something like a leg was once heard. This gentleman's memory of facts, events, dates, and faces is very good.

The patient is often enabled to supply the want of a noun by a paraphrase, as in the following case, quoted by Kussmaul from Bergmann:—

"A hind, 40 years of age, was unconscious for four weeks after a severe injury of the head; he regained his recollection of things and places, but his memory for names was lost. The nouns had disappeared from his vocabulary, but he still had command of the verbs. A pair of scissors he called that with which one cuts; the window, that through which one sees, through which the room is illuminated, &c. He had forgotten most of his songs and prayers. He recovered subsequently."

Sometimes the initial consonants of words are left out of words in speaking and writing (Schlesinger), while in a case recorded by Graves the sight of persons and objects merely suggested the initial consonants of their names, the rest of the

name not being recalled until the corresponding written word met the eye.

"A man, 56 years of age, after an apoplectic attack, lost his memory for proper names and substantives in general, with the exception of their first letters, although the power of speech was not impaired in other respects. He prepared for himself an alphabetically arranged dictionary of the substantives required in his home intercourse, and whenever it became necessary for him to use a noun he immediately looked it out in his dictionary. When he wished to say 'Cow,' he looked under C. As long as he kept his eye upon the written name, he could pronounce it, but a moment afterwards he was unable to do so."

(2) *Inability to Express the Relations between Things.*—In another form of amnesic aphasia the names of persons and things are more or less remembered, but the memory of words indicative of relations and attributes is impaired.

In the case of a somewhat complicated disorder of speech mentioned by Dr. Broadbent the patient could only say: "Brother, brother—New York—America, two brothers in America—letter." This patient was, therefore, able to recall the names of the persons and places intended, but could not express the relations between them so as to construct a sentence.

(3) In another disorder of the receptive faculty of speech the patient is unable to name any object which he sees, or to read a single letter, although he may converse fluently and write correctly (word-blindness—word-deafness). A case related by Dr. Broadbent is a remarkable example of this affection.

An intelligent man, 59 years of age, after an acute cerebral attack, lost completely the power to read printed or written words. He was also unable to recall the name of the most familiar object presented to him. This man could, however, converse fluently, his vocabulary was large, and his words well chosen and arranged, although he occasionally forgot the names of streets, persons, and things. He could also write easily and correctly both from dictation and spontaneously. He died from an extensive hæmorrhage into the left temporal lobe, with rupture into the lateral ventricle. Two foci of softening of older date were observed, one being situated in the temporo-sphenoidal lobe beneath the posterior end of the parallel sulcus, and the other higher up underlying the angular gyrus, and between it and the point where the descending horn of the ventricle is given off.

(4) In a fourth form of amnesic aphasia the patient apparently fails to comprehend written or spoken language, and seems to be unconscious that his speech, which consists of mere jargon,

is unintelligible to others. The following brief abstracts of two cases described by Dr. Broadbent are good examples of this condition :—

A man, aged 60 years, who had previously been a good talker and great reader, suffered, after a fit of some kind, from a peculiar affection of speech, paresis of the right side of the face but no hemiplegia. His speech was a mere inarticulate jargon. When asked a question he would make a brief reply as if he understood and answered ; the modulation of the voice and the emphasis were perfectly natural, and corresponded with the facial expression and gestures, but, as a rule, there was not the least semblance to words in what he said. His replies were often so suitable in length and emphasis that it might have been supposed that he had comprehended the question. When, however, he was told to do anything, it was seen that he did not understand the simplest phrase. He sat up in bed once or twice when required to do so, but as this was not made a test question there would be other indications of what was wanted, and he was extremely ready in comprehending signs. When told to give his hand he invariably put out the tongue. A letter addressed to him at the hospital being handed to him, he took it, appeared to read the name and address, and put it down again. Not attempting to open it, a piece of paper having " Give me your hand " written upon it was handed to him. He took it, held it so as to get a good light on it, and then having apparently read it laid it aside without giving his hand, though asked to do so by word of mouth as well as in writing.

The patient died somewhat suddenly, and at the autopsy a large focus of softening was found in the left hemisphere, limited to its posterior half. Part of the supra-marginal lobule was yellow in colour, shrunken in volume, and soft. This condition extended upwards and backwards to within about half an inch of the longitudinal fissure just in front of the external parieto-occipital fissure, involving, therefore, the postero-parietal lobule. The morbid change implicated the angular gyrus, and nearly reached the occipital lobe ; the adjacent parts of the temporo-sphenoidal lobe, the posterior end of the infra-marginal, and parallel gyri were soft, but not wasted or discoloured on the surface.

The affection of speech in the following case reported by Dr. Broadbent is more complicated than that in the case just described.

The patient was a well-educated and intelligent young man, who had contracted syphilis eight years previous to the date of the report. About a fortnight before his admission into St. Mary's Hospital he was suddenly seized with hemiplegia and loss of speech. He appeared to understand all that was said to him, but could not answer questions at first, although after a few weeks he improved so much as to be able to reply to questions requiring brief and simple answers. He was, however, unable to give a con-

nected account of anything requiring more than a few words. His method of correcting an erroneous statement which had been made that he woke up from sleep paralysed and speechless was as follows: "No—evening, evening—put down my cigar, smoking, smoking not a quarter of an hour—all at once"—indicating by gestures the loss of power in the limbs and adding—"Couldn't speak." He lost at first all knowledge of numbers, and could not tell how many 2 and 2 made; but by practice he could in a few weeks multiply by 2 and 3 up to 12.

When a table, glass, inkstand, and violets were pointed out to him, he was unable to name them, nor could he name his gloves, hat, or a pen. He named, however, some objects, such as his hand and the fire. He perused his newspaper regularly, and with all the marks of intelligent interest. He understood it also, for he went to the sister in a state of great excitement to tell her of the failure of a firm with which he had business relations, carrying the paper in his hand, and pointing out the announcement; and he could always find a given paragraph, when asked to do so as a test. When, however, he was asked to read aloud, the result was gibberish. The following passage was selected:—"You may receive a report from other sources of a supposed attack on a British Consul-General. The affair, however, is utterly unworthy of consideration. No outrage was even intended, and the report was due to misrepresentation of the facts. The Odessa line is again working properly." It was read slowly, and in a jerky manner, as nearly as it could be taken down thus:—"So sur wisjee coz wenement ap ripsy fro fruz fenement wiz ā seconce coz foz no Sophias ā thee freckled pothy conollied. This affaise eh oh cont oh curly of consequences. Uce sudos val oh es es entain ah thee enepol ā oh dee ā ah messequence oh coz foz. The assoil lens ā puff pifl miss corres povety." It was evidently an effort to read aloud, requiring close attention, and he read seriously and steadily, apparently unconscious of the absurdity of his utterances, till interrupted by laughter, which it was impossible to restrain, in which he usually joined. He was never able to give the simplest written answer to a question, or to write from dictation, but he signed his name quite well, and wrote down the names of his brothers, but with the initial only of the Christian name, the surname in full.

When asked to copy a sentence, he wrote the short words quickly, and in a good hand; but a long word he took down slowly, letter by letter, in large schoolboy characters, usually accurately, but, as he wrote each letter, he named it aloud, and *always wrongly*.

§ 749. *Morbid Anatomy.*—So far as is known of the morbid anatomy of cortical disorders of speech may be summed up in a few words. A comparison of a large number of observations shows that the lesion in ataxic aphasia is situated in the posterior portion of the third frontal convolution and the adjoining portion of the Island of Reil of the left hemisphere. In

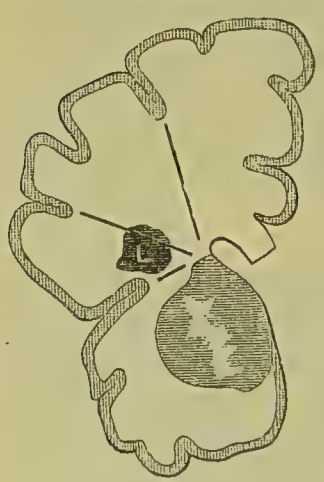
exceptional cases the disease of the third frontal convolution has been found in the right instead of the left hemisphere, and in these the aphasia was associated with left hemiplegia during life; the patients were in most cases known to be left-handed. In other cases of right hemiplegia, but without aphasia, the posterior extremity of the third left frontal convolution has been found disorganised after death, and in such cases also the patients were left-handed.

The portion of the ascending frontal convolution which adjoins the Island of Reil is often involved in the disease. The lesion generally consists of occlusion—either by embolus or syphilitic thrombosis—of the left middle cerebral artery, or at least of the branch which supplies Broca's convolution.

In amnesic aphasia, on the other hand, the lesion is in the area of distribution of the posterior and terminal branches of the left middle cerebral artery, and the region of softening comprises the supra-marginal and postero-parietal lobules, the angular gyrus (visual centre), the posterior part of the infra-marginal convolution (acoustic centre) and the convolutions bounding the parallel and collateral fissures (Broadbent).

Lesions that damage the fibres of the corpus callosum which connect the third frontal convolutions of the two sides, and those which connect the third left frontal convolutions with the internal capsule, produce, as has been pointed out by Broadbent, as permanent an affection of speech as destruction of Broca's convolution itself.

Fig. 264.



A man, aged 66 years, whose case is reported by Pitres, suffered from right hemiplegia, with embarrassment, and finally complete loss of speech. At the autopsy, two small patches of yellow softening were found in the cortex of the left hemisphere, one being situated upon the superior parietal lobule, and the other upon the lobule of the *pli courbe*. No changes were observed in the third frontal convolution, but a large focus of softening was observed in the centrum ovale, which extended anteriorly to the part underlying the posterior extremity of the third frontal convolution, and posteriorly beyond the posterior extremity of the optic thalamus (*Fig.* 264, L).

§ 750. *Morbid Physiology.*—When the structure of the cortex at the posterior extremity of the third left frontal convolution is thoroughly disorganised, the expressive faculty of speech is arrested at its origin. The patient can understand everything that is said to him, he can think and probably clothe his ideas in suitable subjective language, but the objective or expressive part of speech is entirely lost. He can understand the thoughts of others, but cannot communicate his thoughts to others either by spoken or written language or by gesture. Most aphasics present apparent exceptions to this rule, inasmuch as the majority of them are not quite destitute of the power of uttering words. But, as pointed out by Dr. Hughlings-Jackson, a patient may be completely speechless though not entirely wordless. The words that aphasic patients use are recurring utterances like "Yes" and "No," which are repeated on all occasions, whether appropriate or not. The patients have only an interjectional and not a cognitional use of these words, and they must be regarded as part, not of intellectual, but of emotional language. The patient may be able to swear, oaths being part of emotional language. Dr. Hughlings-Jackson thinks that as actions become more and more automatic they tend to become organised in the right as well as in the left hemisphere, and he believes that the recurring utterances and phrases used by aphasics are those which had become automatic either previous to or during the attack, and consequently organised in the right hemisphere. The words which become automatic are those like "Yes" and "No," which have been frequently repeated in the experience of the individual, and words of the character of oaths, which, although they may not necessarily have been frequently used by the patient, have been used under circumstances of excitement and are expressive of emotional states. In those cases in which the patient can repeat words in the form of a proposition, such as the man mentioned by Paget, who was injured in a brawl, and who could only say "I want protection," it is thought probable by Dr. Hughlings-Jackson that the patient was about to repeat the words at the moment of injury. He thinks, therefore, that these words had become automatic in him by being repeated under circumstances of great excitement. But whatever may be

the explanation of the recurrence of such phrases, it is obvious that they possess no value as a form of intellectual expression, inasmuch as they are repeated without any reference to their appropriateness to surrounding circumstances.

But what explanation can be given of the fact that the complex muscular movements which serve for intellectual expression are organised in one hemisphere only? Accepting the crossed connection of the hemispheres of the brain with the muscles of the trunk and limbs as a fact, there can be no difficulty in understanding why in right-handed people the more special muscular adjustments of the hand should be organised in the left hemisphere. It seems strange, however, that the muscular movements concerned in articulation should follow the same rule. It must at least be admitted that it would be an economy of force if the muscles of the two sides concerned in articulation were regulated from one hemisphere, and it is also probable that a greater precision in the execution of these movements is obtained by a unilateral organisation. It is likewise somewhat difficult to understand why in left-handed people both the structural correlatives of the more special movements of the hand and of the articulatory movements of spoken language are found together in the right hemisphere. But whatever may be the explanation, there is abundant clinical evidence that such is the case.

If then ataxic aphasia be caused by a destroying lesion of the emissive organisation of speech, it might be supposed that simple severance of the cortical organisation from the executive organisation would produce the same effect. In other words, it may be supposed that disease of the fibres of the pyramidal tract, which connect the posterior extremity of the third frontal convolution and the nerve nuclei in the medulla, would produce the same effect as disease of the cortex itself. When a commander-in-chief, for instance, sends orders to a general of division to execute a particular movement, the latter cannot obey the order unless the line of communication between the two be kept open, no matter how effective may be the organisation of the emissive department of intelligence at its central end. But it so happens in war that when the direct line of communication is cut off, an indirect one may be dis-

covered. And something of this nature occurs in aphasia caused by disease of the pyramidal tract. We have already seen that the fibres of the knee of the internal capsule connect the third frontal convolution and the nuclei of articulation in the medulla, and when these are interrupted on the left side, the patient suffers from temporary loss of speech. But, as has been pointed out by Dr. Broadbent, the patient under these circumstances makes a good and moderately rapid recovery. The explanation given by Dr. Broadbent of this rapid recovery is that, although the direct line of communication between the emissive organisation and the executive is cut off, an indirect line is readily established.

The usual course is for the message to be conducted downwards by the fibres of the left pyramidal tract and to cross over in the medulla to the nuclei of articulation of the opposite side, and then through commissural fibres to the nuclei of the same side. But when this channel is interrupted the message is sent from the left third frontal convolution through the fibres of the corpus callosum to the corresponding convolution of the right side, and from the latter through the right pyramidal tract to the nuclei of articulation of the opposite side, and through commissural fibres to the nuclei of the same side (§ 89). In this way the organisation in the third left frontal convolution can, after a time, be utilised, but during the time occupied in opening the new channels of communication the patient suffers from greater or lesser degrees of disturbances of speech. But, as has been pointed out by Broadbent, when the fibres of the pyramidal tract in connection with the hemisphere and the fibres of the corpus callosum which connect the third frontal convolutions of the two sides are both interrupted by a lesion in the centrum ovale, the affection of speech is as permanent as if the third frontal convolution itself were completely disorganised.

Speech, in its objective or expressive aspect, consists of highly special and complex movements, and the question arises, why are cases of aphasia not associated with paralysis of the muscles of articulation? The reply is that although there is not a paralysis of the separate actions of the muscles of articulation, yet there is a paralysis of the combinations of action

which are necessary for the production of speech. That ataxic aphasia is of a paralytic nature may be shown in several ways. The third left frontal convolution, for instance, is situated near the centres for the regulation of the movements of the inferior facial muscles and of the muscles of one-half of the tongue, so that the aphasia caused by disease of Broca's convolutions is associated with unilateral facial and lingual paralysis even in the slighter cases in which complete hemiplegia is not produced. But still more cogent evidence in favour of this view may be derived from cases of bilateral diseases of the hemispheres, affecting either the third frontal convolutions or the tracts of fibres which connect these with the nuclei in the medulla, and in which there is not only paralysis of the special but also of the general movements of articulation (anarthria). For the following example of this affection I am indebted to Dr. Leech, who kindly transferred the case to me ten days before the patient died. The notes of the case were taken by Mr. Gordon, when the patient was under the care of Dr. Leech, and by Mr. Luckman after he came under my care.

Joseph C——, aged 49 years, was admitted under the care of Dr. Leech, November 15th, 1880. The patient was healthy until about eleven months ago, when he began to complain of headache, usually situated in the temples and occasionally in the back of the head. It was also observed about this time that his speech was "thick," but no other notable symptoms were observed. Four and a half months ago he fell out of bed three or four times the same night, and was unable to get in again until assisted by his wife and son. From that time up to the present his speech appeared to have become more and more unintelligible, while he complained of general weakness, but there was no distinct paralysis of any of the extremities.

Present Condition.—The patient is emaciated and feeble, so that he soon tires on attempting to walk, but there is no paralysis of the extremities. There is considerable loss of facial expression, and the patient cannot compress his lips or whistle, but can blow out a candle with tolerable facility. He can protrude his tongue, but cannnot curl the tip up towards his nose, or roll it up laterally so as to render it tubular. His speech is almost unintelligible, and great attention is necessary in order to understand the few words he is able to utter. He can pronounce the separate consonants with tolerable distinctness, but finds difficulty with the labials and dentals, the letters *c*, *d*, *f*, *l*, *m*, *n*, *s*, *t*, *x*, *h*, and *w* giving him the greatest difficulty. Food collects between his teeth, viscid saliva collects in his mouth, which has to be constantly wiped away, and

the power of deglutition is impaired. His arteries are atheromatous, but no other important general symptoms are present.

Nov. 26. He has complained of pain for the last few days over the temporal region. The nurse reports that he has been slightly delirious at times for the last few days, that he got out of bed several times yesterday, and that on one occasion he fell down and had to be assisted into bed. During this attack he is reported to have been conscious, but his speech was greatly affected, and there was some degree of paralysis of both the lower extremities. There is at present no distinct loss of power in either the legs or the arms, but his speech is more unintelligible than at any time since his admission.

Dec. 1. The patient is now suffering from diarrhœa, and there are marked fluctuations of the temperature curve. The uvula is somewhat pendulous, although not distorted, and its reflex excitability is diminished. The fauces and epiglottis can be examined with the point of the finger without provoking a cough, while the patient exhibits a remarkable tolerance to laryngoscopic examination. The vocal cords move normally during respiration and phonation.

Dec. 10. Since last report the diarrhœa has proved intractable to treatment, and the patient is much feebler, the temperature curve presents marked variations in the course of twenty-four hours, being at noon yesterday below 97° F., and at midnight 105° F. During the previous thirteen days the temperature varied from between 96·5° F. and 97·5° F. in the morning to between 101·5° F. and 103·5° F. in the evening. The speech has been for some days quite unintelligible. The patient now became gradually comatose, and died in the evening.

The post-mortem examination was conducted by Dr. A. H. Young twenty-four hours after death. The arachnoid over the anterior part of the convexity of the brain was opaque, and the subarachnoid tissue œdematous, but the membranes were healthy posteriorly and over the base. Each cerebral hemisphere presented a single well-defined cystic cavity, containing clear straw-coloured fluid, and occupying the positions of the lenticular nuclei. In the left hemisphere the lenticular nucleus was simply replaced by the cyst, but in the right the cavity was considerably larger than the area of the nucleus, extending anteriorly slightly beyond the anterior extremity of the caudate nucleus, and posteriorly to the wall of the descending horn of the lateral ventricle, although it did not communicate with the latter. The claustrum and internal capsule on each side were unaffected. The ventricles contained a slight excess of fluid. The arteries at the base of the brain were atheromatous. The mucous membrane of the rectum and descending colon was covered by deep ulcers, with thickened margins.

Microscopic examination showed that the nerve nuclei in the medulla were healthy, and no descending changes could be detected in the pyramidal tracts in any part of their course.

Dr. Barlow was probably the first to draw attention to the fact that lesions, symmetrically situated in the hemispheres, may produce symptoms closely simulating those of bulbar paralysis. A boy, aged 10 years, suffering from aortic disease, had an attack of right hemiplegia with aphasia, from which he made a good recovery. Four months subsequently he had an attack of left hemiplegia with aphasia, as well as paralysis of the muscles of articulation, those concerned in the first act of deglutition, and of the muscles of mastication. At the autopsy evidence of an embolus was found in both Sylvian arteries. The obliteration of the vessel on each side was associated with a focus of softening, about the size of a shilling, and situated in the inferior part of the ascending frontal convolution, and the posterior extremities of the second and third frontal convolutions. Soon afterwards an important paper was contributed by Lépine on this subject. In the case observed by this author the symptoms were more or less similar to those just described in the case of Joseph C——, but in the former the difficulty of deglutition was more marked than in the latter. At the autopsy a diseased focus was found in each hemisphere, involving the external capsule and the second and third divisions of the lenticular nucleus, while that in the right hemisphere extended to the convolutions of the Island of Reil and the posterior extremity of the third frontal convolution. Another case of the same kind is quoted by Lépine from Oulmont.

Lépine also refers to a case reported many years ago by Magnus, in which anarthria was caused by a unilateral lesion of the brain, the disease being situated in the corpus striatum of the right hemisphere. Another case is reported by Kirchoff in which bulbar symptoms were caused by a diseased focus situated in the right hemisphere, the left being healthy. The cases in which the lesion was unilateral appear to show that the general or more automatic movements of bilaterally associated muscles are often regulated almost entirely from the right hemisphere of the brain. Cases of this kind suggest the question as to whether the bulbar symptoms were caused directly by the lesion of the lenticular nucleus or indirectly by implication of the fibres of the knee of the internal capsule. In the case observed by me

the most careful microscopical examination of the crusta, pons, anterior pyramids of the medulla, and spinal cord failed to detect any descending changes in the pyramidal tracts. The cysts in the hemispheres were, however, very much distended with fluid, and it is quite probable that a certain amount of pressure was thus exercised upon the fibres of the internal capsule, sufficient to partially interrupt conduction through them, without being sufficient to cause secondary degeneration. This question must, therefore, be left for future observations to determine.

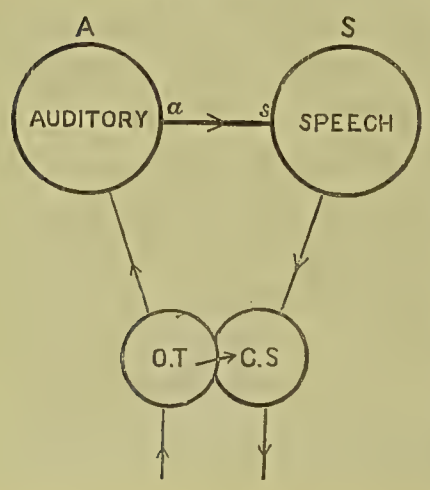
The mechanism by which the different forms of amnesic aphasia is produced is much more difficult to comprehend than that of the ataxic variety. In order to facilitate the comprehension of the various forms of aphasia, several authors have constructed diagrams to represent hypothetically the nervous mechanism concerned in speech. The best of these are the diagrams of Kussmaul and of Broadbent, and although not entirely agreeing with either of them, we shall avail ourselves of the diagrams of the latter. Dr. Broadbent sets out in his explanation with the proposition "that all muscular movements are performed under the direction of a 'guiding sensation.'" It would have been better if he had said under the guidance of "centripetal impulses" instead of "sensation," inasmuch as muscular movements take place in the entire absence of sensation. If, for example, the palm of the hand of a person asleep be tickled, the hand closes under the guidance of centripetal impulses, but independently of sensation, the action in this case being reflex. But when the individual is awake the outgoing portion of the reflex arc can be utilised by the cortex of the brain, and then voluntary closure of the hand takes place. The nuclei of the motor fibres of the peripheral nerves in the spinal cord are, therefore, subservient both to centripetal impulses coming from the periphery, and to centrifugal impulses coming from the cortex of the brain. But the centrifugal impulses from the cortex are initiated and controlled by centripetal impulses coming towards the cortex from the periphery. It thus appears that each movement is represented in the anterior grey horns of the cord by a group of connected cells, and that this group may be called into activity by centripetal impulses

coming from the periphery to the same level of the cord, or by centrifugal impulses from a higher nerve centre, or, in Dr. Broadbent's words, "a motor cell-group is formed under the guidance of a sensory cell-group on the same level, and, when formed, is made use of by a higher centre."

The "motor cell-group" in the case of speech, which for the sake of convenience Dr. Broadbent calls a *word-group*, must combine into orderly action the thoracic muscles in order to obtain an expiratory current of air, the laryngeal muscles for phonation, and the muscles of the lips and tongue for articulation. I shall follow Dr. Broadbent in placing the word-groups in the corpus striatum, although in my opinion it would have been better had he discarded this ganglion from the explanation and merely spoken of the cortex and medulla oblongata, which are connected with one another by straight fibres.

When the cells of the word-group are called into action by centripetal impulses on the same level, the action is reflex, and the resulting contraction would simply represent a complicated muscular adjustment without any reference to intellectual expression, and it is only when its activity is evoked from the cortex that the movement becomes subservient to speech. The cortical outlet for speech is situated in the third left frontal convolution, while the cortical guiding sensory centre for spoken language is situated in the superior temporo-sphenodial convolution (auditory centre). In accordance with the annexed diagram, lesion of S, the speech centre, will cause ataxic aphasia, and lesion of A, the auditory perceptive centre, or of *a s*, the fibres which connect the inlets and outlets, will cause different forms of "mistakes in words." A hypothetical explanation is thus afforded for three disorders of speech. In lesion of S the "way out" for all the muscular adjustments concerned in intellectual expression is destroyed; in lesion of *a s*, the line of communication between the guiding sensory centre and

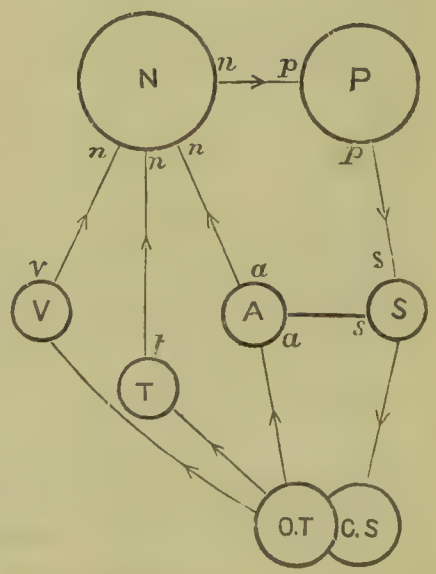
FIG. 265.



the motor outlet is damaged, and mistakes in words recognisable by the patient occur; while in lesion of the sensory centre A, mistakes in words occur, of which the speaker remains unconscious.

But in intellectual expression still higher centres are engaged, and diseases of these produce various complicated disorders of speech. "The formation of an idea of any external object," says Dr. Broadbent, "is the combination of the evidence respecting it received through all the senses; for the employment of this idea in intellectual operations it must be associated with and symbolised by a name. The structural arrangement corresponding to this process I have supposed to consist in the convergence from all the 'perceptive centres' of tracts of fibres to a convolutional area (not identified), which may be called the 'idea centre' or 'naming centre.' This will be on the sensory, afferent, or upward side of the nervous system; its correlative motor centre will be the propositionising centre, in which names or nouns are set in a framework of other words for outward expression, and in which a proposition is realised in consciousness or mentally rehearsed. If we are to have a seat of the faculty of language, it would be here rather than in the third left frontal convolution, with which, however, it may possibly be in close proximity. Expressing this by a diagram, we have V, A, and T, the visual (angular gyrus, Ferrier), auditory (infra-marginal Sylvian gyrus), and tactual (uncinate gyrus), perceptive centres sending converging tracts of fibres, *v n*, *a n*, *t n*, to N, the 'naming centre.' Here the perceptions from V and T (smell and taste are omitted for the sake of simplicity) are combined into an idea, which idea is symbolised by the name reaching N through A. which has always, in the experience of the individual, been associated with the object. P is the propositionising centre in

FIG. 266.



which the phrase is formed, its relations with N and S being sufficiently clear."

According to this scheme lesion of the naming centre N would cause loss of the memory of names or nouns, leaving the patient able to express himself imperfectly in words indicative of relations and attributes.

Lesion of P, the propositionising centre, would render the patient unable to construct a sentence although retaining the use of names. This condition is illustrated by the patient who could say "brother, brother—New York—America—two brothers—America—brother."

Lesion of $v\,n$, the channel of communication between the visual perceptive centre V and the naming centre N, would explain cases of word-blindness; while cases in which the lesion is situated in the auditory perceptive centre A, or its line of communication ($a\,n$) with the naming centre, would explain cases of word-deafness.

When the lesion involves more than one of the sensory centres or their lines of communication with the naming centre, it is manifest that complicated disorders of speech will arise, difficult to analyse into their separate factors. What has here been said with regard to spoken speech may be extended to written speech and intellectual pantomime, inasmuch as all forms of intellectual expression are usually involved in the disorder.

I have so far endeavoured to give a succinct account of Dr. Broadbent's theory of aphasia, while making use as much as possible of his own words. It would be comparatively easy to criticise this scheme, but not so easy to construct a better. I do not, for instance, like Dr. Broadbent's use of the phrase, "perceptive centre." If I look at a patch of yellow colour before me and perceive that it is caused by what I know as an "orange," it is because along with a vivid sensation of colour I feel a faint revival of tactual, gustatory, and other sensations previously experienced in conjunction with a similar sensation of colour. If I stretch out my hand and find that the faint tactual sensation I feel along with the visual sensation cannot be converted into a vivid sensation, I call the patch of colour, not an orange, but an illusion, and I begin to think

that my senses have played me false. The physical correlative of a perception must, therefore, be excitation of a portion of the cortex of the brain in which all the sensory inlets are variously combined, and would, therefore, correspond in the diagram to Dr. Broadbent's naming centre, while his perceptive should be described as sensory centres. On the other hand, I see no good grounds for postulating the existence of a naming as distinct from a perceptive centre.

The process of naming demands a large increase in the size of the perceptive centre, but not the existence of a separate centre. Suppose, for instance, again, that I have an ocular perception of an orange, the presentative element in the cognition is a vivid feeling of a yellow colour, and the representative elements faint revivals of previously experienced feelings of touch and taste. I now close my eye and hear the word "orange" spoken, the sound of the word forms the presentative element of the cognition aroused, while the representative element as before consists of faint revivals of touch and taste, and of sight also now. The process of naming is, therefore, a method by means of which artificial symbols are linked on to groups of previously experienced feelings, and although the exercise of this function demands a great extension and complication on the perceptive centre of animals, yet it does not demand the formation of a separate centre for its exercise. Again, I hardly think that Dr. Broadbent has shown sufficient grounds for assuming the existence of a distinct propositionising centre, but I prefer not to enter upon a criticism of this portion of his scheme.

b. Lesions in the Area of Distribution of the Posterior Cerebral Artery.

The posterior cerebral artery supplies, as we have already seen, the temporo-sphenoidal and occipital lobes, with the exception of the superior temporo-sphenoidal convolution, which receives branches from the Sylvian artery. The experiments of Ferrier and others appear to show that the functions of the cortex of these lobes are purely sensory, and that it is directly connected with centripetal fibres, and only indirectly with centrifugal fibres through the cortex of the parietal lobes.

Disease of the cortex of the temporo-sphenoidal and occipital lobes, however, does not give rise to localised motor disturbance, and, contrary to what the results of experiments on animals would lead us to expect, distinct sensory disorders are also wanting. Lesions of these lobes are, as a rule, *latent.*

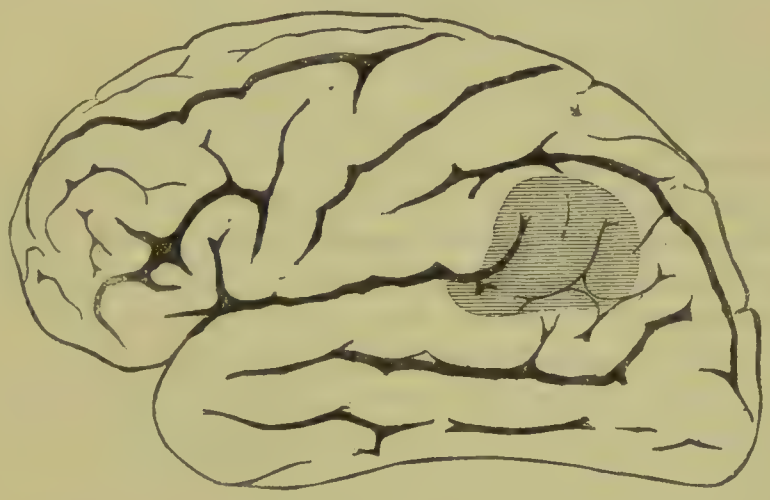
Lesions of the Occipital Lobes.—Ferrier quotes a case reported by Vauttier of yellow softening of the right occipital lobe and of the internal aspect of the left lobe (quadrilateral lobule). There was neither disorder of motion nor sensation, and, with the exception of considerable hebetude, there were no symptoms of a cerebral affection. Pitres reports a case in which an abscess, the size of a billiard-ball, formed in the posterior-inferior aspect of the brain. There were no sensory or motor disorders, mental obtuseness being the only indication of a cerebral lesion. In a case which came to the post-mortem table, when I was pathologist to the Manchester Royal Infirmary, a traumatic abscess, about the size of a hen's egg, occupied the right occipital lobe, destroying nearly the whole of its white substance. Dr. Dreschfeld, who saw the patient during life, assured me that, with the exception of temporary hyperæsthesia of the left side of the body, there was no disorder of the general or special senses. The patient suffered from delirium and general convulsions, but these symptoms were probably due to the presence of meningitis, which had spread over the occipital and parietal lobes of both hemispheres. Many similar cases are recorded (Gull, Rodocalat, Pitres). Marcé records a case of contusion of the right occipital lobe, followed by effusion into the membranes and softening of the cortex, without any sensory or motor disorders.

In a case reported by Sestié there was an abscess in each occipital lobe, but no sensory disturbances were present during life. Charcot has observed cutaneous formication and other paræsthesiæ in cases of softening of the occipital lobes, while Hughlings-Jackson and Bastian believe that disease of the posterior lobes is more frequently associated with mental derangement than disease of other parts of the brain. Hughlings-Jackson also thinks that discharging lesions of the right occipital lobe are more apt to give rise to coloured vision and other ocular spectra than disease of the left lobe. Ferrier quotes the following case from Abercrombie. A boy suffered from an injury of the head causing depression of a considerable portion of the right parietal bone, the depressed portion being forced through the dura mater, and driven inwards upon the brain. He had paralysis of the left side and amaurosis of the left eye. On the depressed portion being removed, the paralysis was greatly diminished, and the eye recovered a considerable degree of vision. On the third day after the operation, the wound in the dura mater was inflamed, with considerable tumefaction, and immediately the left leg and arm became paralysed, the paralysis being preceded by

convulsions, and the left eye again became amaurotic. He had frequent convulsions of the affected extremities for several days, the right side not being in the least affected, when, suppuration having taken place, all the symptoms subsided. It is very probable that the depression of the skull in this case had extended beyond the motor area of the cortex to the angular gyrus, and compression of the latter would probably suffice to explain the temporary amaurosis of the opposite eye.

It has been stated by Bastian that vision is apt to be impaired on the side of the motor paralysis in cases of thrombosis of the posterior cerebral artery. Fürstner has observed unilateral affections of sight in cases of general paralysis of the insane in which the occipital lobes were specially involved in the disease. A most important case in this connection has been reported by Glynn, in which the patient became suddenly and completely blind, and in which a clot was found occluding the posterior cerebral artery of the left side, causing extensive softening of the left occipital and temporo-sphenoidal lobes. In the case of word-blindness observed by Broadbent, and which we have already reported in full, the important lesion was found in the region of the angular gyrus and supra-marginal lobule (*Fig.* 267).

FIG. 267.



Several cases are mentioned by Nothnagel in which disease of the occipital lobe was associated with bilateral hemianopsia, but in most of these cases the disease of the hemisphere was associated with an affection of the optic thalamus. But the external geniculate body, in which the optic tract terminates, is so liable to be implicated in lesions of the optic thalamus that no case in which the thalamus is extensively involved along with the occipital lobe possesses any value for the determination of this question. Even large tumours of the occipital lobe which might injure the external geniculate body by compression do not afford trustworthy evidence. In a case described by Pooley, there was paresis of the right half of the body, diminished sensibility of the right arm, and a sharply-defined right-sided hemianopsia of both eyes. A tumour

was found in the left occipital lobe; but, in addition, the left optic thalamus and the surrounding cerebral substance were completely softened. Another case is described by Hirschberg, in which there were aphasia, right-sided hemiparesis, and right-sided hemianopsia. A tumour was found in the left occipital lobe, surrounded by softened tissue, which extended to the optic thalamus. It is difficult to understand how the external geniculate body could escape being diseased in such a case. Wernicke reports a case in which the symptoms consisted of aphasia, agraphia, alexia, and right-sided hemianopsia. Extensive softening was found in the convexity of the left hemisphere. The area of softening reached posteriorly 2 cm. behind an ideal line drawn vertically downwards from the parieto-occipital fissure; superiorly, it was limited by the intraparietal sulcus; anteriorly, it extended to the ascending parietal convolution above the Sylvian fissure, and involved the superior middle temporosphenoidal convolutions below it. The softening penetrated into the white substance till it reached the ependyma of the posterior horn of the lateral ventricle. The left corpus striatum—both the caudate and lenticular nuclei—was softened; but the optic thalamus, the geniculate body, the corpora quadrigemina, and the optic tracts were normal. Baumgarten mentions a case observed by Jacobson and Jaffe, in which the left halves of the fields of vision became suddenly lost. The affection of sight continued unchanged until death, which occurred a few months later from aortic regurgitation. An apoplectic cyst, about the size of a walnut, was found in the substance of the right occipital lobe, and a small hæmorrhagic focus in the centre of the right optic thalamus. Nothnagel reports a case of left brachial monoplegia, with right-sided hemianopsia of both eyes. The autopsy revealed carcinoma of the pancreas, with secondary deposits in the liver and stomach. The right hemisphere presented yellow softening of the middle third of the ascending frontal and parietal convolutions, which penetrated deeply into the underlying white substance and into the superior parietal lobule. About the size of a hazel-nut of the convolutions on each side of the intraparietal sulcus was of a grey-yellow colour, while the softening extended in the underlying white substance down to the wall of the descending horn of the lateral ventricle. The third occipital convolution of the right hemisphere was also softened. A spot of red softening, about the size of a hazel-nut, was observed in the right optic thalamus. In the left hemisphere the posterior extremity of the second frontal convolution, along with a small part of the adjoining portion of the ascending frontal convolution, was softened. Another small focus of softening was found in the anterior part of the superior parietal lobule, while the whole cortex of the occipital lobe was changed into a softened mass of a dirty yellow colour. No changes were observed in the optic nerves or tracts. Softening was observed in the inferior part of the cervical enlargement of the spinal cord.

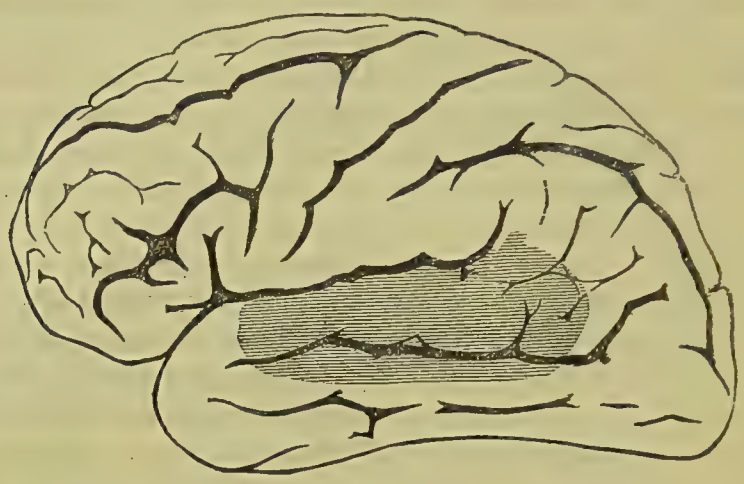
Of the cases just mentioned of disease of the occipital lobes, associated with hemianopsia, only one or two possess real value. In Pooley's case,

the left optic thalamus and surrounding cerebral substance is described as being extensively softened, and it is difficult to understand how the external geniculate body could escape under such circumstances. In Hirschberg's case, the softening which surrounded a tumour in the occipital lobes extended as far as the optic thalamus; and, again, the external geniculate body would be very liable to be diseased. Wernicke's case is, on the other hand, more convincing, but even in it the corpus striatum is mentioned as being softened. It is not contended that the softening of the corpus striatum alone would account for the hemianopsia; but it must be remembered that the surcingle of the caudate nucleus passes in close proximity to the external geniculate body. It is, however, mentioned specially that the geniculate bodies were healthy in this case, so that it must be held to favour the idea that hemianopsia may be caused by disease of the occipital lobe. Baumgarten's case also points to the same conclusion, for although a hæmorrhagic focus was found in the centre of the right optic thalamus, yet its small size and the position it occupied renders it improbable that this was the cause of the hemianopsia. Nothnagel's case is not above suspicion. The red spot in the thalamus probably occurred, as the author asserts, during the last stages of life, and could not, therefore, have caused the hemianopsia; but the lesions observed were so extensive and complicated that it would not be safe to attach much importance to the case. Of the cases just described, those of Wernicke and Baumgarten, and in a less degree that of Nothnagel, are the only ones to which any importance need be attached as indicating that bilateral hemianopsia may result from disease of one of the occipital lobes, but these cases can only be regarded as affording a presumption in favour of this opinion. It is right to add that Bellouard, who has written an admirable monograph on the subject of hemianopsia from cerebral disease, believes that typical bilateral hemianopsia may be caused by disease in the posterior part of the hemisphere a short distance behind the radiations of Gratiolet's fibres. This question must, therefore, be left for future observations to determine.

Lesions of the Temporo-sphenoidal Lobe.—Lesions of the temporo-sphenoidal lobe are often latent as regards symptoms. Charcot and Pitres report a case which presented no sensory or motor disturbances during life, but in which yellow softening was found after death in the cortex of the right hemisphere. The diseased area occupied the posterior half of the Island of Reil, the posterior half of the second and third temporo-sphenoidal convolutions, and the lower two-thirds of the inferior parietal lobule. Ferrier places the auditory centre in the first and second temporo-sphenoidal convolutions, but there is no case on record in which disease of the cortex of the brain has given rise to deafness. The reason of this is that hearing is bilaterally associated, and so long as one hemisphere is unaffected the auditory sense remains unimpaired or only slightly weakened. The condition already described as word-deafness is, however, associated with disease of the first and a portion of the

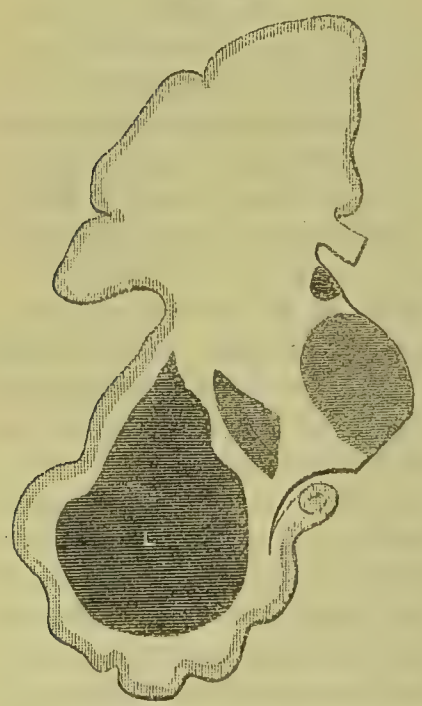
second temporo-sphenoidal convolution. In a case of this kind reported by Wernicke there was softening from thrombosis of the first and a large portion of the second temporo-sphenoidal convolution of the left hemisphere

FIG. 268.



(*Fig.* 268). Dr. Shuttleworth reports a case of microcephalic imbecility, in which hearing was dull during life, and at the autopsy congenital deficiency of the occipital and temporo-sphenoidal lobes was found.

FIG. 269.



The case of a woman, aged 64 years, is described by Pitres, in which death occurred a few hours after an apoplectic attack. Although there was almost complete loss of consciousness from the first, there was no paralysis of any of the limbs, inasmuch as all of them were moved on being strongly pinched. There was no rotation of the head or deviation of the eyes, but the left pupil was more dilated than the right. At the autopsy a recent hæmorrhage was found occupying the whole of the white substance of the sphenoidal lobe (*Fig.* 269). The basal ganglia were healthy.

Ferrier found that destruction of the *subiculum cornu Ammonis* causes loss of smell on the *same* side, while in *hemianæsthesia* from disease of the posterior fibres of the posterior segment of the internal capsule the loss of smell is on the side *opposite* the lesion. As already described, the olfactory nerve has two roots, one of which passes directly to the subicular region of the same side, while the other crosses over to the opposite hemi-

sphere through the anterior commissure of the third ventricle. It is not, therefore, probable that unilateral cortical disease will cause complete anosmia of one nostril.

The anosmia of the opposite nostril, the result of disease of the posterior fibres of the internal capsule, is caused partly by the loss of common sensation in the nose, from severance of the fifth nerve from the cortex, and partly by destruction of the fibres of the internal root of the olfactory nerve. Loss of smell, either alone or associated with diminution of taste, not unfrequently results from blows on the occiput or vertex of the head. Dr. Ogle has described several cases of this kind, and he thinks that the symptom is due to injury of the olfactory nerves, bulbs, or tracts by counterstroke. When the occiput receives a blow, the skull, being elastic, may yield without fracture, and the whole of the cerebral mass is then thrust forwards against its anterior wall. The temporo-sphenoidal lobe must be the first to impinge against the wings of the sphenoid bone, and the forward movement of this lobe is suddenly arrested, while the upper part of the cerebrum is allowed to move forwards until it is arrested by the frontal bone. It is evident that the sudden arrest of the temporo-sphenoidal lobe must tend to rupture the roots of the olfactory tract. The forward movement of the upper portion of the cerebrum will also tend to carry with it the olfactory bulbs, and thus to rupture the olfactory nerves as they pass vertically through the cribriform plate of the ethmoid bone.

A case of abscess of the temporo-sphenoidal lobe is reported by Dr. Glynn, in which the most prominent symptom was complete anosmia. The symptoms consisted of noises in, and partial deafness of the left ear, amblyopia and dyschromatopsia of the left eye, neuralgic pains over the temporal region and above the left ear, the scalp being swollen over these regions, a crop of herpes on the left ala nasi, paresis of the left masseter, slight facial paralysis of the left side of the body, loss of taste over the left half of the tongue, ptosis of left eyelid with contraction of the pupil on that side, double optic neuritis, complete anosmia, and *quasi* hysterical attacks. At the post mortem a circumscribed abscess, about two inches in length, was found situated in the anterior part of the first temporo-sphenoidal convolution, and extending inwards and downwards towards the base of the brain. With the exception of the anosmia, the localising symptoms in this case were caused by compression of the cranial nerves at the base of the brain in the anterior fossa of the skull. Dr. Glynn appears to think that the loss of smell was caused by implication of the cortical centre, but it is more likely to have been caused by compression of the external root of the olfactory tract of the same side at its point of entrance into the temporo-sphenoidal lobe and of the fibres which cross in the anterior commissure of the third ventricle.

Ferrier localises the centre of tactile sensibility in the hippocampal region, but unilateral lesions of the hippocampal convolutions are not known to produce anæsthesia. In the cases of disease of the sphenoidal

irritative or paralytic disturbances, the muscles of the face and neck being first implicated, then those of the arm, and those of the leg last. When lesions of the præ-frontal region are attended by active delirium, or convulsions, it is probable that the primary focus is surrounded by a more or less diffused encephalitis, or at least by a zone of tissue in a state of irritation, which extends to the cortex of the motor area. The characteristic features of lesions in the præ-frontal region of the cortex are afforded by the psychical disturbances, consisting of dementia, apathy, and somnolency. When convulsions are present, they are not preceded by an aura, and the spasmodic phenomena are of short duration, while the stage of insensibility is comparatively prolonged.

CHAPTER VIII.

(II.) SPECIAL CONSIDERATION OF FOCAL DISEASES, ACCORDING TO THE LOCALISATION OF THE LESION (CONTINUED).

3. LESIONS OF THE BASAL GANGLIA, EXTERNAL CAPSULE, AND CLAUSTRUM.

LESIONS of the basal ganglia have already been considered in a general manner along with the affections of the internal capsule, and little remains but to show that those limited to the ganglia themselves do not give rise to decided symptoms during life, or at least that these symptoms are not of an enduring character.

(*a*) *Lesions of the Lenticular Nucleus.*

§ 751. Several cases are now on record in which the lenticular nucleus had been found at the autopsy converted into a cyst, containing serous fluid, but in which paralysis of the opposite side of the body had been completely absent during life (Lépine, Charcot, Nothnagel). When a history of the symptoms can be obtained it is found that the patient had some months or years previously suffered from an attack of apoplexy, followed by temporary hemiplegia. The patient, however, makes a good recovery, and the cerebral attack from which he suffered may be completely forgotten, so that the lesion of the lenticular nucleus is revealed quite unexpectedly at the autopsy. A woman, aged 57 years, suffering from *tabes dorsalis*, was under the observation of Nothnagel for six months before her death, during which time she had no cerebral symptoms, yet a diseased focus was found in the posterior and inferior angle of the right lenticular nucleus.

In the case of pseudo-bulbar paralysis already described, which came under my own observation, both lenticular nuclei were converted into cysts, and yet there was no paralysis of the extremities during life.

Tumours of the lenticular nucleus generally give rise to hemiplegia of the opposite side, the paralysis being sometimes preceded by spasmodic contractions. Speech was affected in six out of sixteen cases of tumours of the lenticular nucleus collected by Ladame, but the size of the tumours in some of these cases precluded the idea that they could have been limited to the area of the lenticular nucleus. In two cases there was difficulty of articulation, in three slowness of speech, and in one only aphasia. The difficulty of articulation probably depended upon compression of the geniculate tract of the internal capsule, the aphasia upon simultaneous compression of the geniculate fibres and those of the corpus callosum which connect the posterior extremities of the third frontal convolutions with one another, while the slowness of speech might either be a symptom of general compression of the brain or of special compression of the Island of Reil and the posterior extremity of the third frontal convolution. A large tumour of the lenticular nucleus might compress the optic tract, either at its origin in the external geniculate nucleus, or as it winds round the crus cerebri, and then bilateral hemianopsia of the opposite side would be present. Tumours, however, which remain limited to the lenticular nucleus do not give rise to decided paralysis.

The case of a woman, aged 30 years, is described by Fürstner, to whom two grammes of chloral had been given as a hypnotic on account of puerperal mania, and who suffered from symptoms of chloral poisoning. She had repeated rigors, lowering of the temperature of the body, palpitation, and acute œdema of the lungs. For some days she complained of general weakness, while an erythematous eruption appeared over the body, and a bed-sore over the sacrum. Pneumonia now supervened, and the patient died seven days after the administration of the chloral. At the autopsy, besides the usual signs of pneumonia, a teleangiectatic glioma was found symmetrically placed on each side, and occupying the position of the middle and internal divisions of the lenticular nuclei, the third division being free on both sides.

Fürstner ascribes the feeling of general feebleness, of which

the patient complained, to the toxic action of the chloral; and even supposing that this feeling was a bilateral hemiparesis caused by the tumours, it must be remembered that the symptom only appeared a week before death; besides it is probable, from the position of the tumours, that the fibres of the internal capsule suffered a certain amount of injury. A somewhat similar case is described by Rondot.

A man, aged 30 years, complained of pains in the neck and head, and of a feeling of weakness of the extremities, but was able to walk about. At no time did the case present any paralysis, contractures, or sensory disturbances. Death occurred somewhat suddenly; and at the autopsy two tumours were found, each being about the size of a large hazel-nut, and symmetrically placed in the hemispheres. A transverse vertical section showed that each tumour occupied the position of the lenticular nucleus; the fibres of the internal capsule were compressed and pushed inwards, while the cerebral substance was softened to the extent of three or four mm. in thickness on the external surfaces of the tumours. The tumours were dense and white, though their peripheral zones were vascular.

A case of syphiloma of the brain has been observed by Schütz, and one of tubercular tumour by Bramwell, in each of which the tumour occupied the position of almost the whole of the lenticular nucleus of the left side, and in neither were there symptoms of a localised cerebral affection. It may, therefore, be laid down, as a general rule, that acute lesions limited to the lenticular nucleus give rise to a transitory hemiplegia of the opposite side, while this symptom may be entirely absent in chronic stationary lesions and slow-growing tumours.

(*b*) *Lesions of the Caudate Nucleus.*

§ 752. Lesions of the caudate nucleus do not, any more than those of the lenticular nucleus, give rise to permanent symptoms during life, unless the internal capsule be implicated. Small cystic cavities and foci of softening are frequently found in the caudate nucleus at a post-mortem examination, in the absence of all history of cerebral symptoms during life. Other cases are reported in which a slight hemiplegia had occurred during life, followed by a speedy recovery, and in which a focal lesion was subsequently found in the caudate nucleus.

The following case observed by myself illustrates the

symptoms which may be present during the growth of a tumour of the caudate nucleus.

Samuel Holmes, æt. 7 years, presented himself as an out-patient at the Southern Hospital, Manchester, on January 26th, 1876.

The following history was elicited from the mother:—He was a bright, intelligent, and healthy boy until about 15 months ago, when he fell from a wall, 5 feet high. Soon afterwards he complained of constant headache, chiefly confined to the forehead. The top of the head was so sensitive that combing his hair caused him much pain. He could not keep still; his legs, especially, were constantly moving, and at meal times he was in the habit of knocking the table with his right hand, as if from impatience. About nine months ago the mother noticed that his mouth was slightly "crooked," and that his left arm hung helplessly by his side. The forearm was twisted so that the palm of the hand was directed outwards and the thumb backwards, his fingers were bent, but she thinks his thumb at first was held straight and drawn away from the fingers. After some weeks, however, the thumb became bent inwards under the index-finger, and she had to pare the nail of the thumb frequently to prevent its cutting the skin of the outside of the middle finger. He now began to drag the left foot in walking, and the forearm was gradually drawn up behind his back, instead of hanging, as at first, by his side.

The mother had had nine of a family, no miscarriages and no still-born children. One child died from convulsions during teething; a second child, who was weakly from birth, died at the age of three months; and a daughter has suffered for the last two years from white swelling of the knee.

On presenting himself at the hospital he was a well-made and fully-developed boy for his years. His head was large, but well-proportioned; face round and plump, although pallid; his incisor teeth were regular, his nose was well formed, the muscular system was well developed, and there was abundance of subcutaneous fat. There was very well-marked left facial paralysis, so that the left corner of the mouth could not be moved. Both eyes could be closed; the pupils were large, equal, sensitive to light, and there was no affection of the special senses. The left elbow was kept a little behind the body, and 2 inches from the side; the forearm was bent at right angles to the arm, and drawn behind the trunk; the hand was strongly pronated; the thumb was adducted, and the second phalanx flexed, so that the point rested against the second phalanx of the middle finger. The first phalanges of the fingers were extended and in a line with the metacarpal bones, and the second and third phalanges were flexed. A considerable amount of muscular rigidity was induced on attempting passive motion at the elbow and wrist joints. By a voluntary effort he could raise his elbow to nearly the level of the shoulder, and then bring the upper arm slowly forwards; but he could neither extend the forearm, produce supination, nor extend the fingers. The left leg dragged during walking, but there was no muscular rigidity, and all

the movements of the leg could be separately performed. The electro-cutaneous sensibility of the left half of the body was increased, especially over the back of the left hand, and the left half of the face and side of the head. The slightest touch of the skin over the vertex of the head to the left of the middle line caused the patient to wince, and the cutaneous sensibility to pain was increased over the left half of the body generally. The other organs appeared to be healthy, and there was no albumen or sugar in the urine. He was ordered four grains of iodide of potassium three times a day; but, as no improvement took place, he was admitted into the hospital on February 28th.

March 10th, 1877.—He was ordered, on admission, fifteen minims of the syrup of the iodide of iron, to be taken three times a day, and the daily application of a weak constant current to the paralysed muscles and nerves. After two applications of the constant current he could extend his fingers to a slight extent, and in a few days he was able to raise his hand to the back of his head. It was observed, however, that the most marked improvement took place at the shoulder-joint; and that improvement in the movements of the forearm and hand was only to a slight extent. This improvement was of short duration, and he now looks decidedly worse than on admission. The pallor of the face is much increased; his appetite has failed; the pulse is 110, weak and irregular; and the nurse says that he has become very stupid. Ordered to be kept in bed, milk diet and a saline mixture.

March 18th.—Since last report he has got steadily worse, has vomited frequently, and to-day has been seized with general convulsions. The convulsions frequently recurred during the next two days, consciousness not being recovered in the intervals, and he died early on March 21st.

Sectio cadaveris, twelve hours after death.—On opening the skull, the convolutions of the brain presented a flattened and compressed appearance, and about 2 ounces of fluid escaped during removal. The brain weighed 51 ounces. On slicing the brain to a level with the corpus callosum the upper surface of a tumour was exposed, which was situated in the centrum ovale of the right hemisphere, immediately to the right of the corpus callosum and at the junction of the anterior and middle lobes. On opening the lateral ventricles, this tumour was felt as a hard nodule, slightly projecting into the right lateral ventricle, and occupying the position of the caudate nucleus and anterior portion of the optic thalamus, and only covered by the ependyma of the ventricle. The tumour measured three-quarters of an inch in the transverse and an inch in the antero-posterior and vertical diameters respectively, so that not only the caudate nucleus and anterior portion of the optic thalamus, but also the anterior two-thirds of the internal capsule and the anterior portion of the lenticular nucleus were destroyed by it.

The growth was pretty sharply defined from the surrounding brain-tissue, and on section it presented an outer grey, somewhat vascular cortex, about two lines thick, and a central core of a yellow colour, and apparently destitute of any structure.

Microscopic examination showed that the grey cortex of the tumour consisted of giant cells, each surrounded by lymphoid cells imbedded in a fibrillated reticulum.

The right lung was closely adherent to the chest wall and to the diaphragm. The lung itself was congested, but every portion of it floated in water. No tubercles nor cheesy glands were discovered, and the other organs were healthy.

Fig. 271.

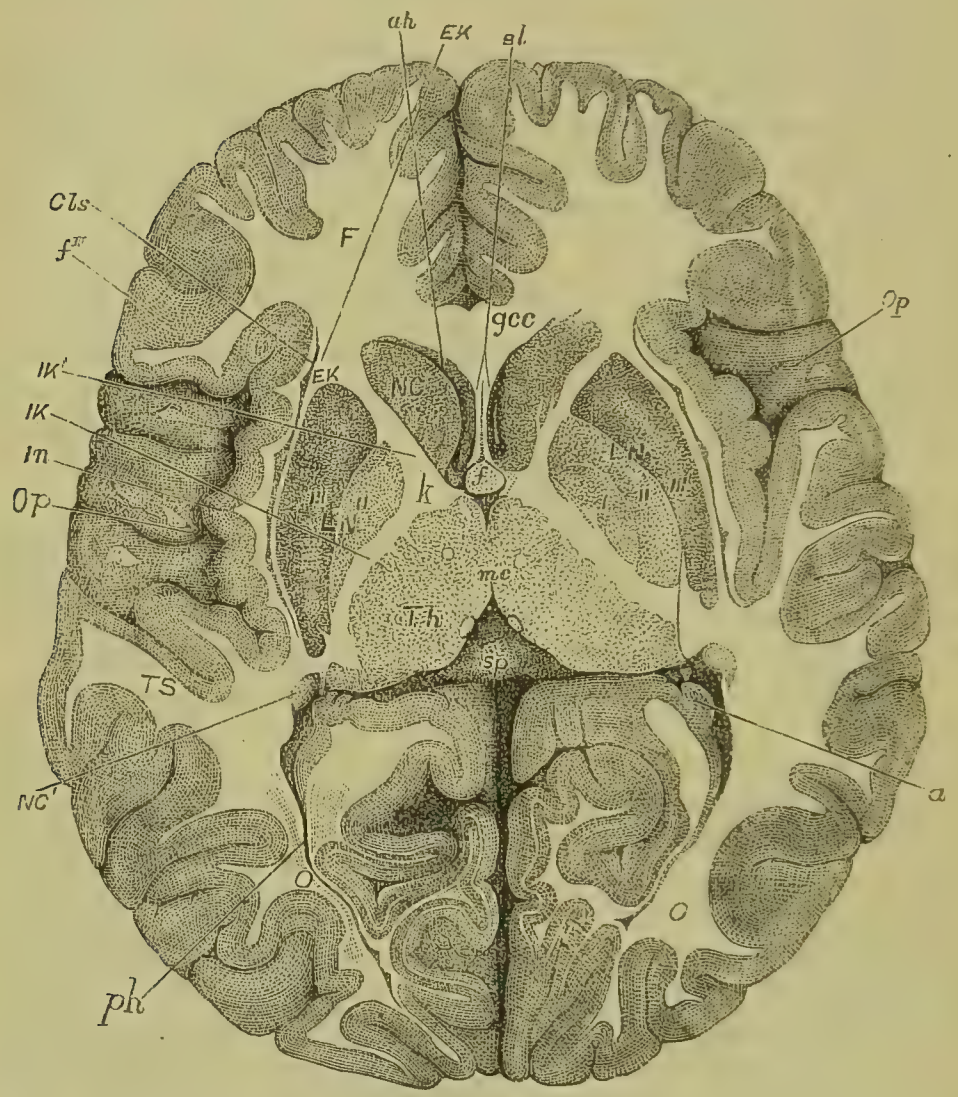


Fig. 271 (From Flechsig). *Horizontal Section of the Brain of a Child nine months of age, the right side being at a somewhat lower level than the left half.*—*F*, Frontal, *TS*, Temporo-sphenoidal, and *O*, Occipital lobes; *Op*, Operculum; *In*, Island of Reil; *Cls*, Claustrum; *f'''*, Third frontal convolution; *Th*, Optic thalamus; *NC*, Caudate nucleus; *NC'*, Tail of caudate nucleus; *LN*, Lenticular nucleus; *I*, *II*, *III*, First, second, and third divisions of the lenticular nucleus; *EK*, External capsule; *IK*, Posterior division, *IK'*, Anterior division, and *K*, Knee of the internal capsule; *ah*, *ph*, Anterior and posterior horns respectively of the lateral ventricles; *gcc*, Knee of the corpus callosum; *sp*, Splenium; *mc*, Middle commissure; *f*, Fornix; *sl*, Septum lucidum; *a*, Cornu Ammonis.

The tumour in this case appeared to have commenced growing in the caudate nucleus (*Fig.* 271, *NC*), although it ultimately extended to the anterior half of the lenticular nucleus (*Fig.* 271, *LN*), and completely destroyed the anterior segment of the internal capsule (*Fig.* 271, *IK'*). The most remarkable feature about the symptoms was that the face was more paralysed than the arm, and the arm than the leg, this being the order in which the paralysis might be expected to appear, provided pressure was exerted on the internal capsule from before backwards. The hyperæsthesia of the head was probably caused by irritation of the posterior fibres of the internal capsule.

(*c*) *Lesions of the Optic Thalamus.*

§ 753. The lesions of the optic thalamus by which the fibres of the internal capsule suffer damage have already been considered. Acute lesions of the thalamus except those of small size are associated with more or less paralysis of the opposite side of the body, but unless the lesion be large the paralysis is only temporary, and it is probable that when permanent paralysis results the fibres of the pyramidal tract are always injured. Lesions of the thalamus are also frequently associated with hemianæsthesia, but the sensory phenomena are in nearly all cases caused by injury of the sensory peduncular tract in its ascent through the internal capsule, and of the optic radiations of Gratiolet.

Lesions of the optic thalamus are frequently associated with bilateral hemianopsia of the side opposite the lesion. When the lesion of the thalamus is of the nature of hæmorrhage or softening, the hemianopsia is probably caused by implication of the external geniculate body in the diseased focus; but when the lesion is a tumour of the thalamus, the defect of sight is often the result of pressure on the optic tract as it winds round the crus cerebri.

(*d*) *Lesions of the Corpora Quadrigemina.*

§ 754. Tumours of the corpora quadrigemina are of rare occurrence. Out of the 331 cases of intracranial tumour collected by Ladame only two were situated in the corpora quadrigemina. Several cases of disease of these ganglia have been

recorded since, but in the majority of them the lesion has not been strictly limited to these bodies. The more usual symptoms of lesion of the corpora quadrigemina are disturbances of muscular co-ordination, disorders of the movements of the eyeballs and iris, and defects of vision.

The disturbances of muscular co-ordination consist of staggering and difficulty of maintaining the erect posture, the symptoms being similar to those caused by disease of the peduncles of the cerebellum. And, indeed, when the anatomical relations between the corpora quadrigemina and the superior peduncles of the cerebellum are considered, it becomes doubtful whether these motor disturbances ought not to be attributed to implication of the latter. The disturbances in the ocular movements are generally caused by paralysis of individual branches of the oculo-motor nerves; and Nothnagel thinks that those disorders are more likely to occur when the posterior pair of ganglia are diseased. The retino-pupillary reflex is also abolished. No definite statement can be made with regard to the state of the pupils. When the anterior pair are affected, blindness is apt to supervene at an early period of the affection, and often precedes the development of optic neuritis in cases of tumour. In two cases of tumour of the cerebellum which I observed, and in which the corpora quadrigemina were secondarily implicated, blindness was an early symptom, and was complete in both cases before the secondary atrophy of the discs had advanced far, although not before the appearance of double optic neuritis.

(*e*) *Lesions of the Claustrum and External Capsule.*

§ 755. A case is described by Brault and Beurmann of a man, aged 71 years, who a few weeks after an injury had an apoplectic attack. On the following day there was slowness of speech, and paralysis of the right half of the body including the face. Three days after the attack speech was still slow, but the facial paralysis had disappeared, and the paralysis of the extremities was much improved, while every symptom of the attack had disappeared six days from its onset. A few days subsequently the patient died from causes unconnected with the apoplectic attack, and the left claustrum and external capsule were found

completely destroyed by a hæmorrhagic focus, 3 cc. long, 2½ cc. in depth, and only 2—3 mm. broad. The hemiplegia in this case was doubtless caused by a temporary slight pressure on the fibres of the internal capsule, while the affection of speech was occasioned probably by pressure on the Island of Reil, but none of the symptoms could be attributed to the destruction of the claustrum and external capsule themselves.

(*f*) *Lesions of the Base of the Skull.*

(i.) Anterior Fossæ of the Skull.

§ 756. Records of lesions limited to the anterior fossæ of the skull are not numerous. Disturbances of smell are not unfrequently present in chronic basal meningitis, but the lesion almost always extends beyond the anterior fossæ, and gives rise to complications. The symptoms caused by tumour in this region are variable, but the most trustworthy is afforded by compression of the olfactory bulbs or tracts. Several cases of tumour in this region have been collected by Longet, of which the following is an example :—

A woman, aged 59 years, suffered from recurrent attacks of dizziness, feelings of formication, and numbness of the left half of the face. After a period of four years she began to suffer from epileptiform convulsions, but enjoyed good health in the intervals. A year later the patient experienced a peculiar disorder of smell, which she could not accurately describe, immediately before each attack of dizziness, and this last was followed by an epileptiform attack. After the expiration of another year the disorder of smell had became less troublesome and finally disappeared. The terminal symptoms consisted of attacks of severe dizziness, frontal headache, loss of intelligence, and coma. At the autopsy a cancerous tumour the size of a duck's egg was found in the anterior lobe of the left hemisphere, lying on the dura mater of the anterior fossa, and extending from the lamina cribrosa to the olfactory roots. The left olfactory tract was completely destroyed.

(ii.) Lesions of the Middle Fossæ of the Skull.

§ 757. Diseases situated in the middle fossæ of the skull are liable to implicate many important structures as the olfactory nerves, the optic commissure and tract, as well as the third, fourth, fifth, sixth, seventh, and eighth nerves. It will thus be seen that lesions of this fossa must give rise to very compli-

cated symptoms. The following case reported by Ziemssen illustrates the symptoms caused by basal meningitis, mainly limited to the pia mater and lying over the middle fossa of the skull.

A man, aged 33 years, contracted syphilis in 1855, and suffered from secondary symptoms. On March 9th, 1856, while walking on the street, he suddenly observed double vision, associated with ptosis of the left eyelid, both symptoms remaining constant. In June he complained of intense headache, the mouth was drawn to the left, and there was difficulty of articulation and deglutition. Great general weakness gradually developed, so that the patient could not stand alone, and when supported by two attendants his gait was staggering. On August 11th, the patient was found in a state of great emaciation; there was inability to close the right eye, owing to paralysis of the lower lid; while the upper lid could be energetically moved, the right eye could be moved upwards, inwards, and downwards, but not outwards, and only slightly outwards and downwards. The pupil was contracted, and reacted feebly to light. In the left eye there was complete ptosis, and the eyeball was immovable. The pupil was dilated and fixed. Apart from diplopia, vision in both eyes was good. The right facial nerve was completely paralysed, and the muscles did not react to the faradic current; the left facial muscles reacted feebly. The movements of the tongue were normal. The sensory disturbances in the region of distribution of the fifth nerves, and the senses of taste and smell, were unaffected. The movements of both the lower and the left upper extremity were normal, but the grasp of the right hand was feeble, the power of moving the fingers was incomplete, and the interossei muscles and those of the ball of the thumb were atrophied. There were no sensory disturbances of the body or extremities. In this case the right facial, left oculo-motor, right trochlear, and the two sixth nerves were completely paralysed, while the right oculo-motor and left facial were partially paralysed. Death took place on August 9th from pulmonary phthisis. The pia mater corresponding to the middle lobes was thickened, while the diseased area extended towards the optic commissure. The pia mater was thickened over the left oculo-motor nerve, which was congested, in some parts thickened and softened, and in others atrophied. In the thickened parts a microscopic examination revealed fine fat globules, without a trace of nerve fibres. The right facial and trochlear nerves were altered in the same manner as the left oculo-motor. A certain degree of thickening was found at the origin of the right oculo-motor nerve; but a microscopic examination revealed, besides fat globules, healthy nerve fibres. The left facial and the two acoustic nerves presented morbid appearances similar to those found in the right oculo-motor nerve. Both the sixth nerves were flattened. The olfactory, optic, trigeminal, hypoglossal, spinal accessory, glosso-pharyngeal, and pneumogastric nerves were normal. The brain was healthy.

The changes in this case extended beyond the middle fossæ of the skull, inasmuch as the facial and acoustic nerves were implicated in the disease, but it nevertheless affords a good example of the symptoms caused by a chronic basal meningitis. In a patient under the care of Dr. Simpson, sent to the Cheadle Convalescent Hospital a few months ago, the following were the leading symptoms observed:—

A man, aged 49 years, suffered for some time from headache and blindness of the right eye. When I examined him in July, 1880, there was complete blindness of the right eye, with atrophy of the optic disc,

FIG. 272.

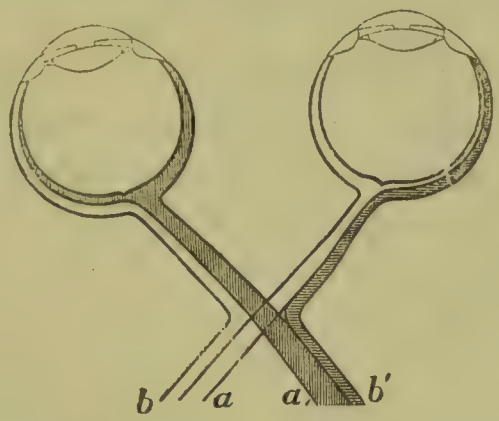


FIG. 272 (From Landois' "Physiologie").—Schema of the semi-decussation of the fibres of the Optic Commissure; *b a*, Left Optic Tract, the fibres of which are distributed to the left halves of both retinæ; *a' b'*, the Right Tract, with its fibres supplying the right halves of both retinæ.

complete immobility of the eyeball, ptosis, and exophthalmos from paralysis of all the ocular motor nerves. Vision was good in the left eye, the field of vision in it was not restricted, and there were no changes in the optic disc. The patient had suffered for some time from neuralgic pains in the region of distribution of the first and second division of the fifth nerve, and at the time of examination the sensibility of this region was blunted but not abolished. A tenacious secretion covered the right eyeball, but the cornea was not cloudy. The right masseter muscle was feeble and decidedly atrophied in comparison with the corresponding muscle on the left. The man died two weeks after my visit, and at the autopsy Dr. Massiah found the right optic nerve close to the foramen, surrounded by a ring of dense, apparently cicatricial, tissue. This thickened tissue, which did not form a distinct tumour, had extended backwards above the cavernous sinus and spread over the Gasserian ganglion and motor root of the fifth nerve. The right optic tract (*Fig.* 272, *a' b'*) was unaffected.

Tumours of the middle fossa of the skull generally give rise

to still more definite symptoms. The symptoms in the following case, under the care of Dr. Dreschfeld, deserve to be compared with those of the case which has just been described.

A man, aged 40 years, suffered for some time from symptoms of cerebral disease. For some weeks before his death his symptoms were blindness of the right eye with atrophy of the disc, ptosis, immobility of the eyeball, and paralytic exophthalmos of the right eye; after a time neuro-paralytic ophthalmia of the same, right facial prosopalgia, optic neuritis of left disc, with temporal hemianopsia of the left eye, and polyuria. The post-mortem was conducted by myself. I found a sarcomatous tumour lying close to the right optic foramen, and compressing the optic nerve; it extended backwards over the cavernous sinus, and compressed all the nerves lying in its walls, and likewise the right optic tract (*Fig.* 272, *a' b'*).

Most of the symptoms in this case were so definite that they scarcely require a word of explanation. The blindness of the right eye and atrophy of the disc were caused by the injury of the optic nerve at its point of entrance into the optic foramen, the temporal hemianopsia of the left eye by compression of the right optic tract, the optic neuritis of that eye was the usual form indicative of the presence of an intracranial tumour, while the paralysis of all the ocular muscles was caused by compression of the third, fourth, and sixth nerves as they pass along the wall of the cavernous sinus. The polyuria present in this case is not an unfrequent symptom of tumours in the neighbourhood of the pituitary body, and glycosuria is sometimes observed in such cases.

Aneurism of the internal carotid artery at the base of the skull does not appear to give rise to any characteristic symptoms, and auscultation of the cranium has not proved of much service. Besides a constantly recurring and distressing headache, the more usual symptoms of aneurism of the internal carotid at the base of the skull are unilateral or bilateral disturbances of vision from compression of the optic tract or nerve, spasms soon followed by paralysis of the ocular muscles, hyperæsthesia, and neuralgia, followed by anæsthesia in the region of distribution of the fifth nerve and great mental disturbances. Paralysis of the extremities of the opposite side may occasionally occur from compression of the pyramidal tract on its way through the crusta.

(iii.) Hæmorrhage into the Lateral Ventricles.

§ 758. Hæmorrhage into the ventricles is followed by deep coma, and the majority of patients die in the course of the first or second day; although occasionally they may live for several days. Hæmorrhage into the lateral ventricles constitutes the majority of those cases which have been called *ingravescent* apoplexy, but some of these are caused by a large hæmorrhage into the centrum ovale, or on to the surface of the brain from the bursting of an aneurism. When rupture into the lateral ventricle takes place the corpus callosum and fornix become partially destroyed, and the hæmorrhage makes its way into the third ventricle and into the lateral ventricle of the opposite side, and passes through the aqueduct of Sylvius to the fourth ventricle. The first stage of hæmorrhage into the ventricle may consist of ordinary hemiplegia, commencing with an apoplectiform, epileptiform, or simple mode of onset. Indeed, the simple mode of onset is not an unusual one, since the hæmorrhage frequently begins in the head of the caudate nucleus, lenticular nucleus, or optic thalamus, and the primary symptoms produced are not well marked. But whenever the brain substance is ruptured, so that blood is poured in considerable quantity into the lateral ventricle, a severe apoplectic attack occurs, characterised by profound coma, general paralysis of the limbs, and dilated pupils. The rectal temperature sinks several degrees, and remains depressed for several hours; but if a fatal result do not speedily occur the initial depression is followed by a rapid rise, which continues, in cases about to prove fatal, till the death of the patient. Hæmorrhage into the lateral ventricle is frequently attended with a spasmodic contraction of the extremities of the opposite side, which may be either temporary or persist until death.

(iv.) Tumours in the neighbourhood of the Pituitary Body.

§ 759. Tumours of the pituitary body are usually of large size. They produce compression of the anterior perforated space, the olfactory tracts, optic commissures and roots of the optic nerves, the corpora albicantia, the posterior perforated space, and when the tumour is large the pons and peduncles of

FIG. 273.

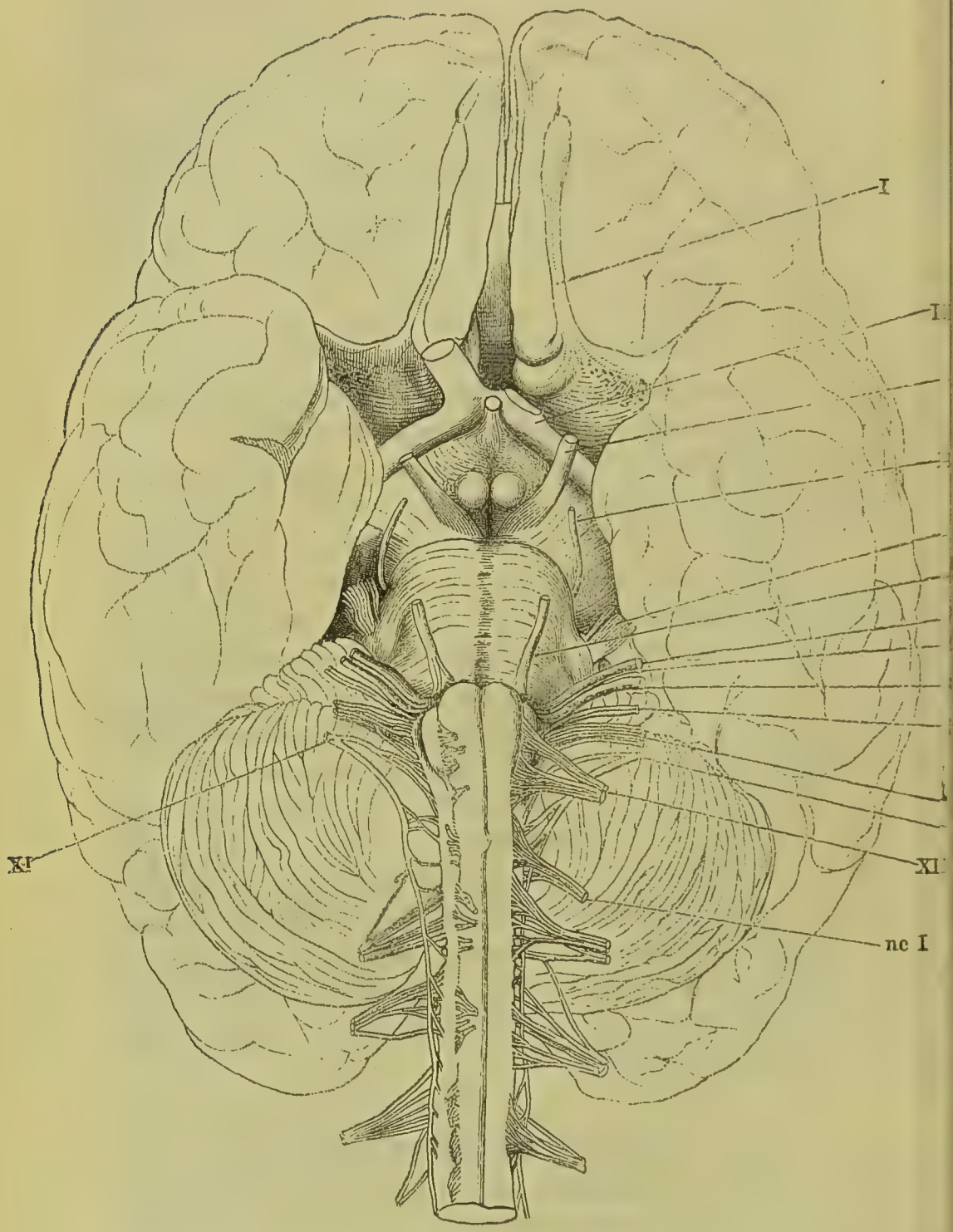


FIG. 273 (From Henlé's "Anatomie"). *The Base of the Brain and adjoining part of the Spinal Cord.*—The Cranial Nerves are represented by the corresponding Roman letters from I to XII. VII', Portio intermedia of the seventh; nc I, First cervical nerve.

FIG. 274.

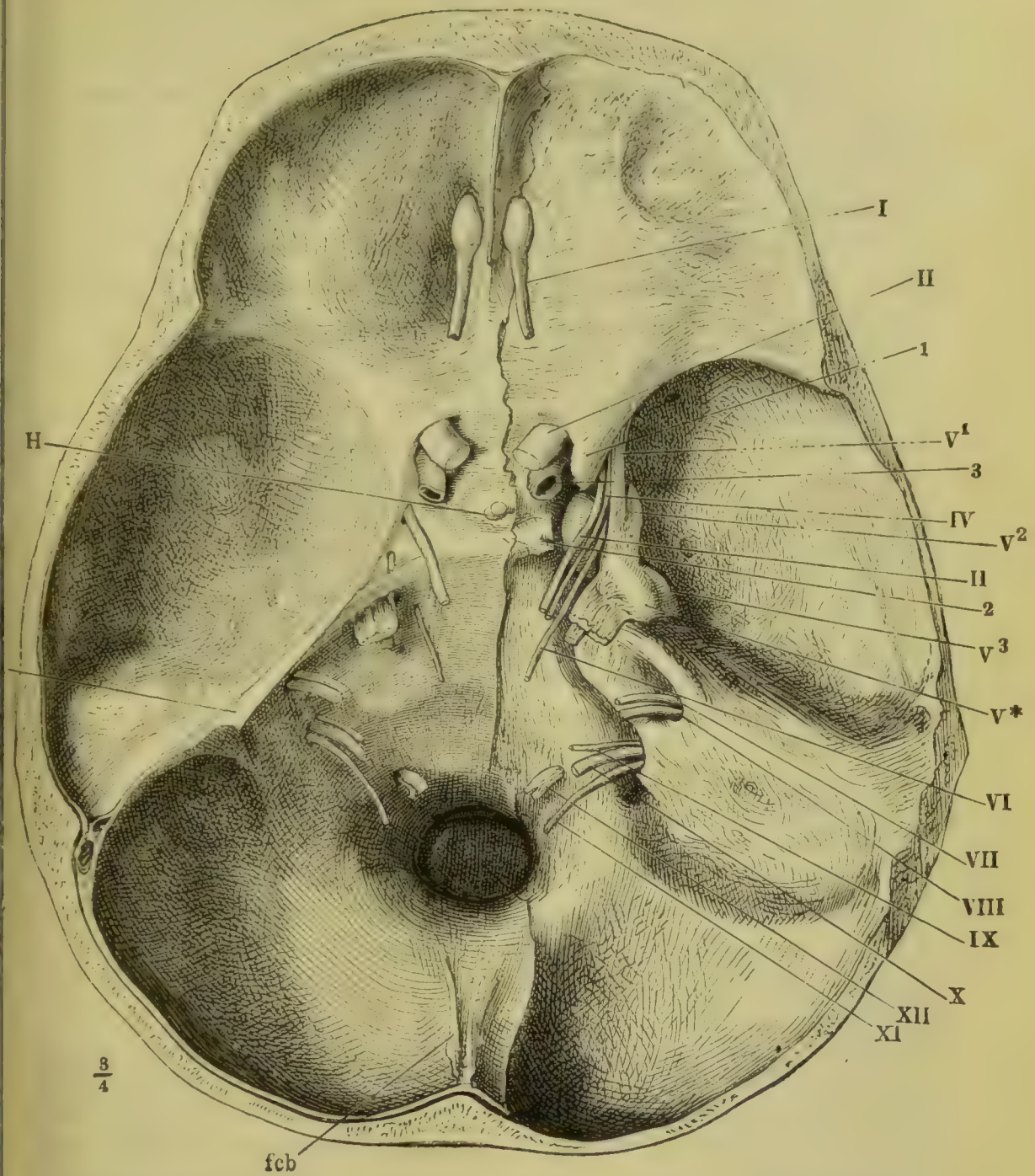


FIG. 274 (From Henlé's "Anatomie"). *Internal View of the Base of the Skull, showing the Places of Exit of the Cranial Nerves.* –The Nerves are represented by the corresponding Roman numerals, from I to XII. V^1, V^2, V^3, the first, second, and third divisions of the fifth respectively. V*, the Gasserian Ganglion. The sensory root is cut short in order to show the motor root of the fifth as it passes under the ganglion. The dura mater is removed on the right side, and the nerves may be followed to the various foramina through which they pass from the skull. The Nerves III, IV, V^1, and VI may be seen passing over the convexity of the curve, which the internal carotid artery makes in the groove of the sphenoid bone. 1, Anterior clinoid process; 2, Posterior clinoid process; 3, Transverse section of the internal carotid artery. H, Peduncle of the pituitary body; t, anterior body of the divided tentorium; fcb, Falx Cerebelli.

the cerebellum may be pressed upon and flattened. They may also encroach upon the cavernous sinuses and sphenoidal fissures and the nerves which pass through them, while the ventricles are not unfrequently distorted or obliterated. These tumours are also very liable to cause softening of the surrounding cerebral tissue, which may extend to the basal ganglia or the centrum ovale.

Periodical headache, usually situated in the frontal and temporal regions and extending forwards to one of the eyeballs and supra-orbital region, is one of the earliest symptoms of tumour in the neighbourhood of the pituitary body. Blindness occurs at an early period of the growth of these tumours, owing to their proximity to the optic commissure. It is important to remember that pressure on the optic commissure or nerves causes secondary atrophy of the discs without being preceded by the "choked disc."

There may also be unilateral or bilateral anosmia due to injury of the olfactory tracts. When the tumour exercises pressure on the cavernous sinuses, incomplete or complete paralysis of the motor nerves of the eye supervenes on one or both sides.

Disorders of cutaneous sensibility are rare and generally of a transitory nature. If the tumour be large, one or more of the branches of the fifth nerve on one or both sides is first irritated and then compressed. When a large tumour compresses the cerebral peduncles and interpeduncular space, spasmodic contractions of the muscles of the extremities, followed by hemiplegia or paraplegia, may supervene.

Two other interesting symptoms are sometimes associated with tumours in the vicinity of the pituitary body, the one being accumulation of fat in the subcutaneous tissue, and the other diabetes. In a case reported by Mohr, as quoted by Ladame, the patient, who subsequently died from tumour of the pituitary body, had become very fat before death; and in a case under the care of Dr. Simpson, in which the symptoms, consisting mainly of paroxysmal headaches and blindness, with simple white atrophy of both discs, pointed to the presence of a tumour in this neighbourhood, the patient became very fat after her illness. During her residence at the Manchester Royal In-

firmary she was passing a considerable quantity of pale urine, being only 1004 in specific gravity. In a case of tumour of the pituitary body reported by Rosenthal the patient voided from 8lbs. to 10lbs. daily, the specific gravity was from 1038 to 1040, and the urine contained a large quantity of sugar. Rosenthal conjectures that in cases of this kind the grey matter lining the third ventricle is first irritated, and that the irritation travels along the aqueduct of Sylvius to the floor of the fourth ventricle.

The following case is a good example of the symptoms caused by tumours in the neighbourhood of the pituitary body, and possesses a melancholy interest, inasmuch as a respected member of the medical profession was the victim of the disease :—

Mr. R——, about 34 years of age, consulted me on May 10th, 1878. He had suffered for some time from frequently recurring attacks of "faintness" and a peculiar defect of vision. He is a tall, dark man, of healthy appearance, there is a slight depression in the right frontal bone, caused by a blow received in childhood, but he does not appear to have suffered any inconvenience from it. During the fainting attacks, one of which I had an opportunity of observing, there is pallor of the face, the pulse beats from 130 to 140 in the minute, and the patient looks agitated, but there is no loss of consciousness. The duration of the attack is only about half a minute. On examining his eyesight he is found to be suffering from double temporal hemianopsia, the inner halves of the retinæ being blind. The optic discs are perfectly normal, this opinion being confirmed by Mr. Windsor.

February 7, 1879. The general symptoms continue unchanged, but the fainting attacks are now accompanied by temporary loss of consciousness. The patient is now blind on the right eye, while there is temporal hemianopsia of the left. Dr. Little, who made an ophthalmoscopic examination, reports white atrophy of the right and incipient atrophy of the left optic disc. The urine is abundant, pale, and of low specific gravity, but does not contain albumen or sugar. The only psychical disturbance observed was a marked and not unnatural tendency to brood over his own feelings and symptoms.

The diagnosis in this case presented no great difficulty. It was manifest that a tumour at the base of the skull was growing in such a way as to compress the centre of the chiasma at first, and that it subsequently extended to the right so as to have compressed the right optic nerve. I saw the patient two

or three times subsequent to the last report, but his symptoms remained unchanged. For the following report of the progress and termination of the case I am indebted to Dr. M'Fie, of Bolton.

On the evening of July 3rd, Mr. R—— was suddenly attacked with vomiting, followed by an epileptiform seizure, both symptoms recurring frequently during a period of an hour and a half. During the convulsive attacks the spasms were more pronounced on the left than on the right side. During that night the patient lay in a semi-comatose condition, but he gradually regained consciousness on the following day. It was now noticed that the left side of the face was paralysed, and that there was some degree of paresis of the left extremities.

August 30. Since last report the patient recovered the use of his limbs, and, although he has suffered from occasional attacks of sickness, he has walked about the garden as usual. This evening, however, he has had another epileptiform seizure.

September 10. Since last report the patient has been downstairs and walking about the garden, but after partaking of his usual breakfast this morning he became somewhat suddenly insensible, and died comatose at night. His temperature immediately before death was 106° F.

At the post-mortem examination the membranes of the brain over the convexity were found normal. The inner table of the skull and dura mater were normal at the point corresponding to the external depression of the right frontal bone. The substance of the brain was observed to be bulged between the frontal and parietal lobes in the right hemisphere. On removing the brain a lobulated tumour was observed lying to the right side of the optic commissure; the right optic nerve was compressed by it, but the left nerve occupied its usual position, and did not appear much altered. The tumour sprang from that portion of the dura mater which covers the body and basilar process of the sphenoid bone, the body of the bone being eroded by the growth. The orbital surface of the frontal lobe of the right hemisphere presented a deep excavation corresponding to the projection of the tumour, and the nervous tissues in the vicinity of the growth were somewhat softened. The tumour was multilobulated and of the colour of liver.

CHAPTER IX.

(II.) SPECIAL CONSIDERATION OF FOCAL DISEASES, ACCORDING TO THE LOCALISATION OF THE LESION (CONTINUED).

4. LESIONS LOCALISED IN THE STRUCTURES SITUATED BELOW THE TENTORIUM.

a. Lesions in the Pons and Peduncles of the Cerebrum.

(i.) LESIONS IN THE PONS.

§ 760. *Hæmorrhage into the Pons.*—If the hæmorrhage be of large size, profound apoplexy, with flapping of the cheeks during expiration, insensibility of the conjunctivæ, and strongly contracted pupils is produced. If the hæmorrhage extend upwards to the grey matter beneath the aqueduct of Sylvius, the ocular muscles may be paralysed, and the pupils dilated and fixed. Death occasionally takes place in a few minutes or in a few hours, although life may be prolonged for a day or two. Bursting of the hæmorrhage into the fourth ventricle is generally attended by convulsions, although convulsions may occasionally occur independently of this accident. If the patient recover from the shock of a central hæmorrhage into the pons, consciousness is gradually regained, but it is found that all the extremities are paralysed, while cutaneous sensibility may be more or less impaired. Double facial paralysis involving the muscles of the eyelids, as well as those of the mouth, may be present, both sides of the tongue are also paralysed, while the patient experiences difficulty in deglutition, there is a copious flow of saliva from the paralysed side of the mouth, and the power of articulation is impaired.

If the lesion be situated in the lower part of the lateral half of the pons, the symptoms may present the appearances characteristic of what Gubler has called *alternate hemiplegia*. There

is well marked facial paralysis on the side of the lesion, and a more or less complete motor and sensory paralysis of the limbs on the opposite side.

Fig. 275.

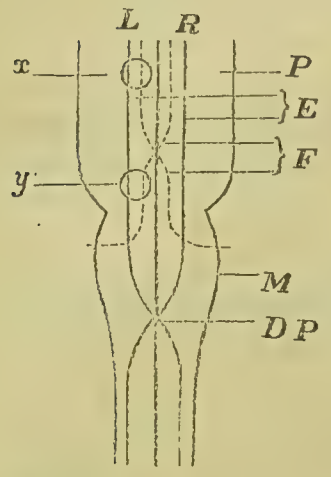


Fig. 275 (From Nothnagel).
L, Left.
R, Right.
P, Pons.
Mo, Medulla oblongata.
DP, Decussatio pyramidum.
E, Nerve fibres for the extremities.
F, Fibres destined for the facial nerve.
x, Lesion in the upper part of the pons.
y, Lesion in the lower part of the pons.

If the upper part of the lateral half of the pons be the seat of the lesion, the facial paralysis is on the same side as the paralysis of the extremities. In order to account for these phenomena it is necessary to assume that the fibres of the pyramidal tract, which connect the cortex of the opposite hemisphere with the nucleus of the facial nerve in the upper part of the medulla, cross over about the middle of the pons, as represented in the annexed figure.

Conjugate deviation of the eyes, with rotation of the head and neck, is a symptom of paralysis of the pons, but the rule with regard to the direction in which the rotation takes place is the converse of that which applies to lesions of the hemispheres. When the lesion is situated in the hemispheres, the head and eyes are turned towards the affected limbs during convulsions, and towards the side of the lesion in paralysis; but in lesions of one lateral half of the pons the head is turned towards the side of the lesion, if the limbs be convulsed, and towards the affected limbs when they are paralysed (Grasset).

Early rigidity of the muscles of the paralysed extremities, the masticatory muscles, and those of the neck, is often observed in lesions of the pons.

Disturbances of cutaneous sensibility are frequently met with in lesions of the pons, which become more profound and definite as the lesion approaches the upper or anterior end of the pons near the crusta. The sensory disturbances generally consist of anæsthesia, although unilateral hyperæsthesia has occa-

sionally been observed, and either condition may be accompanied by painful sensations in the limbs, or by a subjective feeling of coldness, even when the temperature of the part is higher than natural. Implication of the fifth nerve gives rise to anæsthesia, hyperæsthesia, paræsthesiæ, or painful sensations in the region of distribution of the nerve, as well as to partial impairment or perversion of taste on the side of the lesion. The masticatory muscles are often weakened or completely paralysed on the side of the lesion. Acute lesions of the pons are generally attended by hyperpyrexia, and the urine is often abundant and may contain sugar or albumen. These symptoms are caused by irritation of the grey matter on the floor of the fourth ventricle.

Three cases have recently been described by Erb, in which, judging from the symptoms, the primary lesion was situated in the nerve nuclei of the pons and medulla oblongata. The affection began with pains in the head and neck, and attained its full development in the course of a few months. The chief symptoms consisted of ptosis, paresis, often associated with atrophy, of the muscles of mastication, tongue, and back of the neck. There was also weakness of the muscles supplied by the superior branches of the facial nerve, associated with phenomena of irritation in them, such as slight clonic spasms; but the muscles supplied by the inferior branches were unaffected. In one case, the movements of the eyeballs were deficient, but in the other two they were normal. Difficulty of deglutition was present in two of these cases; buzzing in the ears, and an abnormal galvanic reaction of the left acoustic nerve, were observed also in two cases; while great weakness of the extremities is mentioned as having been present in two, and slight weakness of the arms in the remaining case.

One of these cases terminated fatally, but a post-mortem examination was not obtained. Putting aside the weakness of the extremities, which was probably caused by implication of the pyramidal tracts, the other symptoms were evidently due to disease of the nerve nuclei of the pons and medulla oblongata, or of the fibres of the cranial nerves in their passage through these structures. It is interesting to observe that the bulbar nuclei or cranial nerves implicated in these cases are

those that regulate the actions of muscles which are frequently associated in their actions. The association of the actions of these muscles is better observed in animals than in man. Let us suppose that a dog, for instance, is lying in repose, with his eyes closed, and that a rabbit or other animal upon which he preys rushes past to his left. The noise made by the passing object is conducted to the brain of the dog mainly through the left ear, and instantly his eyelids open, the eyeballs and the head are rotated to the left, the mouth opens so as to prepare for closure of it upon the prey, and the tongue is also ready for protrusion. It would appear that some of the muscles supplied by branches of the cervical plexus were affected in these cases as well as those supplied by the spinal accessory nerve. I would suggest it as probable that the *fasciculus rotundus*, the functions of which are not at present known, is the medium of association between the mechanism in the upper end of the pons and crura cerebri which regulates the movements of the eyeballs, and the mechanism in the medulla oblongata and upper end of the spinal cord which regulates the movements of the neck and head.

Tumours of the Pons.—A slow-growing tumour may be situated in the centre of the pons, and attain the size of a hazel-nut, without giving rise to any symptoms.

As in other intracranial tumours, headache is a frequent symptom of tumour in this region. The headache is sometimes frontal, sometimes occipital, and at other times general and deep-seated, but it does not afford any indication of the situation of the growth.

Motor disturbances constitute the most characteristic features of tumours of the pons. General convulsions which are not unfrequently caused by lesions like hæmorrhage, which occur suddenly, are very rare in tumours, and probably never appear except towards the last few days of life or when the affection is complicated by meningitis. Even local spasms of the muscles of the extremities are rare in tumour, inasmuch as the fibres of the pyramidal tract are slowly subjected to pressure without previous irritation. Paralysis is, therefore, by far the most important motor symptom observed in tumour of the pons. When the tumour is situated in one lateral half of the

pons, especially in its posterior half, the paralysis assumes the form of *alternate hemiplegia*. When, for instance, a tumour is situated in the right half of the pons on a level with the nucleus

FIG. 276.

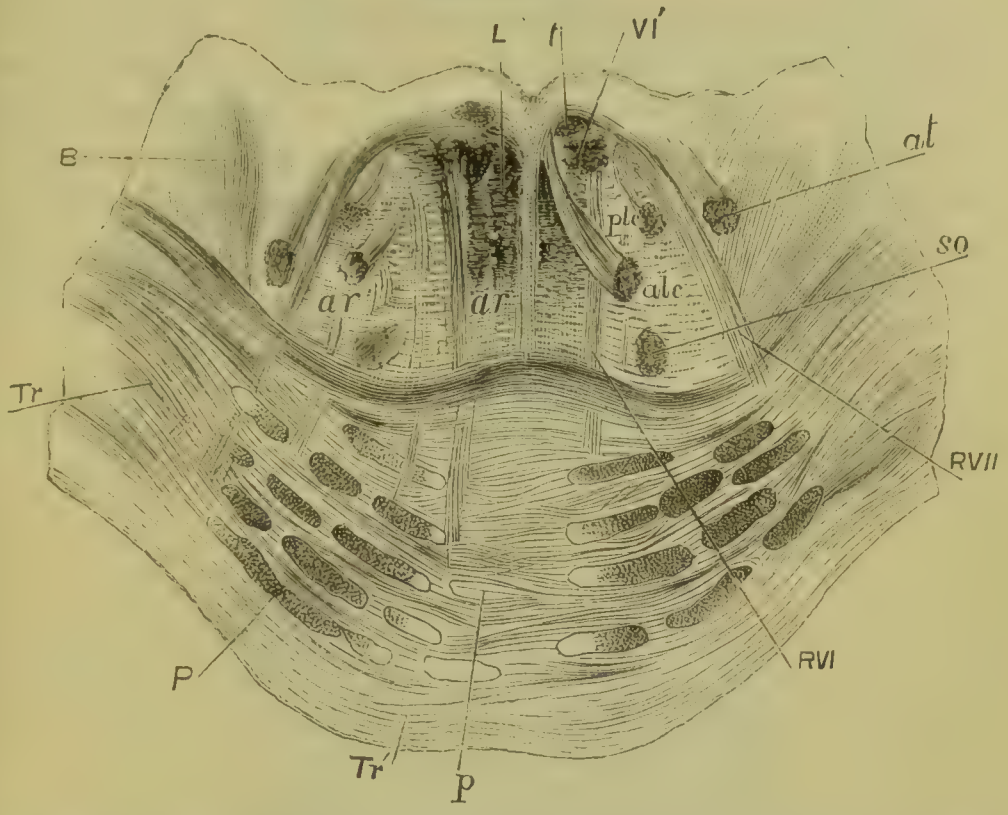


FIG. 276 (Modified from Erb). *Transverse Section of the Pons on a level with the Abducens and Facial Roots, from a nine months embryo.*—The right half represents a section made a little lower than the left. *P*, Pyramidal tract; *p*, accessory portion of the pyramidal tract; *Tr* and *Tr'*, transverse fibres of the pons; *so*, superior olivary body; *alc* and *plc*, anterior and posterior nuclei of the lateral column respectively, representing the nucleus of the facial nerve; RVII, root of the facial nerve; VI', nucleus of the sixth nerve; RVI, root of the sixth nerve; *at*, ascending root of the trigeminus. *B*, The internal division of the peduncle of the cerebellum as it passes from the cerebellum; *L*, posterior longitudinal fasciculus; *ar* and *ar'*, the upward continuation of the internal and external divisions of the anterior root-zone of the spinal cord; *t*, fasciculus teres.

of origin of the sixth and seventh nerves (*Fig.* 276), the extremities and half the tongue on the side opposite the lesion are paralysed from compression of the pyramidal tract before it has crossed, while the facial muscles, including those of the eyelid and eyebrow supplied by the seventh, and the external rectus are paralysed on the side of the lesion. Under these circumstances the facial paralysis is caused by compression of the fibres of the nerve or destruction of the facial nucleus, and the facial muscles often manifest the reaction of degeneration. In a case

of this kind under my own observation the reaction to both currents was diminished in the paralysed muscles, but the reaction of degeneration never appeared, so that this sign is not always trustworthy.

It is remarkable how seldom disturbances of the sense of hearing have been observed in tumours of the pons, weakness of hearing on the side of the lesion being mentioned a few times, but unilateral deafness has, so far as I know, never been described. Anæsthesia of the opposite half of the body and extremities has been observed only in about one-third of the reported cases (Ladame), and is never so prominent a symptom as the motor paralysis. If the tumour be situated in the right

FIG. 277.

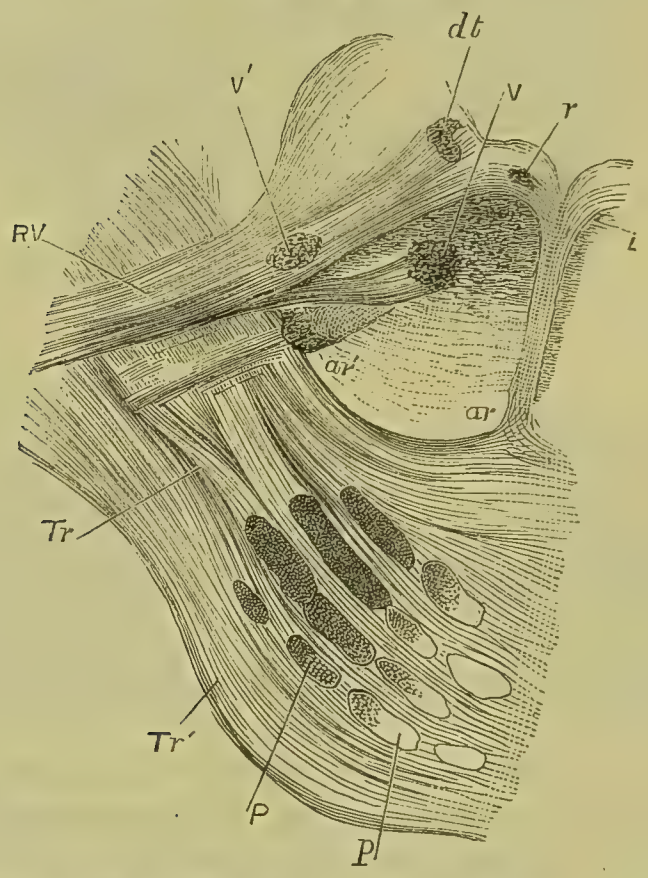


FIG. 277 (Modified from Erb). *Transverse Section of the Pons on a level with the origin of the Trigeminus, from a nine months human embryo.—P*, pyramidal tract; *p*, accessory portion of the pyramidal tract; *Tr*, *Tr'*, transverse fibres of the pons; *at*, ascending root of the trigeminus and gelatinous substance; *dt*, descending root of the trigeminus; *r*, root-fibres of the trigeminus cut transversely; V, motor nucleus of the trigeminus; V', middle sensory trigeminal nucleus; RV, root of trigeminus; *C*, roots of the fifth proceeding from the cerebellum; L, Posterior longitudinal fasciculus; *ar* and *ar'*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord.

half of the pons on a level with the fibres of origin of the fifth nerve (*Fig.* 277), and if it grow forwards so as to compress the pyramidal tract, the extremities and one-half the tongue are paralysed on the opposite side of the body, and the face may still be paralysed on the side of the lesion, either from compression of the fibres of the pyramidal tract belonging to the facial nucleus after they have crossed in the pons, or from

FIG. 278.

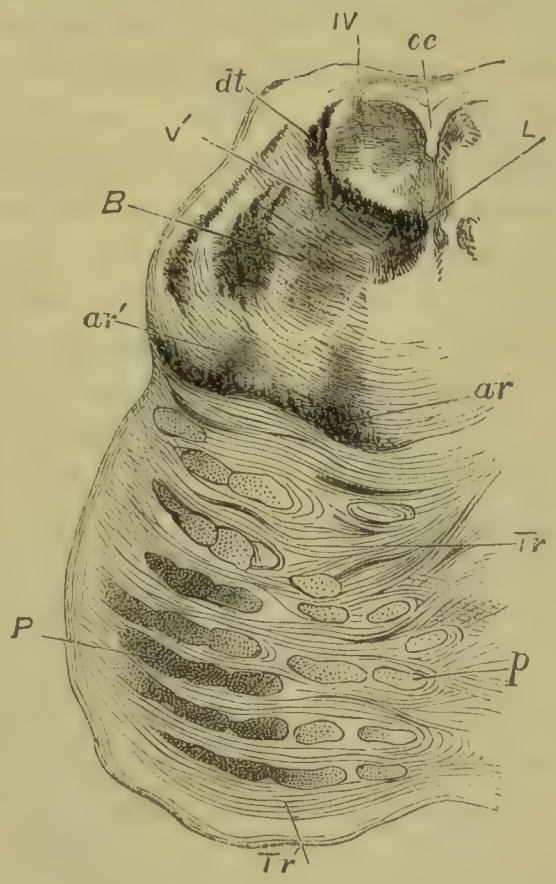


FIG. 278 (Modified from Meynert). *Transverse Section of the Pons on a level with the upper end of the Fourth Ventricle, from a nine months human embryo.*—*P*, pyramidal tract; *p*, accessory portion of the pyramidal tract; *Tr*, *Tr'*, transverse fibres of the pons; *B*, superior brachium of the pons; *L*, posterior longitudinal fasciculus; *ar* and *ar'*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord; V', middle sensory trigeminal nucleus; *dt*, descending root of the trigeminus; IV, nucleus of the fourth nerve; *cc*, aqueduct of Sylvius.

extension of the tumour downwards to reach the fibres of origin of the facial nerve. The masticatory muscles will also be paralysed on the side of the lesion, and various sensory and trophic disturbances will occur in the region of distribution of the fifth nerve, such as hyperæsthesia, neuralgic pains, anæsthesia often

assuming the form of anæsthesia dolorosa, and neuroparalytic ophthalmia. The taste of the corresponding half of the tongue is often abolished, while smell is impaired in the nostril of that side owing to loss of common sensibility. Hemi-anæsthesia of the opposite side may exist, and then the sensory disturbance presents an alternate distribution like the paralysis. When the tumour is situated in the middle of the pons from the first, or extends from one side to the other during its growth, all the extremities may be paralysed, either simultaneously or successively, the muscles of the tongue on both sides may be weakened, giving rise to difficulties of articulation (anarthria) and deglutition. There may be double facial paralysis, complete masticatory paralysis, paralysis of both the external recti muscles, various sensory and trophic disturbances in the region of distribution of the fifth nerves, and abolition of taste on both sides of the tongue. The distribution of the paralysis may present varieties other than those just described. Both sides of the face may be paralysed and the extremities on one side only, or the latter may be unaffected; on the other hand, only one side of the face may be affected and the extremities on both sides. Similar variations may occur with regard to the distribution of the sensory disturbances, although they are seldom so well marked. Disorders of motor co-ordination may be observed in lesions of the pons, especially tumours, similar to those which will be immediately described in connection with disease of the peduncles of the cerebellum.

Aneurism of the basilar artery does not appear to cause symptoms which enable us to distinguish it from a new formation pressing on the pons in the same situation. It is probable that unilateral or bilateral deafness is a more frequent symptom of aneurism than of solid growths.

Psychical disturbances are frequently observed in tumours of the pons, consisting of loss of memory, apathy, and stupor, all of them symptoms indicative of compression of the brain. These symptoms are not, however, directly caused by the affection of the pons, but by effusion into the ventricles of the brain, with which the affection of the pons is frequently complicated.

When the lesion implicates, either directly or indirectly, the pneumogastric nerves or their nuclei of origin, various disorders

of respiration and circulation may be present, but these usually belong to the terminal phenomena.

Albuminuria and glycosuria have been observed in local diseases of the pons, but by no means with exceptional frequency.

(ii.) Lesions in the Peduncles of the Cerebrum.

§ 761. The most characteristic features of lesions of the cerebral peduncle are afforded by an *alternate* hemiplegia, in which the extremities, half the face, and half the tongue are

Fig. 279.

Fig. 279 (Modified from Krause). *Transverse Section of the Crus Cerebri on a level with the anterior pair of Corpora Quadrigemina, from a nine months embryo.*—*cc*, crusta; *P*, pyramidal tract; *p*, accessory portion of the pyramidal tract; *ps*, sensory peduncular tract; *LN*, locus niger; *RN*, red nucleus of the tegmentum; *L*, posterior longitudinal fasciculus; *ar* and *ar'*, upward continuation of the anterior root-zone of the spinal cord; III, third nerve; III', nucleus of the third nerve; IV, fourth nerve; IV', nucleus of the fourth nerve; IV'', crossing of the fibres of the fourth nerves to opposite sides; *dt*, descending root of the trigeminus; *cc*, aqueduct of Sylvius; *x*, crossing of the fibres of the superior peduncles of the cerebellum; *pf*, fasciculus of medullated fibres proceeding to the anterior pair of corpora quadrigemina.

paralysed on the side opposite, and the oculo-motor nerve on the same side as the lesion. If the lesion implicate the sensory peduncular fibres (*Fig.* 279, *ps*), hemianæsthesia may be present on the side opposite the lesion, but the senses of smell and sight are not affected unless neighbouring parts be implicated, or there be secondary atrophy of the optic discs in the case of tumour. A localised lesion in the superior part of the crus may give rise to isolated paralysis of the fourth nerve. Tumours of the crura may paralyse the oculo-motor nerves on both sides. The form of alternate paralysis just described is only indicative of lesion of the crus cerebri, when the paralysis of the limbs and of the motor oculi occur simultaneously. It must be remembered that multiple lesions in syphilis, situated in different parts of the brain, are very liable to cause a grouping of symptoms closely simulating those produced by a single lesion in the crus cerebri.

b. Lesions in the Peduncles of the Cerebellum.

§ 762. One of the most remarkable examples of hæmorrhage into the middle peduncle of the cerebellum is a case described by Nonat:—

> A woman, about 60 years of age, had an apoplectic attack, characterised by unconsciousness, loss of general sensibility, and paralysis. The patient lay on her right side, with the head strongly rotated in the same direction. The eyeballs were immovable, the right being rotated downwards and outwards, and the left upwards and inwards. The patient died on the day following the onset of the attack. At the autopsy a fresh hæmorrhagic focus, about the size of a chestnut, was found situated in the right middle peduncle of the cerebellum, extending to some extent into the corresponding hemisphere. The remainder of the brain and the membranes were healthy.

Tumours of the middle peduncle of the cerebellum give rise to headache and dizziness, trifacial neuralgia, neuro-paralytic ophthalmia, and partial deafness on the side of the lesion, and disorders of motor co-ordination, the tendency to fall being in a lateral direction and towards the side of the lesion. If the tumour press forwards on the pons, then all the symptoms of a lesion in the pons itself may be present. Any of the bulbar nerves may then be implicated according to the position of the

lesion, while compression of the pyramidal tract and of the sensory fibres may cause hemiplegia and hemianæsthesia of the opposite side. Conjugate deviation of the eyes and rotation of the head may also under these circumstances take place, the deviation being directed away from the side of the lesion. According to Nothnagel, lesions of the superior and inferior peduncles of the cerebellum do not cause any characteristic symptoms. He also thinks that stationary lesions of the middle peduncle do not give rise to symptoms, and that even irritative lesions only produce them when the connection of the peduncle with the cerebellum is not completely severed.

c. Lesions in the Cerebellum.

§ 763. Hæmorrhage into the lateral lobe of the cerebellum may or may not occasion loss of consciousness, according to its extent. The symptoms usually consist of intense cephalalgia, generally situated in the occiput, but occasionally in the forehead ; and vomiting, the latter being probably the most constant of all the symptoms of cerebellar hæmorrhage. Hemiplegia is not unfrequently present, caused by pressure upon the pons, the paralysis being sometimes crossed, sometimes direct (Carion).

Spasmodic contractions of the masticatory, facial, or ocular muscles have occasionally been observed, and marked rigidity of the muscles of the neck is a more frequent symptom. Paralysis of the face is exceptional, but when present the orbicular muscle of the eyelid is implicated, and the paralysis is situated on the side of the lesion. Strabismus may occasionally be present owing to compression of one of the motor nerves of the eyeball. Conjugate deviation of the eyes has been observed, and it has always been directed away from the lesion. The movements of articulation and deglutition are rarely affected. The pupils are sometimes dilated, more frequently contracted, and occasionally insensible to light. In exceptional cases a certain degree of anæsthesia of the opposite half of the body has been observed. Blindness has sometimes been observed, as might have been expected from the proximity of the corpora quadrigemina to the cerebellum.

A large hæmorrhage into the middle lobe of the cerebellum may by pressing upon the pons and medulla oblongata cause

sudden apoplectiform symptoms, speedily terminating in death. In hæmorrhages of smaller size the symptoms are less marked or wanting. Hemiplegia is not so frequently present as in lesions of the lateral lobes, and in about one-third of the reported cases excitation of the genital functions is mentioned Large stationary lesions may occur in the cerebellum without giving rise to any recognisable symptoms during life.

Abscesses of the cerebellum have frequently been observed; the symptoms on the whole are like those caused by tumour.

Tumours in the cerebellum give rise, in addition to intense paroxysmal cephalalgia and vomiting, to characteristic motor disturbances. The most usual of these are a staggering gait, reeling, or a tendency to fall to one side. When the tumour is situated in the upper part of the middle lobe, the patient frequently manifests a tendency to fall backwards, while if it be situated in the inferior part of the same, it is probable that the tendency is to fall forwards or to revolve forwards round a horizontal axis. When the tumour is situated in one of the lateral lobes, the patient has a tendency to fall towards the side in which the tumour is situated. If the tumour be growing slowly, the tendency to fall to one side is counteracted to such an extent by cerebral action that the symptom is not readily elicited. A slight stagger may, however, be observed to one side when the patient is asked to turn round suddenly, and especially if the eyes be closed.

Symptoms of motor irritation are also observed in cases of tumour of the cerebellum. The most usual of these is a tonic contraction of the muscles of the neck, causing retraction of the head. This tonic contraction may extend to the muscles of the trunk and extremities, giving rise to tetanic seizures (Hughlings-Jackson). During these attacks the trunk is usually arched, the head retracted, and the various segments of the lower extremities extended upon one another and the trunk, so that the body rests upon the head and heels, as in tetanus. The various segments of the upper extremities are flexed upon one another, this being the position occupied by them in tetanus. The tonic contractions may be more pronounced on one side, and then the body may be arched towards that side.

Movements of the eyeballs are frequently observed in cases of

cerebellar tumour. These movements may be vertical, horizontal, or oblique, and are generally parallel (Mackenzie). Sometimes they are only observed during the convulsive attacks, but when there is permanent rigidity the eyes may be rotated in one direction and fixed, or present slight parallel oscillatory movements.

Tumours of the cerebellum are very liable to be complicated by effusion into the ventricles of the brain, due either to pressure on the venæ magnæ Galeni or to obliteration of the communication between the spinal and cerebral subarachnoidal spaces (S. Mackenzie). This effusion in children gives rise to enlargement of the head and distension of the fontanelles, similar to that occurring in chronic hydrocephalus. When the fontanelles have closed before effusion has taken place, the head is prevented from enlarging, but in these cases sudden death is very liable to occur from compression of the floor of the fourth ventricle, and consequent arrest of the function of the respiratory centre.

As already remarked, the most marked clinical characteristic of a tumour of the superior part of the middle lobe of the cerebellum is a tendency to fall backwards, or to rotate backwards round a horizontal axis. In a case under the care of Dr. Leech, in which this symptom was very marked, and in which the post-mortem examination was conducted by myself, a tubercular tumour, the size of a hen's egg, was found in the right occipital fossa, immediately under the tentorium and close to the falx cerebelli. In the case of a child, aged 4 years, under my own care, there was a tendency to fall diagonally backwards and to the right. I expected to find a tubercular tumour in the superior surface of the cerebellum, situated between the right lateral and middle lobes. Instead of that I found a tubercular tumour, the size of a pigeon's egg, situated under the tentorium in the occipital fossa to the left of the falx cerebelli, and a second tumour, about the same size, in the right middle peduncle. The tendency to fall in a diagonal direction was evidently the result of a composition of forces, the first tumour causing a tendency to fall backwards and the second a tendency to fall to the right.

The following case is an instance of tumour of the inferior

part of the middle lobe of the cerebellum, although the lesion was not limited to that region:—

John Thomas Gould, æt. 14 years, was admitted into the Royal Infirmary on March 5, 1877, under the care of Dr. W. Roberts, to whose kindness I am indebted for permission to publish the case. He was a strong and healthy boy until a few months ago; his parents were also healthy, and there was no family history of consumption or any other constitutional disease. He was a bricksetter by trade, and three months previously to admission fell from a ladder and struck the back of his head on the pavement, and since that time he has suffered from more or less constant occipital headache.

Condition on admission.—As he lies in bed he can move his legs freely in any direction; but on attempting to walk, the feet are alternately projected forwards, the heel coming down forcibly as in locomotor ataxy. He cannot maintain the erect posture unsupported; and when all external aid is momentarily withdrawn, his head shoots downwards and forwards, as if the body were about to revolve round a transverse horizontal axis. When the patient is caught in the act of falling and raised again to the erect posture, he complains of dizziness, and sees objects revolving from right to left. On directing his eyes to the right, a slight oscillatory movement of the eyeballs is observed, but there is no nystagmus when he looks straight in front. The patient is almost quite blind in the left eye, but can distinguish objects clearly with the right eye.

When the right eye is fixed on an object, such as a finger twelve inches in front, a second finger moved laterally and to the right is seen until it is almost nine inches from the first, showing that the field of vision to the right is not sensibly diminished. But starting again from the finger on which the eye is fixed, and moving the second laterally to the left, the latter disappears from view when it is from one to two inches removed from the former. I say from one to two inches, because his replies were not always the same, thus indicating that the sensitive and blind portions of the retina are not separated from one another by a sharp and defined border, but fade insensibly into each other. The pupils are equal, both being dilated and very sluggish to light. An ophthalmoscopic examination reveals double optic neuritis with swollen disc, but there is no atrophy. The other special senses and the mental faculties are unaffected, the urine is free from sugar or albumen, the appetite is good, and all the other functions of life are normal. He was ordered iod. potass., grs. x., to be taken three times a-day. On March 31, four weeks after admission, it is evident that the symptoms have altered considerably. The patient now habitually lies on his back, or slightly inclined to one side. He cannot sit erect without support, but he has still some degree of voluntary control over his legs, although the movements are feeble. Cutaneous sensibility is impaired in the lower extremities; he can still feel when touched, but he cannot localise the touch well. Sensibility to variations of temperature

and to pain is also impaired. There is complete blindness of both eyes, but there is only slight atrophy of the optic discs. Two days ago he passed his urine in bed for the first time. His bowels are very constipated, and there is general emaciation.

April 15th.—He complains much of frontal headache. There is complete anæsthesia, and entire loss of voluntary motion of the lower extremities; the stools and urine are passed under him, and there is a large bed-sore over the sacrum. He cannot hear the ticking of a watch so well with the left as with the right ear.

May 14th.—There is complete anæsthesia of all parts below a line passing round the body on a level with the anterior superior processes of the ilium. Reflex irritability is entirely abolished in the lower extremities, as tested by tickling, pricking, and the faradic current. The muscles of the legs and thighs do not react to either the faradic or constant currents. The calf of the right leg measures $9\frac{1}{4}$ and that of the left only $7\frac{1}{4}$ inches. Each thigh measures $9\frac{3}{4}$ inches. The muscles of the thighs and of the right leg appear only to be emaciated in proportion to the rest of the body; but it is manifest that the muscles of the left leg are specially atrophied. There are deep bed-sores over the sacrum, the prominences of both thighs, the external maleoli of both ankles, and the inside of the left knee. The intelligence is greatly blunted, and he lies in a half stupor, but replies readily to any simple question asked him. His pulse is very feeble and beats about 140 in a minute. His appetite continues remarkably good. About a week ago it was noticed that the left eyelid was only half closed when he was asleep, and he could not close it entirely by a voluntary effort. There was also slight paralysis of the left facial muscles, so slight that the difference between the two sides could scarcely be detected when the face was quiescent, but recognisable when the patient smiled. This affection of the facial nerve only lasted a few days, and has now entirely disappeared. The condition of his hearing cannot be satisfactorily tested, owing to the apathetic state of his intelligence.

From this time he lingered on without any further special symptom manifesting itself. He became more and more apathetic and extremely emaciated, and died on July 3rd, four months after entering the Infirmary, and seven months after the fall, to which in all probability the origin of the disease may be traced.

Sectio cadaveris.—Twenty-four hours after death rigor mortis is moderately well established in both extremities. The body is greatly emaciated. The calves of the legs each measure 7 inches, and thighs $7\frac{1}{4}$ inches. The sacrum, both trochanters, and the external malleolus of the left foot are exposed and denuded in consequence of extensive bed-sores. The tips of both ears are also ulcerated, as well as the inside of the left knee.

On removing the calvarium the brain appeared to project, and the convolutions were flattened. The sinuses and the veins on the surface of the brain were gorged with blood. The substance of the brain was somewhat soft, and the ventricles were distended with fluid; but the cerebrum was

healthy in other respects. Some grumous turbid fluid escaped from between the cerebellum and corpora quadrigemina. On inspecting the cerebellum the edge of a tumour was noticed between the inferior surface of the middle lobe at its posterior margin and the superior surface of the medulla oblongata; while the anterior end of the tumour could be seen between the cerebellum and corpora quadrigemina. On making a vertical section of the cerebellum in the middle line down to the floor of the fourth ventricle, the tumour was seen to occupy the whole of the anterior part of the middle lobe of the cerebellum, being somewhat more developed on the right than on the left side. In consistence the tumour was soft, of a greyish-red colour, the centre being broken down so as to form a small cavity from which the turbid fluid already mentioned had escaped. The tumour was not circumscribed, its margins gradually blending with the surrounding nervous tissue. The growth passed along the right superior peduncle of the cerebellum to reach the corpora quadrigemina, and the latter were somewhat softened and flattened. On opening the spinal canal the cord was seen to occupy the whole of the cavity transversely, the diameter of the cord being about one-third larger than that in health. The whole of the cord felt brawny, like bacon. On making transverse sections, from above downwards, half an inch apart, the membranes in the cervical region were seen to be adherent, slightly thickened, and the cord was softened; but in the upper dorsal region the spinal pia mater, especially on the posterior aspect, was three times as thick as in the healthy cord. In the middle of the dorsal region the thickened membrane had developed into a dense well-defined tumour, which pressed on the cord from behind forwards, so that only a small part of the anterior segment of the cord was left. In the lumbar region, again, the tumour surrounded the cord, so that a central core, about the size of a goose quill, of softened nervous tissue was all that was left to represent the spinal cord. The tumour was the colour and texture of bacon, and much denser than that of the cerebellum.

The left lung was adherent to the chest walls. Both lungs were healthy. The heart was normal. The abdominal organs were healthy. Microscopic examination showed that the tumour consisted of small delicate cells imbedded in a finely granular substance. The growth in the cord presented similar microscopic characters to the cerebellar tumour; but contained a larger amount of intercellular substance.

The first time I examined the patient my diagnosis was "tumour, probably a glioma, situated in the inferior portion of the middle lobe of the cerebellum, and pressing forwards on the corpora quadrigemina." The reasons for regarding the case as one of intracranial tumour are so manifest as scarcely to require mention. They are the history of an injury to the head, the gradual development and progressive character of the

symptoms, the constant headache, and the existence of double optic neuritis. My reason for believing that the tumour was situated in the inferior portion of the middle lobe of the cerebellum was the remarkable manner in which the head and shoulders shot forwards and downwards, as if the patient were about to revolve round a horizontal axis. The circumstances in favour of the tumour being a glioma were that, if the diagnosis with respect to the localisation was correct, it probably grew in the substance of the nervous tissue itself, and not from the membranes; while the appearance of the patient and the family history were against tubercle; there was no evidence of congenital syphilis; and the age of the patient put cancer almost out of the question.

My reasons for thinking that the tumour pressed forwards upon the corpora quadrigemina were that I thought this would to some extent explain the excessive staggering present during attempts at walking; but much more that it would explain the great impairment of vision present at such an early stage of the disease. But the peculiar character of the disorder of vision demands an explanation; and for this purpose let us take Charcot's scheme of the decussation of the optic tracts as our guide.

In this case, vision was almost totally lost in the left eye, and the state of vision of the right eye simulated nasal hemiopia. I say simulated, because it was not a case of hemiopia at all. The condition of vision in both eyes was that of amblyopia in its progress towards amaurosis. The amaurotic condition was nearly reached in both sides of the retina in the left eye; but in the right eye the right half of the retina had become amaurotic, while vision was tolerably good in the left half; and between the halves of the right retina there was a relatively broad border land, where the comparatively good vision of one side faded gradually into the blindness of the other. Such a condition as this could not, therefore, have been caused by the pressure of a tumour on the commissure or optic tracts; it was not likely to have arisen directly from the optic neuritis, inasmuch as there was no atrophy of the disc; and it must therefore have been caused by a lesion interfering with the optic fibres, either directly or indirectly beyond the termination of the optic tracts in the external geniculate bodies.

It appeared to me very probable that a tumour pressing on the right side of the corpora quadrigemina, and extending gradually to the left, would produce the condition of vision met with in this case. The whirling of objects from right to left which the patient described showed that there was a greater amount of irritation of the right than of the left lobe of the cerebellum, and this rendered it probable that the tumour of the middle lobe extended farther to the right than to the left.

Suppose, then, that a tumour is pressing on the corpora quadrigemina from behind forwards, and from right to left, the fibres (*b a*) coming from the left eye, and meeting at LOD, would be first interfered with, then the fibres (*a'*) coming from the right half of the retina of the right eye would be intercepted in their passage behind the corpora geniculata to the opposite side. The fibres *b'* coming from the left half of the retina of the right eye would be the last to be injured, so that the condition of vision which was present in this case would be produced. It may be urged that Charcot's scheme of decussation is merely diagrammatic, and that the points LOG and LOD are

FIG. 280.

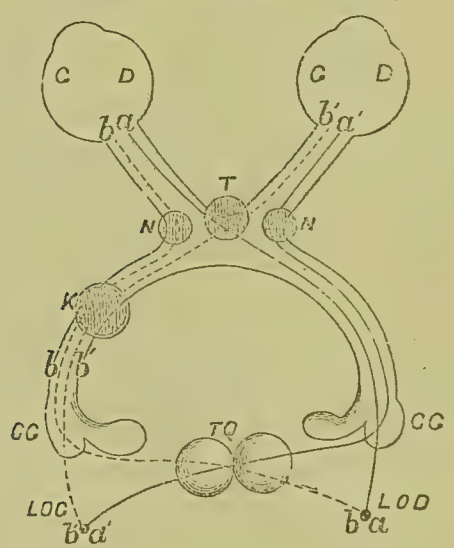


FIG. 280 (After Charcot). *Diagram of Decussation of the Optic Tracts.*—T, Semi-decussation in the chiasma; TQ, Decussation of fibres posterior to the external geniculate bodies (CG); *a' b*, Fibres which do not decussate in the chiasma; *b' a'*, Fibres coming from the right eye, and coming together in the left hemisphere (LOG); K, Lesion of the left optic tract producing right lateral hemianopsia; A, lesion in the left hemisphere (LOG), produces crossed amblyopia (right eye). T, Lesion producing temporal hemianopsia; N N, Lesion producing nasal hemianopsia.

supposed to represent positions in the cortex of the hemispheres; but my reply must be that I am only making a diagrammatic use of it. If there is a semi-decussation of the optic nerves in the chiasma, and if the fibres which do not cross in that place decussate behind the corpora geniculata, then, whatever may be the further course of these fibres, some such effect as that indicated would be produced by a tumour pressing from behind forwards, and from right to left on the corpora quadrigemina. This at least was the process of reasoning by which I came to the conclusion that the case was one of tumour of the anterior part of the middle lobe of the cerebellum, inclining to the right side, and pressing forwards on the corpora quadrigemina, and this conclusion was verified to a considerable extent by the post-mortem. One serious objection I always had to this view was, that it was not manifest how the floor of the fourth ventricle could escape under such circumstances; and yet there was no sugar in the urine, no polyuria, and the breathing was not interfered with. The autopsy explained this. It showed that the corpora quadrigemina were probably not so much interfered with by pressure as by extension of the glioma along the superior peduncle of the cerebellum into the substance of these bodies.

As the case progressed, it became evident that there was an independent affection of the cord, as evinced by the complete anæsthesia, and loss of reflex irritability in the lower extremities, as well as by the trophic changes already described.

Two suppositions could be made with regard to the affection of the cord. Either that there was tumour pressing on the cord of the same nature as that in the cerebellum, or that there was extensive softening in the lumbar region. I must acknowledge that I felt inclined to adopt the latter view, inasmuch as I was only thinking of a circumscribed growth, and was not prepared to find a new formation extending the whole length of the cord. It is very probable that the new growth had begun to develop in the spinal cord at the time the patient was admitted into the Infirmary, and that the symptoms of motor inco-ordination observed were due, in part at least, to implication of the posterior root-zones in the morbid process.

In the following case several tumours were found in the cere-

brum as well as in the cerebellum, yet it was not difficult to diagnosticate the presence of a tumour in the latter :—

Louis Ikin, æt. 3½ years, entered the Southern Hospital on October 3rd, 1877. His mother stated that he was always healthy until 14 months ago, when he had an attack of chicken-pox, after which he suffered from sore eyes. Soon afterwards he began to put his hand to his forehead and to complain of pain there, and he gradually lost flesh. There was also some discharge from the right ear. These symptoms continued for about eight months without any appreciable change; but six months before admission the mother was awakened during the night by a loud scream from the child, who was found on the floor, having been apparently projected from his bed by the violence of a convulsion. On being picked up he was found completely paralysed on the right half of the body, and aphasic.

The paralytic symptoms gradually improved, but convulsions supervened, the spasms being limited to the paretic side, and not attended by loss of consciousness. He had had the last of those attacks a fortnight previous to admission. The convulsive movements always began in the right hand and arm; but the mother could not be sure whether the right side of the face or the leg was next invaded. The parents had already lost one of their children from "water on the brain;" and another, who had reached the age of eight years, had never been able to speak more than a few words (congenital aphasia).

Present Condition. — There is a slight degree of right-sided facial paralysis, only apparent when the child cries or smiles. There is also paresis of the right arm and leg; but he has considerable voluntary power over both. The fingers of the right hand are semi-flexed, and the thumb bent inwards on the palm under the fingers. There is some muscular rigidity on attempting passive movements of the fingers, hand, and forearm, and there is also some degree of rigidity on attempting to move the right leg and foot. The child, on being placed on his feet, can stand and even walk a few steps if the upper part of the body be supported, but when every support is withdrawn his face assumes a frightened expression, and he would immediately fall on the paralysed side unless prevented. The act of falling does not consist of a simple yielding of the paretic leg, but the head and upper part of the trunk shoot laterally to the right, while the leg of the same side is maintained extended.

His sight is not good. He sees an object held out before him, but on putting his left hand out to grasp it he has to grope for it. There is double optic neuritis with commencing atrophy of the discs. His speech is almost lost, the only word he can say being "Mamma."

November 10th.—About ten days ago he had a convulsion which was mainly limited to the left side, this being the first observed since he entered the hospital. He has had as many as three attacks in a day, while some days passed without his having any attack. Altogether he has

had about twenty attacks in the ten days. I was not fortunate enough to see one of these attacks myself, but I gave particular instructions to the nurse to observe whether the convulsion began in the hand, face, or leg. The description of the nurse was always to the same effect, that the attack began with a scream, that the body was bent like a bow, so that the left ankle and left side of the face nearly met, and that immediately after the attack the child resumed its usual manner, without manifesting any tendency to sleep. There is no distinct paralysis of the left extremities.

December 12th.—The child has only had a few convulsive attacks since the last report, and none at all during the last fortnight. He is now nearly blind, being able only to see an object placed to the left of the left eye. There is slight nystagmus. The muscular rigidity on attempting passive movements of the right arm and leg is now more marked, and the paralysis on that side is also more pronounced. The lower extremities are congested, of a blue colour, and cold, but there is no muscular atrophy. The child eats his food well, and there is a fair amount of subcutaneous fat. He is becoming apathetic and passes his water and stools under him.

January 28th.—He now lies on his back, and is getting more and more somnolent and apathetic. The nystagmus is more pronounced and there is conjugate deviation of the eyes to the left. He screams at night and disturbs the other children in the ward, on account of which he was discharged.

I continued to visit the patient occasionally at his home, but the only symptoms of note observed were those of a gradual compression of the brain, enlargement of the head, separation of the fontanelles, and fluctuation over them. He died on March 13th, 1878.

The post mortem was conducted 24 hours after death. The ventricles were much distended by a fluid effusion. Four tubercular tumours, each about the size of a hazel-nut, were found lying along the sulcus of Rolando of the left hemisphere, one of them being situated at its inferior extremity close to the posterior extremity of the third frontal convolution. Another tumour, also about the size of a hazel-nut, was found in the cortex of the right hemisphere near the superior extremity of the ascending parietal convolution. A tumour, about the size of a pigeon's egg, was situated in the left lenticular nucleus, and compressing the internal capsule. The inferior surfaces of the right and middle lobes of the cerebellum were occupied by a tubercular mass, which extended also into the left lateral lobe. A microscopical examination of the spinal cord revealed sclerosis of the right lateral column.

The first time I examined this child the presence of double optic neuritis rendered it clear that the case was one of intracranial tumour, the slight stagger to the right and the purulent discharge from the right ear pointed to a tubercular tumour of

the right lobe of the cerebellum; while the history of unilateral convulsions beginning in the right arm and the aphasia pointed to the presence of one or more tubercular tumours along the sulcus of Rolando. My diagnosis, therefore, was tubercular tumour situated in the sulcus of Rolando of the left hemisphere, and another in the right lobe of the cerebellum. As the case progressed the hemiplegia became so complete that a cortical tumour would hardly be sufficient to account for it, and I consequently assumed the existence of another tumour in the left lenticular nucleus, and compressing the internal capsule. Had I adhered to this diagnosis it would have been absolutely accurate up to a certain point. But when the convulsions began in the left half of the body I began to waver in my previous opinion with regard to the localisation of the tumours. The unilateral convulsions of the left half of the body were either due to irritation of the motor area of the cortex of the right hemisphere, or to irritation of the cerebellum. From the uniform description of the nurse I came to the conclusion that these convulsions were of the nature of tetanic seizures, and therefore due to cerebellar irritation. The unilateral character assumed by them I explained by supposing that the tumour was growing in the left lobe of the cerebellum and causing irritation of the left middle peduncle, but it is of course doubtful whether there is any justification for such a supposition. I also thought that the presence of one tumour, situated in the centrum ovale of the left hemisphere, in such a position as to interrupt the fibres of the pyramidal tract and the fibres of the corpus callosum connecting the posterior extremities of the third frontal convolutions with one another, might account for the right hemiplegia and aphasia, without assuming the existence of a tumour in the cortex and another in the lenticular nucleus. I made a communication to the Manchester Medical Society, several weeks before the death of the patient, in which these various opinions were discussed, and, owing to the reluctance I felt in assuming the existence of five or six tumours situated in various parts of the cerebral hemispheres and cerebellum, I came to the conclusion that a tumour in the centrum ovale of the left cerebral hemisphere and another in the left lobe of the cerebellum might account for the symptoms. Had I assumed

the existence of the larger number of tumours my diagnosis would have been almost absolutely correct. I am even now unable to decide whether the unilateral spasms of the left half of the body were of the nature of tetanic seizures or were true cerebral convulsions caused by the irritation of the tumour found in the cortex of the right hemisphere, near the superior extremity of the ascending parietal convolution.

In the following case the symptoms pointing to an intracranial lesion were very obscure, yet the presence of double optic neuritis and a slight stagger in the gait of the patient rendered it possible to diagnosticate a tumour of the right lobe of the cerebellum. For the notes of the case I am indebted to Mr. Luckman :—

Annie E. M——, æt. 21 years, domestic servant, entered the Manchester Royal Infirmary on October 21st, 1880, under the care of Dr. Ross. The patient was healthy until about six months ago, when she began to suffer from a dull headache, occupying the vertex, and extending to either temple. The headache did not prevent her from sleeping, it was worse, as a rule, on getting up in the morning, and generally improved after she had had a warm cup of tea. The headache was liable to intense paroxysmal aggravation, and during these attacks the patient generally vomited. About two months ago she felt a little unsteadiness in walking, the headaches increased in intensity, and she suffered so much from retching and vomiting that she was compelled to give up her situation.

On presenting herself at the Infirmary as an out-patient, a week ago, the only symptoms complained of were intense headache, while there were great emotional disturbances like those of hysteria. As the patient walked across the floor a slight staggering towards the right side was observed, this being especially marked when she turned suddenly round or closed her eyes. It was not then convenient to make an ophthalmoscopic examination. When she appeared at the end of a week as an out-patient the symptoms were still the same, only the hysterical symptoms were decidedly more pronounced, and she was admitted as an in-patient.

Present Condition.—On admission an ophthalmoscopic examination revealed double optic neuritis, but she could read the smallest print. She never had diplopia, and there was no strabismus or nystagmus. There was no paralysis, no cutaneous sensory disturbances, and no affection of the special senses. The patient still suffered from headache, characterised by remissions and paroxysmal exacerbations, as well as from attacks of hiccough. In walking a slight stagger is occasionally observed, the tendency to fall being always towards the right. She also progressed in a slightly curved line, instead of walking in a straight course. The tendency to stagger is increased when the patient closes her eyes or turns suddenly round.

November 2nd.—No new symptoms were observed since last report until 1 o'clock to-day, when it was noticed that the patient's face and lips had become livid; she also complained of a dull, heavy headache seated on the vertex of the head. At 3 p.m. respiration suddenly ceased, and Dr. Steele, who was immediately sent for, resorted to artificial respiration, and maintained the action of the heart for upwards of twenty minutes, but the pulse ceased to beat four minutes after the artificial respiration was discontinued. During this time it was noticed that the right pupil was dilated and the right side of the face slightly paralysed.

The post-mortem examination was conducted by Dr. A. H. Young, eighteen hours after death. The cerebral hemispheres and basal ganglia were normal. The ventricles were distended with fluid, and the aqueduct of Sylvius was considerably dilated. In the cerebellum a well-defined tumour was found, forming a well-marked projection in the right lateral lobe. The cerebellar substance appeared prominent in the region of the foramen magnum, as though pushed out, and seemed to press upon the floor of the fourth ventricle.

The following case illustrates the movements of the eyeballs sometimes observed in tumours of the cerebellum:—

In the case of a boy, aged 12 years, under my care at the Southern Hospital, the symptoms on admission were headache, double optic neuritis, amblyopia, and a slight stagger on walking, the tendency to fall not being greater to one side than to the other. He lived in the hospital nearly three months, and during that time the optic neuritis gave place to atrophy of the discs, and the amblyopia to amaurosis; while the change in the other symptoms consisted of a progressive impairment of the mental faculties from gradual compression of the brain, the patient ultimately dying comatose. During the last three days of life there was rigidity of the muscles of the back of the neck along with vertical and parallel movements of the eyeballs, consisting of an upward alternating with a downward rotation. The time occupied by each rotatory movement was remarkably uniform, and as many as twenty of these occurred in a minute. At the autopsy two tubercular tumours, each about the size of a pigeon's egg, were found symmetrically situated in the inferior surfaces of the lateral lobes. A third tumour, about the size of a hazel-nut, was found in the superior portion of the middle lobe.

CHAPTER X.

II. DIFFUSED DISEASES OF THE ENCEPHALON.

(I.) ANÆMIA AND HYPERÆMIA OF THE BRAIN.

(i.) Anæmia of the Brain.

§ 764. *History.*—Dr. Marshall Hall was one of the first to direct attention to the symptoms produced by cerebral anæmia, and as these symptoms in children closely simulate those of acute hydrocephalus he proposed to call the condition hydrocephaloid or hydrencephaloid disease. He also pointed out that symptoms which had hitherto been attributed to cerebral hyperæmia were really due to anæmia, and were such as frequently occurred after exhausting hæmorrhages. About the same time Abercrombie gave a similar interpretation to the symptoms of syncope and of *apoplexia ex inanitione.* The study of the effects of ligature of the carotids by Sir Astley Cooper led the way in the experimental investigation of cerebral anæmia, which was completed in more recent years by the labours of Schiff, Kussmaul and Tenner, and many others.

§ 765. *Experimental Investigation.*—When one of the common carotid arteries is compressed, there is first indistinctness of vision, and after a few seconds a prickling sensation is felt in half the face, followed by a similar sensation in the limbs and opposite half of the body. General sensibility becomes indistinct, the sense of touch is impaired, and even trembling and convulsive twitchings may occur; but after three or four minutes these symptoms disappear, because the collateral circulation soon compensates the effects of the compression. Compression of both carotids is followed by indistinctness of vision amounting to almost complete blindness, contraction followed by dilatation of the pupils, the respiration becomes slow, deep, and sighing, and there is a sense of oppression about the thorax. These symptoms are followed by drowsiness, staggering, and loss of consciousness; and if the compression be continued, universal muscular twitching, symptoms of choking, and vomiting appear (Schiff).

If the circulation through both carotid and vertebral arteries be suddenly interrupted, the pupils first contract, but soon dilate again, the eye-

balls roll upwards and outwards, the jaws are clenched, and the respiration, at first short, becomes slow and deep; these symptoms are soon followed by general muscular relaxation, loss of consciousness, and general convulsions. Similar symptoms attend bleeding to death. If the animal be previously enfeebled by loss of blood, death results from syncope without convulsions. The animal may be kept for a short time in a condition simulating death; but if artificial respiration be maintained, gradual recovery takes place when the blood is allowed to flow again through the vertebral arteries.

It has been found that obstruction of the circulation through the carotids in animals only causes trifling effects in comparison with the symptoms produced in man, showing that the anterior lobes are of much greater importance in man than in animals.

§ 766. *Etiology.*—Anæmia of the brain is caused by influences which act upon the vascular system of the brain alone, or it may form only a part of general anæmia. The entire brain may be affected, causing *universal anæmia,* or it may be limited to certain parts causing *partial anæmia.* The symptoms also differ much according as the anæmia is suddenly or gradually produced.

Acute universal cerebral anæmia is caused in its most typical form by a sudden loss of a large quantity of blood. The most frequent causes of this form of cerebral anæmia are *post partum* hæmorrhage, hæmorrhages from the nose, lungs, stomach, and intestines, and large losses of blood from external injuries. A sudden fall of arterial tension from the relaxation of large vascular areas in other parts of the body may also cause cerebral anæmia. The faintness which frequently accompanies the rapid withdrawal of ascitic fluid, or immediately after parturition, is probably caused by the flow of a large quantity of blood into the relaxed abdominal vessels.

Cerebral anæmia is a frequent accompaniment of organic diseases of the heart, more especially of aortic regurgitation, in which death often results from syncope. Weakness of the muscular walls of the heart, whether temporary as after acute febrile diseases, or permanent as in fatty degeneration, is apt to produce faintness from cerebral anæmia. Irritation of the vagus may cause cerebral anæmia by a temporary arrest of the heart's action. Fainting, the result of mental impressions, may

be produced in this way, although spasm of the cerebral vessels from irritation of the sympathetic nerves may be the cause.

Intense pain may cause faintness or even syncope, which may result from reflex irritation of the vagus, or direct irritation of the sympathetic. But the direct effect produced on the nerve-centres must also contribute to the result. The strong nervous discharges caused by the external injury ascend along centripetal fibres and produce a corresponding strong nervous discharge from the higher nerve-centres, which is conducted along centrifugal fibres to the periphery, giving rise to the cries and various bodily contortions which indicate pain. Again, strong nervous discharges from the higher centres must be followed by exhaustion and consequent impairment or abolition of function, just as the discharges of epilepsy are accompanied by unconsciousness.

Acute universal cerebral anæmia may probably be caused by various poisons, although anæsthetic agents, like chloroform and ether, probably act less upon the circulation of the brain than upon the cerebral tissues themselves.

Chronic Universal Cerebral Anæmia.—This variety is caused by any condition which withdraws a large quantity of the nutrient fluids from the body, such as repeated losses of blood, severe diarrhœa, chronic suppuration, and all causes of general anæmia. Chronic anæmia of the brain also occurs in certain valvular lesions and fatty degeneration of the heart. The introduction of foreign matter into the cavity of the skull, as inflammatory exudations in meningitis, the fluid which transudes in œdema, hæmorrhagic foci, and cerebral tumours, may also cause chronic anæmia of the brain.

Partial Cerebral Anæmia.—Partial anæmia is caused when one of the vessels of the brain is obstructed, or when external pressure is exerted by a tumour on a vascular area, but these cases have already been described. Unilateral anæmia is seen after ligature of the carotid on one side; but the symptoms are only temporary, except in the cases in which there is an impervious condition either congenital or acquired of the communicating arteries of the circle of Willis.

§ 767. *Symptoms.*

Acute Universal Cerebral Anæmia.—The initial symptoms are obscuration of the senses, buzzing in the ears, dizziness, contraction followed by dilatation of the pupils, imperfect reaction to external stimuli, and loss of consciousness. The surface becomes cold and pale, the respiratory movements, accelerated at first, become slow, and this condition is frequently followed by general convulsions and coma. The symptoms which are produced by the temporary anæmia caused by powerful mental impressions differ considerably from those just described. At first there is some degree of mental incoherence manifested by the inability of the patient to direct his attention to a particular object, a feeling of oppression in the chest, along with a tendency to gape. The face becomes pale, a cold perspiration breaks out on the forehead and sometimes on the entire body, and there is general muscular relaxation. There is ringing in the ears, dimness of sight, nausea, and sometimes vomiting. The pulse is small, compressible, but regular. The patient may now begin to recover or fall insensible to the ground, and after a few moments in the recumbent position he begins to recover. This constitutes an ordinary *fainting-fit* or syncope.

Chronic Universal Cerebral Anæmia.—In these cases well-marked *mental irritability* is observed, associated with fretfulness, restlessness, uneasy sleep disturbed by dreams, and a certain amount of intolerance of light and sound. These symptoms are frequently succeeded by the phenomena of depression, and sometimes the latter predominate from the first. The patient suffers from almost constant headache, vertigo, nausea, and faintness. The pulse is small and compressible, the cardiac impulse feeble, and there is great disinclination for either mental or physical exertion.

In the severe forms of chronic or sub-acute cerebral anæmia, such as that produced by starvation, or that which arises during the course of exhausting fevers, delirium becomes a prominent symptom. Occasionally delirium comes on after hæmorrhage; but it is generally a late symptom, and occurs more frequently with robust than feeble people. This symptom is more common when the anæmia is due to starvation, and under these circumstances it is called the "delirium of inani-

tion." The delirium which comes on after the crisis or during convalescence in febrile diseases is also to be attributed in great part to defective nutrition of the brain. During the delirium of cerebral anæmia the patients are excited and sometimes maniacal; there are illusions of sight and hearing, and delusions of persecution. The duration of this condition is variable; it may last a few hours or days only, but it sometimes continues for weeks and occasionally passes into permanent insanity.

Cerebral anæmia is seen in infants after severe diarrhœa, or other exhausting disease; and as this is the form which was called by Marshall Hall *hydrocephaloid* or *hydrencephaloid* disease, it demands special notice. The affection may be divided into two stages—the first, that of irritability; the second, that of torpor, resembling the first and second stages of hydrocephalus respectively. In the first stage, the infant is irritable, restless, with flushed face, warm skin, and frequent pulse; the patient starts on being touched or on hearing any sudden noise, sleep is disturbed and interrupted by sighs, moans, or screams. During the second stage the countenance becomes pale, the cheeks and extremities cold, the eyelids are half closed, the eyes sunk in their sockets, there is frequently slight strabismus, and the pupils are dilated and do not contract to light. The breathing is irregular and sighing, the voice husky, and there is sometimes a teasing cough with rattling in the throat. A most important symptom which distinguishes this disease from hydrocephalus is that the fontanelle, instead of being tense as in the latter disease, is depressed. The child inclines almost constantly to fall into a sleep, which may pass into coma and death, but under appropriate treatment gradual recovery usually takes place.

§ 768. *Morbid Anatomy.*—The blood-vessels of the membranes of the brain are usually more or less empty, but there is almost always a certain quantity in the larger veins and sinuses. A very characteristic appearance is presented by the pia mater in cases of chronic anæmia associated with conditions which induce general œdema. The pia mater, especially over the superior surface of the hemispheres, is of a pale colour,

somewhat opaque, and so œdematous that it pits on pressure. This condition is especially marked in chronic Bright's disease, and I have frequently been able to predict on opening the skull at a post-mortem that we should find contracted kidneys. In these cases the cavities of the arachnoid and the lateral ventricles contain together about two ounces of serous fluid, and the choroid plexuses are œdematous. At the junction of the posterior and descending horns of the ventricle the œdema of the choroid plexuses is so great that it gives the appearance of two or three cysts, each about the size of a pea, growing from them. The grey substance is pale, and somewhat decolourised. The white substance is paler than usual, and there is an absence of blood-points.

§ 769. *Morbid Physiology.*—Cerebral anæmia is a complex condition, depending not merely upon a deficiency of the quantity of blood supplied to the brain, but also upon a change in its quality, and upon a diminution in the intracranial pressure. It is exceedingly difficult to apportion to each of these their due share in the production of the symptoms. A glance, however, at the empirical laws of nerve irritability will afford the key to the interpretation of the more prominent symptoms of the disease. When a nerve is imperfectly nourished its irritability is first increased; or, in other words, a slight degree of its usual stimulus will cause it to discharge its energy. When the deficiency of nutrition is continued the increase of the irritability, which is only a temporary condition, is followed by a decrease, and complete withdrawal of nourishment again is followed by exhaustion. This principle will help to explain the leading phenomena produced by cerebral anæmia. When a healthy individual suddenly loses a large quantity of blood, the irritability of the nervous matter becomes increased, and phenomena of irritation, such as contraction of the pupils, restlessness, and ringing noises in the ears, are produced, and there may soon be a large discharge of nervous energy from the cortex of the brain, giving rise to general convulsions followed by unconsciousness.

When the anæmia takes place more gradually, the outgoing discharges will be less powerful, and they will only produce the

signs of mental irritability, to be followed by a drowsy or somnolent condition instead of complete loss of consciousness. If, on the other hand, the nervous energy of the cortex of the cerebrum be already exhausted by overwork prior to the loss of blood, the phenomena of depression may exhibit themselves from the beginning without being preceded by any signs of irritation, and under these circumstances unconsciousness may be produced without being preceded by general convulsions.

§ 770. *Course and Duration.*—Simple faintness from emotional causes usually soon ends in recovery, although a fatal case is rarely met with; but it is probable that in these cases there is some amount of degeneration of the muscular walls of the heart. The syncope which attacks patients convalescent from acute disease, when they assume the erect posture for the first time, is more dangerous and liable to prove fatal.

§ 771. *Diagnosis.*—The symptoms of cerebral anæmia are not unlike those of hyperæmia of the brain, and the delirium from anæmia which arises in the course of acute diseases may very readily be mistaken for the delirium of active congestion. Anæmia and hyperæmia of the brain can, indeed, only be distinguished from one another by careful attention to the concomitant symptoms. The cerebral symptoms themselves are not to be relied upon, as the delirium in anæmia may be as violent as in congestion, and the colour of the face is not always a faithful index of the condition of the cerebral circulation. The diagnosis must be founded upon the general history of the case, the nature of the concomitant symptoms, and the treatment which has been adopted prior to the onset of the delirium. As further aids to the diagnosis, it may be tried whether the erect or horizontal posture has any influence in aggravating or diminishing the symptoms, and whether they are increased or relieved by alcoholic stimulants. The state of the general circulation must also be carefully examined.

§ 772. The *prognosis* in a case of hydrocephaloid disease is generally favourable, provided that the true nature of the affection be recognised and appropriate treatment adopted.

A similar remark may be made with respect to the delirium of anæmia, although it may sometimes be prolonged and occasionally prove incurable. The prognosis of the cerebral anæmia caused by heart disease will depend upon the gravity of the cardiac affection; and fatal syncope is very apt to occur in aortic regurgitation when accompaned by dilatation of the left ventricle.

§ 773. *Treatment.*—The treatment must vary according as the anæmia is acute or chronic, limited to the brain, or affecting the entire body.

In an ordinary *fainting fit* the patient should as speedily as possible be placed in the recumbent posture, and recovery usually takes place without any further treatment. If the symptoms are more persistent, some form of cutaneous irritation may be employed. The most usual and readiest method is to sprinkle the face with cold water, or to fleck the face with the corner of a towel dipped in cold water. A more efficient method, however, is the application of the metallic electric brush if a battery happen to be at hand. Mustard applications have been employed, but they are too slow in their action. The preparations of ammonia and other substances, which irritate the trigeminus and olfactory nerves, are also useful adjuncts to the treatment, and stimulating enemata may be resorted to. As soon as the patient can swallow, and especially if the heart's action be feeble, stimulants, such as coffee or brandy, must be administered. The more volatile the agent the sooner will it be absorbed, hence ether is especially useful; and the same may be said of champagne, since the experiments of Bernard have proved that the presence of the carbonic acid promotes the absorption of alcohol.

In severe cases of cerebral anæmia after profuse hæmorrhage, in addition to the means already mentioned, the body of the patient ought to be covered with warm blankets or other clothing and surrounded by bottles containing hot water; and in order to increase the flow of blood towards the brain the head ought to be kept in a low position, while pressure is maintained over the abdominal and axillary arteries, a proceeding which will direct the stream of blood towards the

carotids and will consequently raise the tension in these vessels. In cases of severe and prolonged cerebral anæmia from loss of blood transfusion should be tried as a last resort.

The cerebral anæmia which arises during the course of acute diseases must be treated by the judicious use of wine and nourishing diet, and if the case admit of it by such tonics as quinine and iron. The patient should also be instructed not to raise his head from the pillow so long as the action is accompanied by dizziness or other symptoms indicative of anæmia. When delirium or other forms of cerebral excitement accompanies the anæmia, the great aim of treatment should be to procure sleep. A full dose of chloral sometimes acts very well in these cases; but according to my experience an opiate is much more reliable and efficacious. A single subcutaneous injection of morphia is frequently followed by calm sleep, and the patient awakes with restored mental faculties. The dose should not, as a rule, be more than from one-eighth to one-fourth of a grain.

Hydrocephaloid disease must be treated on the same general principles. The diarrhœa or other disease which has produced the anæmia must be attended to; and the case must be treated by warm applications, appropriate nourishment, and stimulants, such as wine and musk.

(ii.) Hyperæmia of the Brain.

§ 774. *Etiology.*—Congestion of the brain, like congestion of other organs, may be either active or passive. The former is also called the *hyperœmia of fluxion,* and the latter the *hyperœmia of stasis.*

Active Congestion.—Irritation of the tissues of the brain causes congestion, but such cases generally terminate in encephalitis, and the congestion is usually more or less local. The causes of universal active congestion must, therefore, be sought in the state of the general circulation rather than in the brain itself. All conditions which raise the arterial tension must tend to produce congestion of the brain, unless, indeed, the increased tension be caused, as in chronic Bright's disease, by a diminution of the lumen of the arterioles all over the body, including those of the brain. An increased flow of blood to the brain

may be produced artificially by surrounding one or more of the limbs by Esmarck's bandages, or by compression of the abdominal aorta, or of some of the large arteries of the body.

Exposure of the surface of the body to cold raises the arterial tension and occasions congestion of the internal organs, and the same effect is produced during the cold stage of intermittent fever, and the rigors which usher in most severe acute diseases. The sudden arrest of habitual discharges may produce congestion of the brain by increasing arterial tension.

Hypertrophy of the heart does not often cause congestion of the brain, inasmuch as the hypertrophy is always compensatory to some resistance offered to the onward flow of blood. Congestion of the brain may, however, be caused by the increased activity of the heart, which accompanies emotional excitement. But in such cases the direct excitement of the tissues of the brain, which underlies the emotional disorder, must co-operate with the increased activity of the heart as an important factor in the production of congestion.

Paralysis of the sympathetic or irritation of the inhibitory nerves of the arteries of the brain may produce cerebral hyperæmia.

Plethoric and nervous individuals, especially of the female sex, are sometimes attacked with dizziness, headache, and flushing of the face in the absence of emotional excitement or disturbance of the circulation generally that would account for the symptoms, which must, therefore, be referred to derangement of the vaso-motor nerves of the brain. The vertigo and drowsiness which accompany irritation of the stomach is probably often caused by cerebral hyperæmia. The experiments of S. Mayer and Pribram have shown that direct electrical or mechanical irritation of the walls of the stomach produces an increase of the arterial tension and slowing of the pulse, caused probably by a reflex contraction of the arterioles of the body generally. If, under these circumstances, the contraction of the arterioles of each organ in the body were in a condition of perfect equilibrium with that of the arterioles of every other organ, no congestion of any of them would take place. It is not likely that complete equilibrium can exist between the contraction of the vessels of all the organs of the body in any

case, and it is manifest that the organ whose vessels began first to dilate must become more or less congested.

Some poisonous agents appear to have the effect of producing congestion of the brain; most of the narcotics and stimulants appear to me to act upon the tissues of the brain first, and to produce congestion as a secondary action. The more diffusible stimulants, as ether, chloroform, and alcohol, no doubt cause a certain amount of congestion of the brain, just as they produce flushing of the face by paralysing the sympathetic; but nitrite of amyl and its allies appear to be the only known agents which act specially on the vaso-motor system before affecting the tissue of the cortex of the brain.

The cerebral symptoms in hyperpyrexia and insolation were at one time referred to congestion of the brain; but it is much more probable that the high temperature acts in a deleterious manner on the cerebral tissues. The cerebral symptoms of fevers are probably due quite as much to qualitative as to quantitative alterations of the blood in the brain.

Active cerebral congestion appears to be more common in males than in females, and in adults than in either the old or the young. The statistics of Andral and of Hammond tend to show that the disease is, as might be expected, more common in winter than in summer. Heredity undoubtedly exercises some influence in causing cerebral hyperæmia, but it is probable that the influence is indirect rather than direct, as in the gouty diathesis.

Passive Congestion.—Venous congestion of the brain may be only part of venous congestion of the whole body, or it may be produced by special causes. General venous congestion is caused by diseases of the heart and lungs; and for the mechanism by which this congestion is brought about the reader is referred to works devoted to diseases of these organs.

Congestion is also caused by all local diseases which retard the return of blood from the brain.

§ 775. *Symptoms.*—Congestion of the brain gives rise to symptoms which vary widely in different cases; but for clinical purposes three varieties may be described—(*a*) the *slight*, (*b*) the *severe*, and (*c*) the *apoplectic* form.

(*a*) In the *slight* form of congestion the prominent symptoms are sensory disorders. The patient complains of severe headache, either deep-seated or lancinating, aggravated by movement, light, sound, or heat, while all intellectual efforts become impossible. Patients at the same time complain of dizziness, tinnitus aurium, and optical illusions. Sleeplessness is an early and important symptom. It is accompanied by restlessness and agitation; and if sleep do supervene, it is disturbed by horrible dreams, and the patient awakes unrefreshed without relief to the headache.

In the arterial variety of congestion the patients are fretful, restless, and excitable; but although they avoid mental exertion for fear of aggravating their sufferings, their intellectual faculties are not impaired; on the contrary, there may be excessive mental activity. There may be some numbness and formication of the extremities, but there are no motor disorders. There is generally obstinate constipation. This form of congestion frequently occurs in plethoric subjects. The slightest exciting cause, such as a full meal after a prolonged fast, or unusual mental fatigue, often suffices to induce an attack, which may last for a few hours only or persist for some days.

In the venous variety of hyperæmia the phenomena of mental depression are usually more marked than those of excitement. There is a dull sense of oppression in the head, the face is livid, there are mental torpor with a tendency to sleep, and a certain amount of confusion of ideas, especially on awaking after a short sleep.

(*b*) In the *severe* forms other symptoms are added. The patient suffers so much from vertigo that he is unable to maintain the erect posture, and sudden vomiting may occur in the absence of any gastric irritation to account for it. The pulse is slow, full, and hard; the arteries of the head and neck beat forcibly; the face is frequently, although not always, flushed, and may at times be livid, while a sensation of flying heat shoots over the head and neck. The pupils are generally contracted, and there is some degree of intolerance of light and sound. The patient complains of intense headache, and is the subject of hallucinations and illusions which pervert the judgment, and

may lead to strange and disorderly acts. He sometimes endeavours to quit his bed, and to pursue or run away from imaginary objects; he is loquacious or bursts into a flood of tears, and struggles with and tries to escape from his attendants. After some hours of excitement and struggling the skin becomes covered with sweat, the pulse is accelerated, the face of a deep red colour, and the patient presents the leading symptoms of encephalitis; the thermometer shows that the temperature, if at all altered, is only slightly above the normal. If these symptoms persist for some time, the phenomena of excitement are succeeded by those of depression, the delirium gradually gives place to mental torpor, the muscular agitation is replaced by muscular relaxation, the respiration becomes stertorous, there are involuntary evacuations, and the patient falls into a state of coma.

In some cases, especially in aged people, the severe form of congestion declares itself suddenly during the night, as a simple delirium of action. The patient awakes, does not know where he is, gets up and performs various disorderly actions of which he has no knowledge. In the morning he is quiet and sensible, but is sad, morose, and depressed; and these phenomena may be repeated for several consecutive nights, but it generally ends at last in an attack of delirium similar to that which has been just described. An abundant secretion from the conjunctiva and mucous membrane of the mouth is said to be a frequent symptom of congestion of the brain in old people (Durand-Fardel.)

Convulsions are the most frequent and most striking symptoms of cerebral congestion in infants. It must not, however, be thought that cerebral congestion always accompanies the convulsions of children. It is indeed probable that convulsions from congestion of the brain in children are rare in comparison with those which are secondary to other diseases. The symptoms of the cerebral congestion of infancy are similar in many respects to those of meningitis. Both are attended with partial or general convulsions, headache, contraction of the pupils, vomiting, and constipation. The course of the two affections, however, enables them to be readily distinguished. In congestion the child has been in good health up to the beginning of

the attack, there is little or no elevation of temperature, and the disease terminates in recovery in two or three days at most.

(*c*) The *apoplectic* form is characterised by sudden and total loss of consciousness and complete resolution of the limbs, but reflex excitability is preserved. The patient recovers consciousness in a few hours, and after a short time, two or three days at most, all the symptoms disappear without leaving a trace behind. Sometimes, however, after complete restoration to consciousness, a certain amount of muscular paralysis remains in one limb, or assumes the hemiplegic form and persists for some time.

§ 776. *Morbid Anatomy.*—It is necessary to be on one's guard against certain causes of error with respect to post-mortem appearances. Both arterial and venous hyperæmia may disappear at death without leaving a trace behind. On the other hand, when, as is usually the case, the body is laid on its back, a large quantity of blood may be found in the veins and sinuses of the occipital fossæ, caused by the influence of gravity after death, aided probably by hypostatic congestion during the last few hours of death. The act of dying by respiratory paralysis may also cause a hyperæmia of the cerebral veins when there were no symptoms of congestion during the course of the disease.

In pathological hyperæmia, when the calvarium is removed, the vessels of the diploe are frequently found congested. The veins of the dura and pia mater are prominent and full of blood, and so also are the choroid plexuses and sinuses. In the severer forms of congestion the brain is swollen, and the gyri are flattened from compression. The grey substance is of a dark red colour, its consistence is increased, and the cut surface of the white substance presents a large number of red points, from which drops of blood exude. The white substance may be of a yellowish-red colour, while at other times its colour is little altered.

In chronic congestion the vessels themselves become altered. In the venous variety the veins, especially those of the membranes, the surface of the brain, choroid plexuses, and velum interpositum are enlarged and tortuous. In chronic arterial

congestion the coats of the arterioles become hypertrophied, and the smaller vessels may present visible openings after section; miliary aneurisms are often discovered by careful examination, while a certain amount of pigment is found in the perivascular spaces. In very chronic cases œdema of the pia mater and of the choroid plexuses is found along with increase of the ventricular fluid, while the brain itself may undergo a certain degree of atrophy. The *état criblé* of Durand-Fardel consists of a sieve-like appearance of the brain caused by the round or oval openings of the vessels, some of which are as large as a pin's head. These openings are supposed by some pathologists to depend upon dilatation of the perivascular lymph-sheaths. They are commonly found at the autopsies of elderly people who have suffered for a long time from cerebral congestion.

§ 777. *Morbid Physiology.*—In the first stages of active general congestion the increased quantity of blood within the cavity of the skull is compensated by displacement of a corresponding quantity of cerebro-spinal fluid, but congestion beyond a certain degree must augment the intracranial pressure, as may be readily proved to occur in children by the increased tension of the anterior fontanelle. During the first stage of active congestion the increased supply of blood to the organ must increase its functional activity, thus explaining the irritative symptoms observed at the onset of the disease. On the other hand increase of the intracranial pressure, beyond the point at which it is compensated by the cerebro-spinal fluid, must compress the substance of the brain, and so give rise to the phenomena of depression observed in the second stage of congestion.

In the apoplectic form of congestion, attended by unconsciousness and subsequent paralysis, it is probable that a serous transudation takes place into the interstices of the tissues. In venous hyperæmia the blood pressure is transferred, so that increase of venous implies a diminution of arterial tension along with a superabundance of carbonic acid and a deficiency of oxygen, conditions which tend to narcotise the brain. The conditions present, therefore, combine to produce the phenomena of depression and not those of excitement; and the few symptoms which simulate those of increased excitement, such

as sleeplessness, restlessness, and mental irritability, are those which occur in imperfectly nourished brains or in cerebral anæmia.

§ 778. *Diagnosis.*—The thermometer may be of use in distinguishing hyperæmia from inflammatory diseases of the brain, but its indications are not to be too implicitly relied upon. Congestion may be distinguished from focal disease by the absence of the usual symptoms of a localised lesion. The apoplectic form of congestion is distinguished from true apoplexy by the transitory nature of the symptoms in the former, but it must be acknowledged that rupture of a blood-vessel in some parts of the brain may give rise to symptoms which closely resemble those of congestion.

The form of congestion attended with delirium may be mistaken for delirium tremens; but the two diseases may be distinguished by a knowledge of the habits of the patient, and the circumstances which have preceded the attack. At the same time the trembling of the lips and hands, the skin bathed in perspiration, the soft compressible pulse, the timid and frightened look, and the busy character of the delirium form a group of symptoms so characteristic that it is difficult for a practised eye to mistake the alcoholic disease for any other. A certain kind of delirium may be caused by lead poisoning, which may simulate that from congestion; but the two may be distinguished by the history of the case and by the condition of the gums.

The respiratory movements and the pulse are retained in greater integrity in congestive apoplexy than in syncope. The coma which succeeds an epileptic attack may be mistaken for the coma of the apoplectic form of congestion. These affections must be distinguished mainly by the history of convulsions, and by the fact that the tongue is often bitten during the epileptic attack.

The lighter forms of congestion may be mistaken for gastric vertigo, or for the vertigo which is caused by venereal excess. Gastric vertigo is associated with some forms of dyspepsia, it often diminishes or ceases after food, and is accompanied by nausea.

After having determined that the symptoms are caused by cerebral hyperæmia, it is then necessary to decide whether the congestion be arterial or venous, and whether it be primary or secondary. A careful examination of the lungs, heart, and blood-vessels, and the condition of the urine, will enable us to decide whether any mechanical condition is present which would cause venous or arterial congestion. If none of these conditions be present, then the congestion must be regarded as active, and its primary or secondary nature will be revealed by a knowledge of its cause. The most ordinary causes of primary congestion are insolation, unwonted mental efforts, wakefulness, and excess in eating; while the most ordinary causes of secondary congestion are gout, rheumatism, suppression of the menses and other habitual discharges, and cerebral lesions, as tumours of the encephalon.

§ 779. *Course and Prognosis.*—Great differences exist in the severity and duration of cerebral congestion. The severer forms may cause death, and even the lighter forms, if they do not present any immediate danger, are apt to recur and to produce permanent bad effects.

The prognosis in the severer forms of congestion is grave, and when delirium is present the case often terminates in hæmorrhage.

Cerebral congestion is most dangerous in old people, because the degenerated vessels are apt to rupture.

In cases of chronic disease of the brain, as tumour or vascular degeneration, cerebral congestion may aggravate the symptoms or prove the immediate cause of death.

§ 780. *Treatment.*—The treatment of cerebral congestion will vary according as its cause is found in general plethora, organic diseases of the heart, vaso-motor disturbances, or a pre-existing focus of disease in the brain.

During the attack the patient should lie in bed with the upper part of the body raised, the room darkened, and the utmost quiet enjoined. If delirium be present and the patient of a plethoric habit, a small quantity of blood may be drawn from the arm, followed by the administration of a saline purgative.

In the congestion of the brain caused by suppression of the menses, or of hæmorrhoidal discharge, leeches may be applied to the anus or to the upper part of the thigh, and be followed by a smart purgative. Aloes, or aloes in combination with sulphate of magnesia, is very useful in these cases.

In the treatment of active congestion with irritative symptoms from such causes as insolation and excessive fatigue general bleeding is no longer permissible, and the main reliance must be placed upon saline or other hydragogue purgatives, mustard pediluvia, and cold steadily applied to the head by means of an ice bag or evaporating lotions. If the patient be of a gouty constitution, a saline mixture with colchicum may be administered after the bowels have been acted upon. Aconite is a useful remedy in many cases.

The diet, of course, must be plain and unstimulating during the attack. In venous congestion a small bleeding may occasionally be advisable, inasmuch as the lowering of the tension within the veins permits the arterial blood to pass more freely through the capillaries, and the tissues become better nourished. The main reliance in the treatment of venous hyperæmia of the brain must be placed upon drastic purgatives, diuretics, and cardiac tonics as digitalis. Those who have once suffered from one or more attacks of active cerebral congestion should adopt certain hygienic precautions to prevent a repetition of the attack. Their diet should be plain, consisting in large part of herbaceous vegetables and fruits, and all stimulants, as wine, tea, and coffee, should be proscribed. They should avoid everything tending to cause mental excitement, such as public speaking, theatres and concerts, intellectual efforts, late hours, and venereal excess.

CHAPTER XI.

II. DIFFUSED DISEASES OF THE ENCEPHALON (Continued).

(II.) ATROPHY AND HYPERTROPHY OF THE BRAIN.

(i.) Atrophy of the Brain.

§ 781. *Atrophy of the Corpus Callosum.*—The corpus callosum begins to develop towards the end of the fourth month of intra-uterine life by the outgrowth of two lateral stumps from the internal surface of the hemisphere vesicles. These grow towards one another, and unite between the sixteenth and twentieth weeks of intra-uterine life, the union taking place from before backwards. The development of the corpus callosum may be arrested at any period, so that it may be entirely wanting, or it may grow on each side to nearly the normal size, but union fails to take place in the middle line. At other times the union may be partial and the corpus callosum be represented by a rudimentary bridge, or sieve-like plate of tissue. When the corpus callosum is entirely wanting, its radiating fibres are also absent, the cavities of the lateral ventricles are unusually large, they are at the same time filled with serous fluid, the ependyma is granular and thickened, and the choroid plexuses are generally found diseased.

§ 782. *Symptoms.*—Congenital deficiency of the corpus callosum has generally been found associated with idiocy, or at least with some degree of mental deficiency. The mental defects in such cases do not present anything characteristic, so that this condition cannot be recognised during life. Some cases of arrest of development of the corpus callosum have, however, been reported in which no marked mental deficiency was observed during life (Paget, Jolly, Malinverni, Eichler).

§ 783. *Atrophy of the Cerebellum.*—Slight degrees of atrophy are often observed, either as complications of cerebral diseases, or as consequences of diseased foci in the organ itself, but these lesions cannot be recognised during life. Cases of uncomplicated atrophy of the cerebellum are rare, but the few which have been observed are important from the light they throw on the functions of the organ.

§ 784. *Etiology.*—In the cases reported by Lallement and Otto, and probably also to some extent in Combette's case, the atrophy was congenital. In a case reported by Meynert and another by Pierret, fright is assigned as the cause. In Clapton's case the nervous symptoms appeared after measles, and gradaully diminished in severity.

§ 785. *Symptoms.*— The following were the symptoms in Combette's case: The girl Labrorse at 12 years was weak-minded and suffered from epileptic attacks. She could not stand or walk until five years old. At seven her lower extremities were feeble, and she often fell. During the last three months of life she was bedridden and could scarcely move her legs, and her articulation was imperfect.

Motor disturbances were observed in the cases reported by Meynert, Pierret, Fiedler, Clapton, Dugnet, and Moreau. Most authors describe these as those of ataxia; while others state that the patients could walk, but only slowly and carefully; that they fell frequently, especially backwards; and that in walking they seized hold of objects within their reach. All these patients had also either persistent or temporary disturbance of speech.

No motor disturbance was noticed by Lallement and Otto. Otto's patient was impulsive in his movements, but whether due to psychical causes or disturbance of co-ordination could not be determined.

Weakness of mind, even idiocy, characterised the patients of Clapton, Otto, and Fiedler; while Pierret's case suffered from weakness of memory.

Epileptiform convulsions are frequently mentioned, but do not appear to have any special significance.

Symptoms occasionally noticed are analgesia (Fiedler) and slight disturbances of sensibility (Pierret).

§ 786. *Morbid Anatomy.*—In Combette's case the entire organ had disappeared. There was no trace of a pons, although the cerebral arteries were present and of normal size. In other reported cases the cerebellum has been found reduced to about half the normal size.

Lallement mentions a case in which the left lobes of the cerebellum, including its middle and superior peduncles, was reduced to the size of a nut, and the transverse fibres of the pons, the right corpus striatum, and the right olivary body were atrophied. In Dugnet's case the cerebellum was about half the normal weight. The atrophy was bilateral and general, and the substance of the organ showed well-marked sclerosis. Somewhat similar cases have been reported by Clapton and Fiedler-Bergmann. No statement is made with regard to the condition of the pons.

Meynert describes a similar degeneration in the pons, which he regards as a secondary degeneration, and not as the starting point of the affection. The cerebellum itself was much altered, especially on the right side. The posterior pyramids of the medulla were implicated as well as the pons and the crus cerebelli ad pontem. In Pierret's case there was an intense degree of sclerotic atrophy, which affected chiefly the vertical diameter of the organ, the grey substance being specially affected. The transverse fibres of the pons and both olivary bodies were atrophied and replaced by connective tissue. In Otto's case the left lobe was the more atrophied, and the pons on the left side was narrower than on the right. The space usually occupied by the cerebellum was replaced by hyperostosis of the occipital bone.

§ 787. *Complications and Diagnosis.*—Atrophy of the lateral lobe of the cerebellum has been found associated with atrophy of the transverse fibres of the pons on the same side, and with atrophy of the olivary body and cerebral hemisphere on the opposite side. In some few cases the atrophy of the cerebrum and cerebellum occurs on the same side.

Atrophy of the cerebellum is difficult to distinguish from chronic affections of the organ, but headache and vomiting, which are common in the latter, are rare in the former. The sensory and reflex disturbances of locomotor ataxia serve to distinguish it from atrophy of the cerebellum. The symptoms of the initial stage of insular sclerosis may be similar to those of atrophy of the cerebellum, but when the characteristic tremors of the former appear the diagnosis is easy.

(ii.) Hypertrophy of the Brain.

Hypertrophy of the brain includes several different morbid conditions. It is also usual to include along with hypertrophy a new formation of cerebral substance within the substance of the brain itself, a condition which Virchow has called Heterotopia of the brain. Hypertrophy may be divided into *general* and *partial* hypertrophy.

§ 788. *Etiology.*—Hypertrophy of the brain appears to be generally congenital. Several of the reported cases were associated with peripheral multiple neuroma (Hesselbach, Hitchcock, Betz), and both of these conditions are frequent accompaniments of idiocy or delayed mental development. The affection is almost always developed soon after birth or in early infancy. A few cases appear to have developed subsequently to an injury to the head (Tuke, Dance), while the disease appears to have been a result of chronic lead poisoning (Andral, Laennec, Bright).

Symptoms. — Severe headache, with remissions or even complete intermissions, is a prominent symptom of hypertrophy of the brain. Epileptiform convulsions, local spasms, attacks of laryngismus stridulus, and tremors are also commonly observed. The pulse is usually retarded, but it may occasionally be much accelerated (Steiner). The symptoms of *chronic* cerebral hypertrophy are not well known. The affection in children is sometimes associated with premature development (Elliotson), or at least a degree of development corresponding to their age. In other cases, again, there is more or

less weakness of mind, amounting even to the highest degree of idiocy. The tongue is often increased in size, and often protrudes from the mouth. Drowsiness is an occasional but by no means constant symptom. Some of the affected children are liable to fall frequently, being over-balanced by the great weight of the head. Disturbances of the nerves of the general or special senses are comparatively rare. The optic nerve in particular is seldom mentioned; Steiner and Neurenthar alone speak of the sudden occurrence of blindness, others mention photophobia. A careful ophthalmoscopic examination of the optic discs might have given more positive results. Tinnitus and subjective noises in the head are sometimes present. Death often results from an attack of convulsions, or in coma due to cerebral compression, while many of those affected die from some intercurrent disease.

§ 789. *Morbid Anatomy.*—The anatomical appearances differ according as the hypertrophy is partial or general.

General hypertrophy begins in the earlier years of childhood, and the skull enlarges just as in hydrocephalus. If the disease appear for the first time after the bones of the skull have become ossified, the bones are subjected to compression from within and undergo atrophy at certain points. It is probable that this condition is, however, connected with the changes which the cranial bones are known to undergo in congenital syphilis.

The *cerebral membranes* are generally compressed against and become adherent to each other and to the bones of the skull. The membranes are thin, their blood-vessels are scarcely visible, and every trace of cerebral spinal fluid is absent.

The *lateral ventricles* are compressed so that they either contain no fluid, or only a small amount. The convolutions are flattened and so pressed together that the sulci seem entirely obliterated. The brain substance shows a marked change of consistency; it is tough, like boiled white of egg or cheese. Tuke could make no impression on it by a column of water five feet high.

The brain is, as a rule, found anæmic on section, and the grey substance so pale that it differs little from the white. This

extreme anæmia, however, appears to be a terminal phenomena due to the increased compression.

The average weight of the brain in adults is, according to Huschke, from 1,500 to 1,600 grammes; although the weight of the brains of persons prominent in literature has considerably exceeded 2,000 grammes. The absolute weight of the brain, considered without reference to its density and other circumstances, only warrants the diagnosis of hypertrophy when the average is considerably exceeded.

The *specific gravity* of the cerebral mass should also be taken into account. Tuke found the specific gravity unchanged on the diseased side in his case of unilateral hypertrophy, being 1,036 on both sides, but the result differed from that obtained with normal brains in the fact that it was the same in the grey as in the white substance.

The cerebrum is as a rule alone affected with hypertrophy, but there are a few cases in which the cerebellum also is said to have been affected (Sweatmann).

Virchow attributes the increased size of the brain to hyperplasia of the neuroglia.

Partial hypertrophy is rarer than the general form of the affection, and even some of the reported cases are not beyond suspicion, inasmuch as gliomatous tumours were probably mistaken for partial hypertrophy of the brain. Hesselbach mentions the case of a man who inherited the disease from his father, and who, besides multiple neuromata of the peripheral nerves, presented a considerable enlargement of the sympathetic ganglia, and of one of the middle cerebellar peduncles.

§ 790. *Course.*—It is very difficult in many cases to estimate the duration of the disease, inasmuch as even in the cases which appear to be primarily acute the course may actually have been protracted, the disease being latent until the space in the cranium became limited. Many chronic cases suddenly assume an acute character, and terminate quickly in death. The fatal termination is often caused by an intercurrent disease, such as diarrhœa or bronchitis.

Chronic cases may extend over many years, the disease apparently remaining stationary. A sudden increase in volume,

whether in a brain previously healthy or in one already chronically enlarged, may cause rapid death.

Acute hypertrophy produces the symptoms common to all diseases causing compression of the brain; while the *chronic* form, especially in children, can scarcely be distinguished from chronic hydrocephalus.

§ 791. *Diagnosis, Prognosis, and Treatment.*—The diagnosis is always uncertain, but the possibility of this condition ought certainly to be borne in mind before puncturing a hydrocephalic head. The prognosis is always unfavourable, but on account of the impossibility of making a diagnosis a prognosis cannot well be given. No treatment is of any avail.

§ 792. *Heterotopia of Brain Substance.*—This condition was first described by Virchow, and has hitherto been principally of interest to the morbid anatomist. Simon found small accessory gyri situated on the summit of the convolutions. Virchow observed in one case an apparently new formation of gyri within the white substance of the posterior lobe. He also found a hyperplastic malformation of the caudate nucleus. Klob found a mass of white cerebral substance, the size of a bean, hanging from a pedicle between the optic nerves.

Microscopic examination of the heterotopic grey substance shows, as a rule, similar elements to those of the normal cortex, but the ganglion cells in the former are pigmented and fatty.

These conditions have hitherto been found in epileptics, idiots, or in persons otherwise mentally affected, but their clinical significance is somewhat doubtful. All authors regard these malformations as congenital.

CHAPTER XII.

II. DIFFUSED DISEASES OF THE ENCEPHALON (Continued).

(III.) SHOCK AND CONCUSSION.

(i.) Shock.

§ 793. *Definition.*—Shock results from profound bodily or mental impressions, and appears to depend upon a sudden temporary impairment or permanent extinction of the functions of the nervous system, the cardiac, vaso-motor, and respiratory centres in the medulla oblongata being specially affected.

§ 794. *Etiology.*—Amongst the predisposing causes of shock may be mentioned constitutional peculiarities or idiosyncrasies. Some people inherit so unstable a nervous system that an injury inappreciable to others may produce in them all the phenomena of shock. The irritability of the nervous system is greater in youth than in old age, and consequently the phenomena of shock are produced by slighter causes in the former than in the latter. The recuperative power, on the other hand, is greater in youth than in the aged, hence the phenomena of shock, although less obvious, are more grave in the latter than in the former. Shock is, as a rule, more easily produced in the female than the male sex. An undue irritability of the nervous system is sometimes acquired rather than inherited, and all conditions which tend to impair the nutrition of the nervous system, as exhausting diseases, cerebral anæmia from whatever cause arising, dissipation, prolonged pain, the depressing passions, predispose to the production of shock. During the stage of depression of shock the irritability of the nervous system is nearly exhausted, a greater resistance is thus offered to the conduction of impulses from the periphery to the higher nerve-

centres, and consequently during this stage a second injury produces a much less effect than the first. During the stage of reaction, however, the irritability of the nervous system is excessive, so that a slight stimulus may produce a profound effect. In persons of powerful will and stable nervous systems the effect of an unexpected injury is greater than if the patient were prepared for its reception; while in emotional patients, with unstable nervous systems, previous knowledge of an impending injury greatly intensifies its effects. Injuries of the abdominal viscera, genitals, joints, and bones produce more profound effects than injuries of other parts of the body.

The exciting causes of shock are sudden and severe or extensive injuries of any part of the body, whether produced by accidental wounds or burns, or by surgical operations. Shock is also produced by strong emotional excitement of any kind, although the depressing passions, as fear and anger, are more liable to cause it than pleasurable passions, like joy.

§ 795. *Symptoms.*—Cases of shock may be divided clinically into two forms—(*a*) cases in which the symptoms of depression predominate, and (*b*) cases in which the symptoms of prostration are mixed with those of excitement (Travers, Savory). Dr. Lauder Brunton has proposed to call these forms respectively *torpid* and *erethismic* shock.

(*a*) *Torpid Shock.*—In the torpid form of shock the patient lies utterly prostrate; the surface of the body is pale, cold, and covered by a clammy sweat, which collects in drops on the forehead and eyebrows; the lips are bloodless, the nostrils dilated, and the countenance of a dull aspect and shrunken, while the eyes have lost their lustre, are sunk in their sockets, and partially concealed by the drooping lids. There is complete muscular relaxation, which may even extend to the sphincters. If the patient be conscious, he may complain of feeling cold and faint, while the whole body may tremble. The pulse is frequent, irregular, unequal, and feeble or imperceptible at the wrist, although the fluttering action of the heart may be heard on auscultation. The respiratory movements are irregular and gasping, or short and feeble, the respirations being sometimes so superficial that they are scarcely visible, although a slight

movement of the diaphragm may generally be discovered by careful observation. The temperature of the body is depressed. The patient suffers from vertigo and dimness of vision, while in the less severe cases there is nausea, vomiting, and hiccough.

The psychical symptoms consist of mental depression, restlessness, confusion of thought, incoherence, or drowsiness, although the patient generally gives rational replies to definite questions. At other times the patient appears singularly calm and rational, while the various senses remain unaffected, hearing being sometimes unusually acute.

(*b*) *Erethismic Shock.*—This form of shock is rare, the majority of cases in which symptoms of prostration are mixed with those of excitement being preceded by a distinct, though it may be transient, stage of collapse. The skin is at first hot and dry; the face is flushed and wears an anxious expression; the pulse is frequent, quick, and bounding, but always compressible; the respirations are hurried, imperfect, and interrupted by sighs; the tongue is tremulous; and the patient complains of thirst, rigors are occasionally present, while vomiting is a frequent and sometimes obstinate symptom. The mental and bodily prostration of collapse is succeeded by tremor and twitchings of the muscles, there is restlessness, jactitation, præcordial anxiety, and delirium. The psychical disturbances observed are somewhat variable. At times the patient merely presents a peculiar irritability of manner, with an increased disposition to talk, sometimes rationally, occasionally incoherently. At other times the patient has strange illusions, attended with a peculiar dread of impending evil. In some cases, however, there is the fiercest maniacal raving, which is most pronounced during the night, or the delirium may assume all the characteristics of that observed in *delirium tremens.*

The patient either obtains no sleep, or it is partial, interrupted, and unrefreshing. As the exhaustion increases the skin becomes covered with a cold, clammy, and often profuse sweat. The face becomes pale and the expression haggard, the pulse is frequent, irregular, fluttering, and uncountable. Subsultus and slight convulsions supervene, and the patient dies comatose.

§ 796. *Course, Termination, and Duration.*—The degree of shock varies greatly. In the *milder forms* of the affection the symptoms are chiefly those of an ordinary fainting fit. Loss of consciousness is, however, a more marked characteristic of syncope than of slight shock, whilst recovery from the former is more rapid than from the latter.

In the *severest form* of shock the functions of the nervous system are suddenly abolished, and the heart ceases to beat.

Between the slighter forms of shock, which resemble an ordinary fainting fit, and the severest form, which terminates in instantaneous death, innumerable transitional forms may be observed. The medium degrees of shock are known under the name of *collapse.*

The slighter degrees of shock terminate speedily in direct recovery, while the severest form terminates of course in death. In the intermediate forms recovery may ultimately take place, but it is preceded by a stage in which the symptoms of collapse give place to those of excitement, this stage being called the *period of reaction.* The period of reaction is characterised by improved pulse and respirations, restoration of muscular power, and increase of temperature. Reaction may sometimes be in excess and febrile symptoms supervene.

The symptoms of the period of reaction may gradually give place to health, but in some cases relapses are not unfrequent, and convalescence is then protracted. In other cases the torpid may be replaced by the erethismic form of the disease. Recovery from severe shock is often partial only, the irritability of the nervous system remains permanently increased, a condition which predisposes to the production of shock from the application of slight exciting causes.

§ 797. *Morbid Anatomy.*—No definite changes have been found in the nervous system in cases of shock. All the cavities of the heart are usually distended with blood and the venous system is engorged. Travers, however, relates instances of death from shock when, on dissection, both sides of the heart were found empty. Dr. A. H. Young informs me that he has observed cases of this kind in the post mortem room, and that

in such cases the abdominal veins have not unfrequently been enormously engorged with blood.

§ 798. *Morbid Physiology.*—In its widest acceptation shock is the sudden impairment or abolition of the functions of protoplasm by the application of an excessive stimulus. The functions of the protoplasm of all the organs of the body are doubtless impaired by severe injuries; but in the higher animals the disorder occasioned in the functions of the nervous system becomes so predominant that the direct effects of injuries on the protoplasm of the other tissues of the body may be practically disregarded. The most striking phenomena of shock are those which cluster around the organs of circulation. The experiments of Goltz, repeated by Brunton, show that shock probably results from cardiac paralysis and vaso-motor paralysis of the large vascular trunks of the abdomen. Brunton states that blows of moderate severity on the abdomen of frogs produce in some stoppage of the heart, without dilatation of the abdominal vessels, and in others vascular dilatation, without arrest of the cardiac pulsations, while severe blows generally produce both effects simultaneously. The vessels of the abdomen are so large that when fully relaxed they are capable of containing almost all the blood in the body, and consequently the condition resulting from their rapid dilatation is equivalent to a sudden hæmorrhage. This double condition of cardiac failure and vascular dilatation produces anæmia of the nerve-centres, and this accounts for the pallor and coldness of the surface of the body, and the weak, compressible, and fluttering pulse. It must not, however, be forgotten that the injury which has disordered the functions of the vaso-motor and cardiac centres in the medulla oblongata must also have produced a direct deleterious effect upon other nerve-centres. The disorders of respiration, the cries of pain, and the various bodily contortions which are caused by bodily injuries, or severe mental excitement, show that excessive stimuli occasion powerful outgoing discharges from the higher nerve-centres. But a powerful discharge from a nerve-centre is followed by temporary impairment or abolition of its functions, and it is probable that the arrest of the functions of the higher nerve-centres,

caused by the application of a sudden and powerful stimulus, is the most important factor in the production of the phenomena of shock.

The symptoms of the erethismic variety may be explained partly on the supposition that the nervous tissues are in the irritable condition frequently observed when they are imperfectly nourished, and partly on the supposition that the phenomena of excitement are in great part due to the abolition of the functions of the higher nerve-centres, thus permitting a greater activity of the lower centres to take place.

§ 799. *Diagnosis.*—The symptoms of shock may be mistaken for those of syncope, but the former are more protracted than the latter. If a history of an injury or of the presence of some other exciting cause of shock can be ascertained, the diagnosis is rendered easy. It is not always easy to distinguish profound collapse from actual death. The difficulty can only arise in those rare cases of collapse in which the action of the heart ceases to be heard on auscultation and the respiratory movements fail to be detected, or powerful cutaneous irritants cease to excite any reflex action. The most certain test consists of the electrical examination of the muscles and nerves, all reaction in them ceasing in from one and a half to three hours after death.

§ 800. *Prognosis.*—The prognosis depends upon the degree of shock, and the constitution of the patient. Speaking broadly, the prognosis is the more favourable the less the intensity of the shock, and the shorter the time which elapses before reaction takes place.

§ 801. *Treatment.*—The treatment of shock is the same generally as that of syncope, the great aim being to excite reaction. It must, however, be constantly borne in mind that reaction, once excited, is apt to become excessive. In the severer forms of shock the heart must be excited to action. The mode of procedure to be adopted depends upon whether the arrest of the heart's action is of purely nervous origin, or it is complicated or caused by great hæmorrhage. In

the former case the heart is probably distended and the cervical veins engorged, and consequently venesection from the external jugular veins should be immediately resorted to (Savory); while in the latter condition the cavities of the heart are empty, and transfusion of blood appears to be the only means offering a chance of success. In any case warmth is indicated, and the patient should be well wrapped up in warm blankets and surrounded by hot bottles. Stimulants must now be given internally, brandy being generally the readiest and best. If the patient be unable to swallow, ammonia or ether may be administered subcutaneously, or the former may be injected into a vein, or a stimulating enema may be given. Tincture of digitalis may be administered in half drachm doses, but its action is much too slow to be of much use in the early stage of urgent cases.

(ii.) Concussion.

Concussion is a special form of shock, the disturbance in the functions of the nervous system being caused by direct commotion of the substance of the brain.

§ 802. *Etiology.*—The exciting causes of concussion are severe injuries, as falls from a height or blows on the head, which cause the whole mass of the encephalon to be jolted or shaken. Concussion may be complicated by fracture of the skull, and in such cases the effects of the concussion are often less severe than in uncomplicated cases, apparently because a certain amount of the applied force is expended in producing the fracture.

§ 803. *Symptoms.*—The symptoms of concussion may be described under four stages: (*a*) The stage of *collapse*; (*b*) the stage of *rallying* or of *vomiting*; (*c*) the stage of *reaction*; (*d*) the stage of *gradual convalescence* (Hutchinson).

(*a*) *The Stage of Collapse.*—The symptoms during this stage are very variable both in character and duration. In the slighter forms the patient suffers from transient confusion of ideas and slight giddiness. He may feel weak and faint, and be unable to maintain the erect posture. In the more severe forms the

symptoms are those of collapse, with loss of consciousness; but paralysis, such as occurs in compression of the brain, is never present. The patient is semi-conscious or insensible, most reflex actions are abolished, the skin is cold and pallid, the respirations superficial and shallow, the pulse feeble or imperceptible at the wrist, whilst the pupils may either be contracted, dilated, or unequal.

(*b*) *The Stage of Rallying or of Vomiting.*—After a period varying from a few minutes even up to days, according to the severity of the attack, the patient usually begins to show signs of rallying. This stage is often ushered in by vomiting, or very occasionally by an epileptiform attack; the pulse improves in strength, the respirations become less shallow and more perceptible, the body becomes warmer, reflex actions can be excited, and the patient gives evidence of returning sensibility, while he may exhibit signs of mental distress.

(*c*) *The Stage of Reaction.*—The symptoms of the stage of rallying are succeeded by those of reaction. In this stage the phenomena of febrile reaction manifest themselves by the usual symptoms, hot and dry skin, quick and hard pulse, and scanty urine; while the patient is drowsy, yet quite conscious when roused by a question addressed to him. In some cases these symptoms gradually develop into those of compression and the patient dies comatose, while in other cases the symptoms of reaction give place to those of inflammation of the brain. This stage may continue from three to twelve days in cases which recover.

(*d*) *The Stage of Convalescence.*—Reaction is followed by a progressive subsidence of the symptoms, and either by a gradual restoration of the patient to health, or the establishment of one or other of several chronic affections of the nervous system.

Cerebral Irritation.—In another form of nervous disturbance following injuries of the head, and described by Erichsen under the name of cerebral irritation, the phenomena of cerebral excitement are mixed with those of loss of function. The patient assumes a peculiar attitude; he lies with the body bent forwards, the knees drawn up on the abdomen, the legs bent on the thighs, the forearms flexed on the arms, and the hands drawn. The patient is restless, and frequently changes his

position, but never stretches himself out nor assumes the supine posture (Erichsen). The eyelids are firmly closed, the pupils are contracted, the surface of the body is pale and cold, and the pulse is small, feeble, and slow, being seldom above 70 beats per minute. The sphincters remain, as a rule, unaffected.

The patient is indifferent to everything around him, and is only partially conscious. He may, however, be roused when addressed in a loud voice, and then looks up, mutters indistinctly, or frowns and turns hastily away. His sleep is not stertorous.

After a period of from one to three weeks, the pulse improves, the body becomes warmer, the flexed attitude is abandoned, and the mental irritability gives place to mental feebleness and torpidity.

§ 804. *Course, Duration, and Terminations.*—The mildest cases of concussion usually make a speedy recovery, although the patient may suffer for many days from confusion of thought, listlessness, and indisposition for mental exertion. In the severest cases rapid death may occur. Between these extremes every intermediate degree in the severity of the symptoms is observed. In some cases the patient may never rally, but die after a more or less prolonged stage of collapse. In other cases the patient rallies, but the symptoms of reaction are excessive, and followed either by those of compression or of encephalitis. But even when the period of reaction is safely passed, serious consequences may be observed during and subsequent to the period of convalescence. In some cases complete recovery may apparently take place, and the patient resumes his ordinary avocations; but he remains excitable, and gives way to uncontrollable bursts of passion. He complains of persistent headaches, his mental powers are impaired, his speech may be indistinct and stuttering, while vision, smelling, and hearing may be permanently impaired. The severity of the remote consequences of concussion do not always bear a direct ratio to the severity of the symptoms of the first stage of concussion, apparently trivial cases being sometimes followed by serious consequences.

In cerebral irritation recovery is slow, but may ultimately be perfect, although remote consequences are not unfrequently manifested.

§ 805. *Morbid Anatomy.*—In most cases of death from concussion the autopsy reveals actual structural changes in the brain, consisting of superficial lacerations, or of minute hæmorrhagic extravasations, either studded on the surface of the brain or in its substance, and occasionally of diffused ecchymosis of the pia mater (Hutchinson). The most common sites of these superficial hæmorrhagic extravasations are opposite bony ridges, and at projecting parts of the brain. In some cases no structural lesions of any kind have been discovered. It is probable that in the majority of the cases which recover no structural changes which could be recognised even by microscopical examination are produced. It is much more likely that the essential structural alterations in concussion consist of a molecular disturbance of the substance of the cerebro-spinal centres.

§ 806. *Morbid Physiology.*—Various hypotheses have from time to time been advanced to account for the phenomena of concussion. Nothnagel thinks that the strong irritation of the sensory nerves produced by the injury causes contraction of the vessels of the brain, which in its turn produces anæmia and loss of function of the cortex. Fischer, on the other hand, attributes the phenomena with more justice to vascular paralysis; but if the shock of the blow is sufficient to paralyse the vaso-motor centres, what is to prevent it from paralysing a more extended portion of the nervous system? By far the readiest way of accounting for the loss of function of the cortex is to assume that the injury has produced a molecular disturbance of the protoplasm of the tissues of the brain, which is accompanied by an impairment or abolition of their functions.

§ 807. *Diagnosis.*—Concussion may be distinguished from most other affections by the presence of the symptoms already described directly following a distinct injury. It is most likely to be mistaken for compression of the brain. It may be distinguished from compression by the absence of any obvious cause of pressure on the brain, of paralysis, and of stertorous breathing. In compression from hæmorrhage a short interval elapses before the symptoms are developed, and they gradually become more

profound, while in concussion they immediately follow the injury, and usually undergo progressive improvement. In compression the pulse is slow and full, while in concussion it is frequent and feeble.

§ 808. *Prognosis.*—The prognosis of concussion is always grave, even in apparently trivial cases, inasmuch as they may be followed by serious remote consequences. The prognosis is also more grave in those who inherit a neuropathic disposition, or have led dissipated lives.

§ 809. *Treatment.*—The treatment of concussion consists of absolute and prolonged rest. One of the most important rules of treatment is to abstain from giving stimulants, except in unusually severe cases, during the stage of collapse. The patient in this stage should be surrounded by warm blankets, while hot bottles may be applied to the feet. Absolute rest in a darkened room should be enjoined until the stage of reaction is passed, while the diet should be plain and unstimulating, consisting mainly of milk. During the stage of reaction a moderate purgative may be given, and ice should be applied to the head at an early period, if agreeable to the patient. Even in slight cases prolonged rest should be insisted upon in order to prevent, if possible, the development of the remote consequences of concussion.

§ 810. *Contusion of the Brain.*

Whenever the skull undergoes a sudden change of form as the result of external injury, the substance of the brain may be contused or lacerated. The contusion may be situated immediately beneath the portion of the skull where the injury was inflicted, or on the opposite side of the brain as the result of *contre coup*, or both these places may be simultaneously affected.

The morbid appearances presented in contusion of the brain consist of capillary hæmorrhagic extravasations, which are usually limited to the cortex of the brain, but may sometimes extend into its substance. When the injury is limited to a small portion of the brain, the extravasations may be so closely

aggregated that the part affected may present the appearance of a hæmorrhagic infarction. At other times the extravasations are more diffused. The brain is liable to be lacerated by loose splinters, or a depression of the bones; and when there is fracture of the skull, large portions of the brain may be disorganised.

The symptoms of contusion are always complicated by those of concussion and of compression. The diagnosis of contusion must be made, in the absence of the signs of a fracture of the skull, from the presence of symptoms indicative of a local lesion, as monospasms and monoplegia, in addition to the symptoms caused by a general injury to the brain.

The prognosis of these cases is usually serious, but not necessarily fatal.

§ 811. *Compression of the Brain.*

Compression of the brain may occur after injuries from the pressure of a fractured portion of the bones of the skull, the presence of extravasated blood, pus formed within the skull, or of a foreign body lodged there.

The patient becomes unconscious, the breathing is slow, deep, and stertorous, while the cheeks are puffed out during respiration. The surface of the body is cool at first, but soon becomes hot and bathed in perspiration. The pupils are dilated or unequal, the pulse is slow and full, the fæces pass involuntarily, and there is retention of urine. This condition of stupor sometimes alternates with paroxysms of delirium, while local spasms or paralyses are sometimes observed, but it is probable that in these cases the motor area of the cortex has been lacerated or contused.

For further information with regard to contusion and compression of the brain the reader is referred to surgical works.

CHAPTER XIII.

II. DIFFUSED DISEASES OF THE ENCEPHALON (CONTINUED).

(IV.) ENCEPHALITIS.

ENCEPHALITIS consists of primary inflammation of the substance of the brain followed by softening and in certain instances by abscess. Two kinds are usually described, namely, (1) *diffused* or *general;* and (2) *partial* or *local* encephalitis.

§ 812. *Etiology.*—The most frequent cause of acute inflammation and abscess of the brain is recent injury. Primary traumatic encephalitis is most acute when the atmospheric air is allowed to gain access to the wound, and in such cases it is associated with meningitis; but encephalitis may result from contusions of the brain in the absence of any perforating wound of the skull, and such cases often terminate in chronic abscess of the brain.

Affections of the bones of the skull, such as caries and accumulations of pus in the petrous portion of the temporal bone, may cause encephalitis, either by an inward extension of the inflammatory process or by infection. The presence of tumours gives rise to inflammation of the surrounding brain tissue, and a certain amount of encephalitis is always met with in cases of infantile apoplexy.

Multiple cerebral abscesses occur in connection with acute febrile affections, more especially typhoid fever, and are generally occasioned by metastasis from other organs. In scarlet fever abscesses of the brain result from affections of the internal ear and petrous part of the temporal bone. Localised inflammatory processes occur in the brain in measles, and

Westphal found them also in the spinal cord in variola. Circumscribed affections of the brain are met with in carbonic-oxide poisoning, but these appear to be of necrotic origin (Huguenin). Ulcerative endocarditis may give rise to abscess of the brain by multiple embolism of the cerebral vessels. Chronic putrid bronchitis and bronchiectasis are especially liable to produce secondary abscess in the brain, and unhealthy suppurations in other parts of the body may have the same result.

Encapsulated abscesses are produced for the most part by the same causes that give rise to acute abscess. Lebert found that injury was the cause of encapsulated abscess in about a sixth of his cases, Schott in thirteen out of forty, Meyer in twenty-one out of eighty-six, and Huguenin in one case out of every four. The encapsulated abscess is frequently situated at a point in the brain opposite to that where the injury was received.

Abscesses of the brain secondary to affections of the ear are situated most frequently in the hemispheres, less frequently in the cerebellum, and in very rare cases in the pons; the right hemisphere is oftener the seat of the abscess than the left. According to Mr. Toynbee, the inflammation of each portion of the organ of hearing is transmitted to a particular region of the brain. Affections of the cavity of the tympanum cause abscess of the cerebrum; those of the meatus auditorius externus induce disease of the lateral sinus and of the cerebellum; while affections of the labyrinth cause disease of the medulla oblongata. He also states that when the mastoid cells are diseased in early life the cerebrum is the part most likely to suffer; while in later periods of life, the cerebellum is generally affected.

Inflammation is transmitted in a small number of cases to the brain from the nose, Antrum of Highmore, and orbit. Polypi in the nose and frontal sinuses may cause absorption of the frontal bone, inflammation of the dura mater, and an abscess in the anterior lobe of the brain. Gull mentions two cases where the inflammation was transmitted from the nasal to the cerebral cavity. Caries of the other bones of the skull, especially that due to syphilis, also gives rise to cerebral abscess.

Suppuration occurs around tumours, but it is less frequent than red softening.

The male is more liable to abscess of the brain than the female sex, probably from the former being more exposed to injuries, and for a similar reason the greatest number of cases occur between the twentieth and thirtieth years of age. Cerebral abscess is very rare after the sixtieth year of age.

§ 813. *Symptoms.*—No general description of acute encephalitis can be given which will apply to all cases. Injuries to the head are often accompanied by contusions of the brain, which may be followed by acute localised encephalitis. Superficial lesions, provided they be exposed to the air, lead to red softening, with consecutive acute diffused suppuration of the brain; while deep contusions may be followed by red softening and suppuration, which tend to develop into chronic encapsulated abscesses. The stage of encephalitis without suppuration is generally transitory, and its symptoms are difficult to recognise, more especially as the symptoms often commence during the period of unconsciousness caused by the original contusion.

1. Diffused or General Encephalitis.

When the contusion is superficial encephalitis is complicated by meningitis, and it is impossible to distinguish the symptoms which belong to each affection.

When diffuse meningitis supervenes after injury, local symptoms such as isolated spasm, paralysis, or aphasia may occur, and a distinct spot of red softening be found at the autopsy to account for them. But when the lesion of the cortex is situated beyond the motor area, psychical symptoms of a very general character are alone produced.

2. Partial or Local Encephalitis.

An encephalitic focus in the substance of the brain may also give rise to localised motor or sensory symptoms when the pyramidal tract or the sensory peduncular fibres are affected; but a focus of acute inflammation may exist in the frontal, temporal, or occipital lobes without giving rise to any symptoms indicative of localised disease. An acute encephalitis

following an injury of the head without fracture may consequently run its course without our having a suspicion of its existence.

When a contusion in the interior of the brain has taken place, the patient first suffers from the usual symptoms of concussion, and it is only when these have disappeared that the symptoms of local encephalitis can be recognised. The patient lies in a semi-conscious condition, and when roused complains of headache and dizziness, and staggers on attempting to walk. The pupils are variable in size, generally equal and reacting slowly to light. The countenance is usually suffused, but at times turns pale, and the pulse, which was frequent and irregular during the stage of concussion, sinks to 60 or 70 beats, and the thermometer may reveal the existence of fever of remittent type.

The symptoms are at times so insignificant that after a few days the patient feels quite well, or indefinite symptoms may continue for two or three weeks.

Suddenly, however, these symptoms become more intense, the fever increases, but is still of irregular type, the dizziness and headache become more marked, vomiting is not unfrequent, the pupils are dilated and fixed, the pulse is slow, the patient falls into a condition of sopor, which may be accompanied by delirium, or may pass directly into complete unconsciousness.

As the case progresses graver symptoms appear in rolling of the eyes, transitory divergence, sudden permanent paralysis of the abducens, motor oculi, or facial nerve, and in a few cases hemiparesis or hemiplegia. Convulsive symptoms are sometimes present, usually consisting of twitchings of both hands, or there may be clonic convulsions of the limbs. In some cases a general convulsion occurs which varies greatly in duration and intensity in different cases. The sopor now grows deeper, the previously slow pulse becomes quick and irregular, and death takes place in coma. The course of the temperature varies, but a continuous elevation until death is exceptional.

The *duration* of the symptoms is variable. Beck found an abscess of the brain on the fifth day after an injury of the head, and Huguenin on the twelfth day. When the air obtains

access to a peripheral cerebral contusion, and suppuration occurs, death supervenes more rapidly.

The dangerous symptoms of traumatic encephalitis may occasionally disappear, and the patient be restored to comparative health. The inflammatory focus may be transformed into a relatively innocuous condition, but in many cases chronic changes of a diffused character are occasioned which give rise to more or less permanent symptoms. The more usual groups of symptoms caused by these changes are the following:—

(i.) Chronic psychosis in the form of irritable melancholia, followed by recovery (Huguenin).

(ii.) A psychosis characterised by severe headache, dizziness, anxiety, and hallucinations; the intellectual faculties are impaired, and there are intercurrent periods of excitement and constant illusions of the senses. A few cases recover, but in the majority this condition continues for years, and at last ends in complete imbecility.

(iii.) Symptoms resembling those of dementia paralytica supervene at a variable period of weeks or years after the injury. The development of the disease after an injury is slow, and the course is protracted.

(iv.) A psychical vulnerability frequently remains, which is apt to develop into some form of insanity from some slight exciting cause. In these cases the disposition of the patient is generally changed, there is great mental irritability and hyperæsthesia along with diminution of the power of sustained thought, and insanity may supervene many years after the injury.

(v.) Epilepsy is a frequent result of the chronic changes in the skull that follow an injury. Old depressions of the skull are often associated with epilepsy.

(vi.) Tumours of the brain have been known to follow injury (Griesinger, Recklinghausen).

(vii.) Diabetes has supervened immediately after a fall on the back of the head.

a. Acute Encephalitis, complicating affections of the petrous portion of the Temporal Bone, and of other Bones of the Skull.

§ 814. *Symptoms.* — The symptoms caused by the acute encephalitis, which accompanies caries of the petrous bone, are often obscured by co-existing meningitis and thrombosis of the sinuses. An abscess in the temporal lobe may attain a considerable size, and cause general symptoms of compression before giving rise to symptoms of local disease, inasmuch as this lobe does not contain any direct sensory or motor conducting tracts. Acute abscesses of the temporal lobe are consequently seldom recognised during life. Otorrhœa may occur at all ages, although it is most common in scrofulous children especially after attacks of scarlet fever; while a purulent discharge from the ear has occasionally been observed immediately after birth, the affection being then apparently congenital. Acute cerebral abscess from otorrhœa runs a very rapid course, its duration being from four to twenty or more days.

The symptoms are those which usually result from a sudden and progressive compression of the brain, but general convulsions may precede the development of complete coma. Fever, of variable type, is usually present, the pulse is slow, and the pupils contracted and sluggish.

Severe headache is usually the first symptom of the affection, but it is soon followed by vomiting, ringing in the ears, confusion of ideas and loss of memory, and mild delirium. As the disease advances the headache becomes more and more intense, the patient is delirious and at times unconscious, epileptiform convulsions supervene, and the case soon terminates fatally amidst profound coma.

In some cases the general symptoms just described are complicated by those of a localised disease. In such cases the abscess increases rapidly in size, and involves the base of the lenticular nucleus, compressing the fibres of the internal capsule, and thus causing an incomplete hemiplegia with various sensory disturbances. It may also compress the cerebral peduncle, and thus cause paralysis of the oculo-motor nerve, while paralysis of the facial nerve has occasionally been observed.

Cases of this kind pursue a rapid course, and terminate fatally in a short time.

A few cases are associated with acute meningitis or thrombosis of the lateral sinus.

b. Acute Pyæmic Encephalitis.

The initial symptoms of this affection are somewhat variable and often masked. There are frequently rigors, but these are symptomatic of the general disease. The brain affection is ushered in by severe headache, usually frontal, dizziness, mental disturbance, slight somnolence, occasionally delirium, unilateral convulsions in an arm or leg or both, formication and other forms of dysæsthesia, or a slight diminution of sensibility.

The disease usually makes rapid progress, and graver symptoms soon supervene. There is intense headache, dizziness is so great that the patient cannot stand or walk, the mind is confused, and delirium supervenes, but soon gives place to profound coma.

The local symptoms consist of convulsive movements of the eyes, face, or of one of the limbs, which may end in unilateral or general convulsions. There may be at times a considerable elevation of temperature, but the intensity of the febrile symptoms is variable.

c. Encephalitis around pre-existing lesions in the brain, such as tumours, necrotic softenings, and extravasations of blood.

(*a*) *Cerebral Hæmorrhage.*—Within a variable period after a cerebral hæmorrhage, a zone of red softening is found around the primary focus, in which an abundance of migrated corpuscles may be observed. Suppuration, however, is rare, if it ever occur. Beyond the area of red softening a second zone may be observed in which the tissues of the brain are unusually dense, owing to a great increase of the neuroglia corpuscles, while the tissues surrounding this zone may be extensively œdematous. Secondary hæmorrhages may occur in the circumference of the primary apoplectic focus.

(*b*) *Necrotic Softening from Thrombosis and Embolism.*—The primary focus consists of a hæmorrhagic infarct, which is

followed by inflammation of the surrounding tissues. The infarct is thus surrounded by a red areola, studded with capillary extravasations, which in its turn is surrounded by a yellowish zone, and the latter by a more or less extensive zone of œdematous tissue.

The inflammatory process around the focus, as a rule, gradually ceases, the central portion of the part affected becoming transformed into a cyst containing a clear serous fluid, and sometimes connective tissue septa, or into a number of small lacunæ containing a cloudy serum.

(*c*) *Tumour.*—The secondary softening caused by tumours of the brain is usually most marked around tumours like the carcinomata, which grow quickly.

The processes around tumours may be divided into several varieties:—

(i.) Simple softening and œdema of the surrounding tissues, caused by retardation of the circulation, and probably by thrombosis.

(ii.) Capillary and larger extravasations, probably due to fatty degeneration of the walls of the vessels.

(iii.) Genuine encephalitic red softening, accompanied by rupture of minute vessels, migration of cells, and extensive œdema of the brain.

(iv.) Suppuration around tumours is occasionally observed.

§ 815. *Symptoms.*

(*a*) *Cerebral Hæmorrhage.*—Some of the symptoms which follow a cerebral hæmorrhage must be ascribed to consecutive encephalitis. The patient may have made a good recovery from the early symptoms of an apoplectic attack, but several days afterwards there is a fresh elevation of temperature, and the pulse becomes hard and frequent. The patient complains of headache, there may be slight wandering and confusion of ideas, and he may fall into a somnolent condition. The general are soon followed by local symptoms, consisting usually of the well-known secondary contractures. Some patients may manifest only slight tremor of the paralysed limbs; in others the flexors are in a state of contracture, while in a third series of cases

these conditions alternate. The temperature of the paralysed side is often considerably elevated, and anomalies in the secretion of sweat are observed. The somnolence may now increase to a deep sopor, which lasts several days, and may pass into profound and fatal coma.

In those cases that recover symptoms frequently persist which show that a chronic encephalitis is established. There is persistent headache, frequent attacks of dizziness, and the patient is subject to congestive attacks, each of which may cause new convulsions in the paralysed limbs. The paralysed limbs are generally subject to pains of variable character, situated either in the joints, bones, skin, or muscles. Secondary encephalitis is also the chief cause of the atrophy of the brain observed in many of these patients, and which is always associated with profound psychical disturbances.

(*b*) *Thrombosis and Embolism.*—In senile encephalomalacia the symptoms of secondary encephalitis are caused by an increase of the intracranial pressure on the one hand, and irritation of the surrounding parts on the other. The symptoms of inflammatory reaction are slight; and when a certain degree of senile atrophy of the brain had existed previous to the occurrence of the attack, the mental functions become progressively abolished without being preceded by symptoms of active irritation or by those indicative of a gradual compression of the brain. It frequently happens that, after the formation of a diseased focus in the brain, a febrile condition, attended by a drowsy delirium or somnolence, continues for some time, and either develops into permanent imbecility or gives place to partial restoration of the mental faculties. When the last result occurs the mental condition of the patient is characterised by weakness of memory, irregular and causeless outbursts of temper, and a disposition to the shedding of tears, and other emotional displays. The patient is liable to congestive attacks which occasion temporary unconsciousness, and during these new foci of softening may be developed in the brain. Convulsions of the partially paralysed limbs may occur, and in rare cases general convulsions.

(*c*) *Tumour.*—A great many of the symptoms observed in tumours of the brain must be ascribed to the secondary

encephalitis in the surrounding tissue. The symptoms which may with probability be ascribed to encephalitis during the growth of a cerebral tumour are the occurrence of sudden apoplectiform attacks, the rapid conversion of slight muscular weakness into complete paralysis, partial convulsions followed by paralysis, general convulsions, and the gradual development of coma. When coma is suddenly developed in the course of the growth of a cerebral tumour, it is more likely to be caused by hæmorrhage, or sudden œdema, than by encephalitis. Every secondary encephalitis, however slight, produces violent headache, although such attacks may be due to a congestive swelling of the tumour itself. Encephalitis around a tumour which involves the sensitive fibres of the corona radiata is liable to cause disturbances of sensation in the opposite side of the body.

d. Chronic Abscess of the Brain.

Chronic abscess of the brain may be subdivided into (i.) *primary* and (ii.) *secondary* chronic abscess.

(i.) *Primary chronic abscess* is usually caused by some injury of the brain. All the symptoms, or nearly all, may disappear soon after the injury, and a period relatively free from symptoms may follow, forming the latent stage of chronic abscess. The average duration of the latent stage is, according to Lebert, from one to two months, but the period may vary in individual cases from a few days to years. When once a chronic abscess is formed, the symptoms caused by it are more or less similar to those of cerebral tumour; and when the former is situated in the motor areas of the cortex and centrum ovale, or injure the sensory peduncular fibres, the symptoms of a local lesion are present from the beginning. The symptoms may be divided into those of (1) the *latent*, and (2) the *terminal* stages.

(1) *Symptoms of the Latent Period.*

(*a*) In some cases a persistent headache subject to paroxysmal exacerbations is the only symptom present which could lead to the suspicion of the existence of an intracranial affection, and in a few rare cases this symptom may be absent.

(*b*) In other cases symptoms of a local disease appear which

make it possible to diagnosticate the *locality* of the lesion, while the history of the case and the concomitant phenomena may place the *nature* of the lesion beyond doubt. The local symptoms caused by chronic abscess are the same as those of all other focal diseases.

(*c*) In other cases the symptoms of the latent stage are those of a moderate degree of intracranial pressure. These consist of constant headache, with paroxysmal exacerbations accompanied by slight febrile disturbance, dizziness, nausea, and occasionally vomiting. The headache may be limited to the spot where the injury was received, or correspond to the part of the brain where the abscess is situated, the latter being often at a point of the brain exactly opposite the seat of injury. Paroxysmal exacerbations of the headache are indicative of congestion around the abscess, and when these frequently recur the abscess is likely to prove fatal within a brief space of time.

(*d*) In a fourth series of cases the symptoms are indicative of intermittent pressure on the brain, with intervals of comparative freedom from all cerebral symptoms. The patient, in the midst of comparative health, may suddenly complain of intense headache, he becomes somnolent, and falls into a deep but transitory coma of several hours' duration, from which he rapidly recovers. Such attacks are probably due to sudden pressure on the brain from congestion.

(*e*) The so-called latent stage is sometimes characterised by epileptiform convulsions, which may be regarded during life as true epilepsy (Hutchinson, Jackson). General convulsions are rare during this stage.

(2) *Symptoms of the Terminal Period.*

When once the terminal period begins, abscess of the brain generally leads to death in a few days. The symptoms of this period differ widely in individual cases, and the following groups may be distinguished :—

(*a*) *Terminal œdema of the brain* is the most usual mode of termination of chronic abscess. In the majority of cases the tissues of the brain are compressed to such an extent that death by coma results in two or three days, while in a few cases the course of the symptoms is characterised by temporary improve-

ments and aggravations, so that the fatal issue may be delayed for some time. In some cases there may be a transitory initial stage of irritation, characterised by mental irritability, restlessness, illusions, violent delirium, and a slight elevation of temperature. The irritative symptoms soon give place to those of depression, the patient complains of headache, the temperature falls, the pulse is slow, the pupils are dilated and react feebly to light, there is mental confusion, and the patient falls into a somnolent condition, from which he may be roused temporarily when pressed by questions; but in a short time coma supervenes, and the case soon terminates fatally, the pulse becoming small, quick, and irregular before death.

Chronic abscess in the region of distribution of the posterior and anterior cerebral arteries may run, as we have seen, their course without giving rise to marked motor or sensory symptoms. But even when the abscess is situated in one of these regions convulsions are not always absent during the terminal period. An abscess in the præ-frontal region may, for instance, give rise to partial convulsions, either limited to or beginning in the face and extending to the arm, these convulsions being followed by paralysis. General convulsions are sometimes caused by abscess situated in the latent regions of the hemisphere, and these are usually followed by profound coma. The symptoms of local disease which may have been present during the latent stage of the abscess are variously modified in the terminal stage. Partial convulsions which may have been present become more violent and are soon followed by paralysis, or they may become transformed into a series of epileptiform attacks terminating in coma.

(*b*) *Rupture of the Abscess on the Surface of the Brain.*—When abscesses make their way to the surface of the brain, an acute and rapidly fatal meningitis generally results. The symptoms of irritation are at first predominant, consisting of general convulsions and delirium. The patient, however, soon becomes unconscious, and dies comatose in a short time.

(*c*) *Perforation of the Abscess into the Ventricle.*—This occurrence causes a group of symptoms which may be recognised, if the existence of an abscess have been previously suspected. A sudden cerebral attack, attended by bilateral, but

more or less partial convulsions, such as spasms of both legs, or of the facial muscles on both sides, is an indication of rupture into the ventricles, provided the patient be not already in an unconscious condition. General convulsions have sometimes been observed. Clonic spasms of the ocular muscles soon appear, caused probably by irritation of the corpora quadrigemina. The patient becomes rapidly unconscious, hemiplegia and death in profound coma take place generally in from four to twenty-four hours after the rupture of the abscess.

(*d*) Abscess of the cerebellum may terminate suddenly from arrest of the respiratory functions produced by pressure on the medulla oblongata.

(*e*) Occasionally the brain is found in a condition of remarkable anæmia, and in such cases the immediate cause of death is not evident.

(ii.) *Secondary Chronic Abscess of the Brain.*—Secondary chronic abscesses are generally caused by affections of the inner ear. The diagnosis of the presence of chronic abscess of the brain is difficult, inasmuch as only a small proportion of such cases give rise to characteristic symptoms. When the abscess is encapsulated it may remain latent for a long period, so that no disease of the brain is suspected until the terminal period. Even the terminal symptoms present varieties which tend to obscure the diagnosis, these symptoms sometimes resembling those of diffuse meningitis, and at other times those of thrombosis of a sinus.

§ 816. *Varieties.*—The following varieties of chronic abscess secondary to disease of the ear may be distinguished:—(*a*) Chronic abscess with distinct typical course; (*b*) Chronic abscess with terminal stage alone distinct; (*c*) Chronic abscess with thrombosis of the lateral sinus; (*d*) Chronic abscess complicated during the terminal period by meningitis.

(*a*) In affections of the inner ear abscess may form in the temporo-sphenoidal lobe. In a long-standing case of disease of the internal ear, where rigors and other general inflammatory symptoms are associated with severe pain in the head, vomiting, convulsions, and other cerebral symptoms, the forma-

tion of an abscess in the brain may be suspected. These symptoms may pass off, and the patient enjoy apparent health for months, with probably occasional headaches. The terminal stage is announced by intense headache and dizziness soon followed by loss of consciousness and stertorous breathing. Consciousness may be partially restored in a few hours, and the patient then suffers from intense headache and vomiting. After a short time the patient lapses a second time into a semi-conscious condition, and convulsions, generally unilateral, supervene. Spasm followed by paralysis of the ocular muscles is not an unfrequent symptom, and when the abscess is so large that it extends to the lenticular nucleus and compresses the internal capsule, or the fibres of the pyramidal tract in the crusta, a certain degree of hemiplegia may be present.

(*b*) Chronic abscess of the brain is sometimes observed in cases of caries of the petrous bone, in which the terminal symptoms have not been preceded by those indicative of irritation or encephalitis.

(*c*) Chronic abscess of the brain sometimes precedes, at other times succeeds to thrombosis of the lateral sinus. The chief initial symptoms are, besides those of the ear affection, dizziness, intense headache, and occasionally transitory delirium, followed by somnolence. The patient suffers from frequently-repeated rigors if the temperature of the body be raised, and the fever assumes a remittent type. In the further progress of the case the symptoms may pursue either of two directions. The symptoms may be those of progressively increasing pressure upon the brain, ending in coma, or the general symptoms indicative of compression may be associated with those of a localised disease, provided the abscess has attained a sufficient size to press upon the internal capsule. General convulsions may occur immediately before death.

(*d*) Chronic abscess of the brain may be complicated during the terminal period by meningitis, and when the initial stage of the former is latent, the terminal symptoms may be so similar to those of primary acute meningitis that the two affections cannot be distinguished from one another during life.

§ 817. *Morbid Anatomy.*—In encephalitis the affected tissue assumes a reddish colour, and is studded by a number of capillary extravasations, each about the size of a pin's head, these being sometimes so numerous that the affected part presents the appearance of a hæmorrhagic infarct. These capillary extravasations are followed by more or less œdema, which may extend for a considerable distance into the surrounding tissues. In consequence of this œdema the affected portion of the brain becomes voluminous, and the cut surface rises above the level of the surrounding tissues, the latter of which assume different shades of colour from the imbibition of the colouring matter of the blood set free in the central part of the effusion. When the inflamed focus presents a deep red colour it is surrounded by a red zone, which shades off into brown, then into yellow, and finally into the normal colour of the cerebral tissue.

The *microscopic* changes observed in the first stage of inflammation are great hyperæmia and dilatation of the vessels and capillaries. Hayem asserts that he has seen the vessels dilated to six times their normal calibre. The vessels are surrounded by migrated white blood corpuscles and the tissues are infiltrated by leucocytes, probably derived from multiplication of the nuclei of the neuroglia and proliferation of the cellular elements of the walls of the vessels. A large number of granule cells (Gluge's corpuscles) may also be observed in the inflamed focus. These are probably derived from the ganglion cells, the nuclei of the neuroglia, the nuclei of the capillary vessels, and the endothelial cells of the sheaths of the vessels. The large size and granular appearance assumed by these cells are supposed by Hayem to be due to the absorption of nutriment and to be analogous to the cloudy swelling of Virchow, but the granular appearance is at least more likely to be caused by commencing degeneration. The ganglion cells swell up, their protoplasm undergoes molecular disintegration, and after the inflammation has ceased they undergo various degrees of atrophy and sclerosis, and in chronic cases the calcification and pigmentary infiltration described by Förster.

§ 818. *Further Transformations of the Primary Focus.*

The primary focus undergoes various changes according to the extent and situation of the inflammation. In a large proportion of cases the

inflammatory process ceases before an abscess forms, and the affected part presents a strong resemblance to a primary necrosis with subsequent peripheral encephalitis. The resulting conditions are similar in the two affections, although they are essentially different in nature.

(1) An encephalitis of slight intensity and small extent, such as that caused by traumatic contusion, may undergo complete repair.

(2) After the inflammatory process in the larger foci has ceased a residuum is left behind which undergoes the well-known destructive changes, followed by absorption of the fluid contents. All the cellular elements in the focus are transformed into granule cells, which undergo a gradual disintegration; the contents of the focus becomes thus converted into a thick emulsion, coloured brownish or yellowish by the blood pigment. All the nuclei of the vessels and the neuroglia, and the white blood cells, which are enclosed in the focus, disappear, and its contents become more homogeneous. After a time a focus of yellow softening forms which gradually becomes more colourless, and at last may be transformed into a cavity filled by a thin milky fluid.

(3) But the focus after a time manifests a delicate stroma supplied with delicate vessels, the interspaces of which are filled by a thin turbid fluid. The stroma consists of delicate connective tissue supplied with vessels; the formed elements of the fluid consist almost wholly of large quantities of granular fat and albuminous bodies, together with a little free pigment. The spaces become gradually larger and the fluid clearer, so that a chasm remains which is traversed by a number of delicate septa of connective tissue, and surrounded by somewhat condensed cerebral substance. Apoplexy and infarct may terminate in the same way, and the nature of the preceding affection cannot be positively determined from the study of the lesions in their later stages.

(4) Local encephalitis may lead to the production of firm sclerotic cicatrices situated usually near the surface of the brain, more rarely deep in the interior of the organ. These cicatrices are of a dirty-white colour, tough, and firm; the tissue surrounding them is atrophied, so that the affected hemisphere is less than the other. Even distant portions of the brain, especially of the cortex, may be found in a state of atrophy. When the cicatrices are situated deep in the brain, cavities are found within them at an early period. At a later period the cicatrix contains a nucleus differing from the rest, and containing fat and pigment granules, hæmatoidin crystals, and amorphous detritus.

Such inflammatory processes in the brain very seldom become quiescent; they are followed by a gradually progressing atrophy of the entire brain, which causes symptoms during life that even at the present day are frequently included among those of dementia paralytica. Hasse has also drawn attention to the fact that an encephalitic cicatrix may excite fresh inflammation at a later period resulting in the development of a new zone

of red softening with capillary apoplexy. It may also excite a rapidly progressive yellow softening, which may prove fatal.

The inflammation excited around embolic foci may give rise to sclerotic capsules, consisting of a dense fibrous connective tissue. Huguenin examined one that contained a large number of spindle cells with oblong nuclei, which he surmised to have been derived from migrated white blood corpuscles.

(5) The encephalitic focus may be transformed into a collection of pus, which may be subdivided into recent and old abscesses. An abscess is said to be new or fresh when it has been developed rapidly and does not possess a capsule, but this distinction does not hold good in all cases. An acute abscess has a tendency to spread in every direction and it presents an irregular cavity in the substance of the brain, whose walls have a rough, shaggy surface. The shaggy projections consist of shreds of cerebral tissue, which are attached to the larger blood-vessels. The tissue around the abscess is in a condition of red softening, and in many places the softened tissue is of a predominantly yellow colour; while at a greater distance from the focus the cerebral tissue is œdematous. As the pus in the central cavity accumulates pressure is exerted on the whole of the surrounding tissue, which arrests its circulation and leads to further destruction, and advance of the abscess.

The abscess by virtue of its tendency to enlarge may reach the surface of the brain, and as soon as perforation occurs an acute purulent inflammation of the pia mater results.

(6) *Old abscesses of the brain* possess a fibrous capsule which may attain a thickness of several millimetres. Rindfleisch affirms that there is a gradual transition from the capsule to the surrounding nervous tissue, but the connection is not always very close, inasmuch as the abscess can be enucleated without much difficulty. The internal surface of the limiting membrane is smooth, and an opaque yellowish-white appearance is given to it by a continuous layer of cells in a state of fatty degeneration. Outside this there is a layer of embryonic tissue.

The pus of the abscess is of a greenish colour, and greasy consistency, is odourless, and has an acid reaction. After a variable period of quiescence the abscess enlarges and produces manifold changes in surrounding parts. The intracranial pressure becomes increased, the nervous tissue in the vicinity of the abscess may be compressed so that its nutrition is arrested, and as a result there follows more or less extensive yellow softening. Local inflammatory processes in more distant parts are met with.

Accessory Changes in the Brain.—Perforations on the surface of the brain and into the ventricles occur in connection with encapsulated as with recent abscesses.

Extensive acute œdema of the brain often results from abscess, and is probably due to the increase in the intracranial pressure.

Anæmia of the brain, and more especially of the cortex, also results from the increase of the intracranial pressure.

Chronic internal hydrocephalus results whenever an abscess is situated in the cerebellum in such a position that it lessens the cavity of the fourth ventricle or of the Sylvian aqueduct.

§ 819. *Diagnosis.*—The symptoms of abscess of the brain are very similar to those of tumour, but the two affections may generally be distinguished by the history of the case and the progress of the symptoms. Abscess is more frequently preceded by a distinct history of injury to the head than tumour, although the latter also occasionally develops soon after injury to the skull. The course pursued, however, by the two affections subsequent to the injury, differs widely. If the symptoms of acute encephalitis occur immediately after the injury, and be then followed by a remission or complete intermission and break out again after a latent period of variable duration, either with or without the phenomena which indicate a local disease, abscess may be diagnosticated rather than tumour.

If the other causes which give rise to abscess of the brain, as chronic otorrhœa, caries of the temporal, frontal, or nasal bones, bronchiectasis and purulent cavities in the lungs with putrid secretions, and pyæmia are present, along with the symptoms of a localised lesion of the brain, then also the existence of an abscess rather than tumour may be inferred.

The symptoms of tumour are sometimes characterised by frequent remissions and exacerbations, while in abscess the symptoms may be latent or stationary for a comparatively long time, but when the terminal period is ushered in the remissions are of short duration and never frequently repeated. In many cases of tumour the course of the disease is continuous and progressive from the first, and the symptoms increase not only in intensity but in number and extent, the general symptoms of headache, dizziness, and vomiting of the first period becoming slowly complicated by local spasms, unilateral convulsions, pareses passing on to distinct paralyses, sensory disturbances, slight at first but becoming more profound, and various disorders of the special senses. Although these symptoms may all be present at a given time in the course of chronic abscess, yet they never appear in the same

progressive manner in the latter affection as they do in tumour. Repeated chilliness and slight elevation of temperature are more characteristic of abscess than of tumour, but it must be remembered that the latter is liable to be complicated by attacks of acute encephalitis.

In the absence of a distinct history of the case, it may be impossible to distinguish the terminal period of abscess of the brain from cerebral hæmorrhage, or occlusion of a large vessel; but if there be a history of injury to the skull, followed by the symptoms of acute encephalitis, then the diagnosis is less difficult. This difficulty is likely to arise when, after a long latent period, the abscess makes its way into the ventricles, or when sudden œdema of the brain occurs. When the abscess ruptures on the surface of the brain the terminal symptoms are those of acute meningitis, and the diagnosis between primary meningitis and abscess must again be made from the history of the case and the symptoms.

Abscess of the brain is difficult to distinguish from necrotic softening. The diagnosis must be made on the one hand by the history of an injury to the skull or the presence of one of the other causes which give rise to abscess; and on the other hand by a careful general examination of the patient, especially of the organs of circulation, to ascertain the presence or not of the conditions which lead to embolism or thrombosis.

§ 820. *Prognosis.*

(*a*) *Meningo-Encephalitis.*—A superficial contusion of the brain usually terminates in acute suppuration associated with meningitis, and the affection is generally fatal. Recovery is possible only when diffuse meningitis does not take place, and when the pus is discharged through a wound, produced either by the original injury or by surgical operation.

(*b*) *Suppurative encephalitis* without accompanying meningitis, resulting from contusion of the brain, does not appear to be capable of spontaneous absorption. Recovery, however, may take place either from spontaneous perforation externally or from artificial evacuation. Acute abscess may, instead of at once terminating fatally, pass into the chronic form.

(*c*) *Chronic traumatic abscess of the brain* is, as a rule, fatal.

A small number of cases have recovered spontaneously after perforation through the skull. The pus has been evacuated by fortunate trephining, but the number of cases in which the patient was saved is small.

(*d*) *Otorrhœal Abscess of the Brain.*—Several cases of recovery of otorrhœal cerebral abscess are on record in which the pus made its way through the diseased ear. Acute otorrhœal abscess of the brain often passes into the chronic form, but the ultimate prognosis in both varieties is unfavourable.

(*e*) *Pyœmic Abscesses* of the brain, whatever may be their cause, are always fatal.

(*f*) *Acute Traumatic Encephalitis without formation of Pus.*—Cases of recovery from undoubted traumatic encephalitis have been collected by Bruns. The prognosis of encephalitis around chronic abscesses, tumours, chronic softening, and apoplexies depends upon the nature and extent of the primary disease. The prospect is least favourable in abscess and tumour. The age and strength of the patient is an important factor in estimating the danger of encephalitis secondary to necrotic softening and cerebral hæmorrhage, but the complication is always a serious one, and even if the patient survive the general and local symptoms are usually aggravated by an increase in the destruction of tissue caused by the primary focus.

§ 821. *Treatment.*—The physician's advice may be sought to aid the surgeon in determining questions of cerebral localisation, and the other delicate points of diagnosis which are likely to arise in the progress of such cases, but the decision with regard to the treatment to be adopted must rest with him.

The secondary inflammation which is liable to supervene in the course of necrotic softening, cerebral hæmorrhage, and intracranial tumours is the form of encephalitis which is most likely to come under the care of the physician. This variety is best treated by complete rest in a darkened room, mild purgation, and cold applied to the head ; more active measures like bleeding and blistering are worse than useless.

Chronic abscess of the brain does not admit of any special medical treatment, but the general health of the patient must be attended to, and his diet and habits carefully regulated.

Paroxysms of severe headache may sometimes be relieved by chloride of ammonium, while more active symptoms like delirium may be combated by bromide of potassium, either alone or in combination with chloral, and an opiate may sometimes be found useful. Drs. Russell Reynolds and Hammond speak favourably of Cannabis Indica in the treatment of the more active symptoms of suppurative encephalitis.

CHAPTER XIV.

DISEASES OF THE MEMBRANES OF THE BRAIN.

I. DISEASES OF THE DURA MATER.

Inflammation of the dura mater may be divided into (i.) external, and (ii.) internal pachymeningitis.

(i.) External Pachymeningitis.

External pachymeningitis consists of inflammation of the outer lamella of the dura mater.

§ 822. *Etiology.*—The chief causes of the affection are the following:—

1. Injuries which detach the dura mater from the inner surface of the skull, and occasion an extravasation of blood between them. The clot may be so large as to compress the brain and cause death in a short time. At other times inflammation is set up in the surrounding tissues and the bone is threatened with necrosis.

2. Perforating injuries of the skull, as incised, punctured, and bullet wounds, which either directly or indirectly injure the dura mater.

3. Extension of inflammation from neighbouring tissues. Caries of the petrous portion of the temporal bone is one of the most frequent causes of external pachymeningitis, and it usually gives rise to the suppurative form of inflammation.

Purulent inflammation of the external lamella may also follow caries of other cranial bones, especially the ethmoid and the flat bones of the skull, as well as by caries of the upper cervical vertebræ and their ligaments.

4. The external layer of the dura mater is subject to chronic fibrous thickening in old age, and becomes adherent to the bone.

§ 823. *Symptoms.*—In the traumatic form of external pachymeningitis the patient may recover from the immediate effects of the injury, and appear for a time in perfect health. At the end of two or three weeks he complains of pain in the head, is feverish, and should there be an external wound, it assumes an unhealthy aspect. If the inflammation spread further, the headache increases in intensity, and the patient suffers from vertigo, nausea, and vomiting, while monospasms or unilateral convulsions may occur if the motor area of the brain be implicated. The internal membranes of the brain now become affected, the previously convulsed limbs become paralysed, the patient becomes delirious, and fatal coma soon supervenes.

When an abscess forms between the dura mater and the bones of the skull, the symptoms may be those of gradual cerebral compression, and when it is situated over the motor area of the cortex, monospasms or monoplegiæ may result.

The pachymeningitis of old age is often discovered after death without having been suspected during life. In other cases the meningitis may have manifested itself merely by a persistent dull headache. After death atrophy of the brain, compensatory hydrocephalus, and serous infiltration of the pia mater are observed.

§ 824. *Morbid Anatomy.*—The dura mater is at first congested, and presents punctiform extravasations. In a more advanced stage the membrane becomes swollen and infiltrated with numerous white blood corpuscles. These cells may after a time become transformed into spindle cells, and ultimately developed into bundles of connective tissue. Portions of the membrane may sometimes become ossified.

If the inflammation progress to actual suppuration, the white blood corpuscles become more numerous, and make their way through the internal lamella to the free surface, so that a purulent internal pachymeningitis is added to the external pachymeningitis. At times, however, the abscess is cut off

from the internal layer by the development of adhesions, and in that case the external layer becomes disintegrated, soft, and friable; while the internal layer becomes adherent to the pia mater and brain. In the large majority of cases the inflammation spreads to the internal lamella.

§ 825. *Prognosis.*—The prognosis in traumatic cases turns on the possibility of the pus finding a free escape. If a free discharge can be obtained, the affection of the dura mater may occasion but little trouble; while, on the other hand, if the matter is pent up, grave symptoms must result, both on account of the increased intracranial pressure, and the rapidity with which the suppurative process spreads. The prognosis of purulent inflammation of the dura mater resulting from caries of the petrous portion of the temporal bone is very unfavourable.

§ 826. *Treatment.*—The treatment of the acute stage must be conducted according to general principles. The question of trephining will arise in connection with the formation of an abscess or extravasation of blood between the dura mater and the bone, but this belongs to surgery.

(ii.) Internal Hæmorrhagic Pachymeningitis.
(Hæmatoma of the Dura Mater.)

§ 827. *Etiology.*—Hæmatoma is associated with all those diseases which profoundly affect nutrition, and many of them are diseases like scorbutus, in which hæmorrhages are liable to occur in other parts of the body. The disease is found in atrophy of the brain accompanied by œdema of the pia mater and hydrocephalus internus. On the whole, therefore, it seems probable that when hæmatoma of the dura mater does not result from direct injury, it is caused either by constitutional disease producing profound alteration in the quality of the blood, degeneration of the vessels of the brain, or by diseases associated with passive congestion of the brain. It is not therefore surprising that the affection should occur chiefly in old age.

§ 828. *Symptoms.*—The symptoms of hæmatoma vary greatly in different cases, but the following are the more usual:—

Headache is a very constant symptom, being rarely absent. The pain is described as if something were moving about in the head, or as a throbbing accompanied by a sensation of pressure in the head. The pain is not accurately localised, but is sometimes more violent on the side of the hæmatoma. It is much increased when a fresh hæmorrhage occurs; but if there be great atrophy of the brain, as in dementia paralytica and in cases of senile atrophy, a hæmatoma may attain a considerable size without causing much headache. On the other hand, when there is no shrinking of the brain a thin layer of blood causes violent pain.

The *motor disturbances* consist of muscular twitchings of one or both sides, followed by rigidity. In other cases, paresis first of one and then of the other extremity of the same side occurs followed by distinct hemiplegia, the facial and hypoglossal nerves becoming involved. In some cases the paralysis extends to the other side, showing either that the affection has spread to the opposite hemisphere, or that a fresh hæmorrhage has occurred. Impairment of co-ordinate movements frequently occurs, as uncertainty of gait, difficulty in writing and speaking. Conjugate deviation of the eyeballs towards the side of the lesion is not unfrequently observed, but strabismus and ptosis rarely if ever occur.

Sensory disturbances are not very common symptoms. Patients sometimes complain of formication and numbness on the paralysed side, but impairment of sensation in the absence of paralysis is not met with.

Psychical disturbance occurs in a large number of cases. This is sometimes due to the primary disease, as in dementia paralytica, while in other cases it is due directly to the hæmorrhage. Symptoms of irritation are present in the beginning, evinced by great mental irritability, abnormal sensitiveness to light, and ringing in the ears, but these soon give place to those of compression. In other cases the disease begins by a regular attack of apoplexy.

Drowsiness is an important symptom, and Griesinger has called attention to its occurrence in connection with contracted pupils and chronic headache, but at times dilatation of the pupils is observed.

Slowness of the pulse occurs in the majority of cases during the hæmorrhage, but the quickness of the pulse varies, and it becomes frequent and irregular before death.

The *pupils* are generally contracted and insensible to light during the irritative stage, but dilatation predominates when the symptoms of compression supervene.

§ 829. *Varieties.*—The symptoms may be subdivided into several groups, according to the extent and localisation of the hæmorrhage and the diseases with which it is associated.

1. In the first group the hæmorrhage is so severe from the beginning that death soon occurs, and in these cases the symptoms cannot be distinguished from those of ordinary apoplexy. Contractures of the extremities and slight transitory twitches are sometimes observed in these cases, and, as a rule, the pupil is more contracted and fixed in hæmatoma than in intra-cerebral hæmorrhage. In a small number of cases the extravasations on one side cause convulsive movements of the opposite side; but the compression soon becomes so great that the irritability of the cortex is abolished.

2. In a certain number of cases extravasations are found after death, though there had been no suspicion of their existence during life. These hæmorrhages, however, are usually small, and are generally found in cases of dementia paralytica.

3. In a third series the symptoms are at first slight, but gradually increase in severity and soon prove fatal. The chief symptom in the beginning is severe cephalalgia, with or without vertigo, followed by drowsiness increasing gradually to sopor, and ending in profound coma. The pupils are contracted in the early stage, but when coma supervenes they gradually dilate and may become unequal. Hemiparesis generally shows itself on the side opposite to the lesion, while symptoms of irritation may appear on the same side, but after a time all the extremities may be paralysed. Similar symptoms are caused by any meningeal affection, and especially by tubercular meningitis. In establishing a diagnosis, therefore, the conditions under which the disease has developed must be taken into account. Hæmatoma is more likely to be present in old persons, when atrophy of the brain is to be suspected; tubercular meningitis, on the other hand, usually occurs in young persons.

4. In a fourth series of cases recovery takes place from a first hæmorrhage, but after an interval of apparent health a second occurs which results in death. In these cases unilocular or multilocular sacs are found on one or both sides, and one of the subordinate sacs always contains a considerable effusion of blood which has caused death by compression.

§ 830. *Course, Duration, and Terminations.*—The course of the affection is extremely variable, but it usually begins with more or less acute symptoms, followed by an interval of comparative health; after a time, however, a fresh acute attack occurs which may lead to death or be followed by a second interval.

During the interval the symptoms are those indicating a lesion which produces a certain amount of irritation along with compression of the brain. These consist of cephalalgia, diminution of intelligence, impairment of memory, drowsiness, partial paralyses, disturbances of speech, and sudden mental excitement without cause, frequently mixed with symptoms of dementia paralytica.

The *duration* of the affection is not well known, inasmuch as it is not always possible to fix with accuracy its commencement. The majority of cases of pachymeningitis end fatally, but recovery may take place in many cases, only traces of the affection remaining, such as a slight degree of paresis, headache, sleeplessness, and some weakness of intelligence.

§ 831. *Morbid Anatomy.*—Hæmorrhagic pachymeningitis presents itself as an organised mass situated between the dura mater and surface of the arachnoid, and presenting different appearances in different cases. Various opinions have been entertained with respect to the seat and nature of the affection, but the explanation which was first given by Virchow is now pretty generally accepted by pathologists. According to this view the pachymeningitis begins with hyperæmia of the dura mater, occupying generally the area supplied by the middle meningeal artery. The inner surface of the dura mater assumes a rosy colour, and after a time a loose yellowish coating forms, which is dotted with a number of confluent or separate hæmorrhagic points.

This coating can be stripped from the dura mater, tearing many small vessels which enter its substance. Rindfleisch says that this membrane is rich in vessels, which are usually three times as large as capillaries and present varicosities. The basis substance between the vessels is composed of star-shaped connective tissue cells. Emigrant corpuscles pass from the

blood-vessels of the sub-epithelial layer of the dura mater, and develop into a loose connective tissue. Hæmorrhages, varying in quantity at different times, take place from the vessels which enter the false membrane; the clots become partially organised, and the delicate capillaries which develop in them become the source of new hæmorrhages, so that a large quantity of blood may in this manner be poured out between the thickened membranes. It is right, however, to state that Huguenin believes that the first stage of hæmatoma is not the formation of a false membrane, but simply an extravasation of blood on the inner surface of the dura mater, which undergoes the changes which usually take place in a coagulum. This coagulum undergoes partial organisation so as to form a vascular layer, from which hæmorrhage takes place, giving rise to further extravasation, which in its turn becomes organised.

Pachymeningitis hæmorrhagica is most frequently found in the upper part of the brain along the falx cerebri, spreading down the curved portion of the frontal and occipital lobes, and laterally towards the Sylvian fissure. Kremiansky found that in fifty-four out of sixty-five cases its extent exactly corresponded to the parietal bones. In cases of dementia paralytica, in which the brain is frequently found shrunk and atrophied, the blood often extends much further, and may reach the base of the brain. In rather more than half of the cases described the hæmorrhage extended over the surfaces of both hemispheres, while in the remainder it was limited to one hemisphere.

Changes in the skull have been described by various authors, but none of them are constantly present; some of these, such as elevation, thinning, and thickening of the bones, are in all probability anomalies, which are entirely independent of the hæmatoma. Osteophytes on the inner surface of the skull have been described by Rokitansky and Cruveilhier.

The pia mater is often the seat of changes, such as are found in atrophy of the brain, consisting of slight opacities, œdema, and small fibrous thickenings.

The substance of the brain is variously affected according to the thickness of the hæmatoma. The thin extravasations and membranes do not cause any marked changes in the brain, but large hæmatomata compress it, and produce consecutive anæmia and atrophy.

The *brain* is often found atrophied and contracted independently of the hæmatoma. The decrease in the size and weight of the brains of drunkards, and senile atrophy are important factors in the production of hæmatoma. The affection is frequently associated with atheroma and calcification of the intracranial arteries. Diffused sclerosis and the chronic degeneration which accompanies dementia paralytica are often associated with hæmatoma. A glance at all the varied changes found associated with hæmatoma will show that this affection in the great majority of cases occurs along with alterations which occasion a reduction in the size of the brain.

§ 832. *Prognosis.*—The prognosis depends in great measure upon the fundamental affection which is present along with the pachymeningitis. It is always grave, although not necessarily fatal.

§ 833. *Treatment.*—The treatment of hæmatoma of the dura mater will greatly depend upon the underlying affection, and it it is consequently desirable to examine carefully for disease of other organs. If venous stasis be present, a small bleeding or a smart watery purgative gives temporary relief to the circulation, but energetic antiphlogistic treatment must be carefully avoided.

During the stage of hæmorrhage ice should be applied to the head; but counter-irritation, if used at all, should be reserved for a later period. The patient must, of course, be kept quiet and all excitement prevented.

CHAPTER XV.

DISEASES OF THE MEMBRANES OF THE BRAIN (Continued).

II. DISEASES OF THE PIA MATER.

Inflammation of the Pia Mater (Leptomeningitis).

Hyperæmia of the pia mater is always accompanied by congestion of the brain, and does not demand separate notice. Attempts have been made to distinguish inflammations of the visceral layer of the arachnoid from those of the pia mater, but anatomists are now agreed that the former membrane is only the thickened external layer of the latter, and clinical records show that in the cases of so-called *arachnitis* the internal layer of the pia mater is always affected.

1. Leptomeningitis Infantum (*Hydrocephalus sine Tuberculis*).

Acute inflammation of the pia mater may occur in infancy in the entire absence of tubercle, and the simple like the tubercular variety is attended by effusion into the ventricles of the brain. Acute ventricular effusion from simple inflammation of the pia mater is most common in children between one and two years of age, but sometimes occurs in younger and sometimes in older children.

§ 834. *Symptoms.*— The clinical history of this affection may vary in no important particular from that of tubercular meningitis, but the premonitory symptoms are not so well marked in the former as in the latter.

The symptoms of the period of invasion differ considerably

in individual cases. Some cases begin with slight fever, which may be symptomatic of the meningitis or of another affection, and the febrile condition may be ushered in by an attack of convulsions. In meningitis the symptoms of intense cerebral hyperæmia are soon superadded to those of ordinary pyrexia. In other cases violent headache is the first symptom, and vomiting is frequently present. The child is restless, the eyelids are only half closed during sleep, the eyeballs roll about, the pupils are contracted but react well to light, and there may be slight convulsive twitching of the extremities but there is no paralysis. The children are abnormally sensitive to light and sound, and the lightest touch on the skin may cause pain. Older children are either unable to stand, or totter when they attempt to walk, they complain of buzzing in the ears, are fretful, morose, and taciturn. In younger children the fontanelles may be seen to pulsate strongly, but they are not arched. The countenance has a vexed or angry expression. The features are at times distorted, the forehead is wrinkled, and a distressing moan is frequently uttered.

These symptoms may continue for two or three days, and then the child may be attacked by convulsions. These begin by conjugate deviation of the eyes and rotation of the head, the upper and lower extremities of one side are chiefly affected, and then the spasms cross over to the opposite side, when the convulsion becomes general. The state of the pupils vary, but they are generally dilated and fixed during the convulsions. The temperature rises during the attack, and may exceed 104° F.

Death may occasionally occur suddenly during the attack, and when the convulsion is not fatal the subsequent course of the affection is marked by an aggravation of the cerebral symptoms. Young children lie in a disturbed sleep, with the eyelids tightly closed, and showing evidence of pain by wrinkling of the brow and moaning, but the pulsation of the fontanelles may now diminish owing to the distension of the ventricles by effusion. Older children lie for hours in a state of stupor, they are listless and indifferent, and do not appear to see distinctly, or they may be absolutely blind. The pulse is very variable in rate at different times. The respirations are sighing and

irregular. The temperature continues more or less elevated, but its course is very irregular. Persistent vomiting and obstinate constipation are common, while the power of deglutition may be impaired at an early period.

The child rapidly emaciates, the skin is dry, with the exception of that of the face, which may be bathed with perspiration.

The patient now sinks into a condition of profound coma, but tetanic spasms of the muscles of the neck and extremities may persist for a time.

The temperature often falls below normal before death, but hyperpyrexia has been observed in rapidly fatal cases. The pulse then becomes very rapid, irregular, and intermittent.

§ 835. *Course, Duration, and Terminations.*—Complete recovery may take place, probably even after effusion has occurred, but as a rule recovery is partial, and the patient subsequently suffers from mental feebleness or depraved moral character. In the latter case the cortex of the brain has probably undergone some degree of atrophy from the pressure of the effusion. The majority of cases terminate fatally in from nine to fourteen days, some die at an earlier period in an attack of convulsions, while in other cases the disease may be protracted beyond thirty days, its course being marked by remissions and exacerbations.

§ 836. *Morbid Anatomy.*—The cranial bones present different degrees of congestion, and the fontanelles are distended. The convolutions of the brain are flattened and the sulci obliterated, owing to the pressure exerted by the distended ventricles. The fluid is never found between the dura and outer layer of the pia mater, and the outer surface of the latter is usually remarkably dry. The cortex and white substance are compressed and but moderately filled with blood, and no capillary extravasations are found in the cortex. The dilatation of the ventricles is usually symmetrical, and a considerable amount of softening not unfrequently exists around the ventricles, but this may be due to post-mortem changes.

The choroid plexuses are unusually voluminous, they often

contain punctiform extravasations, and the aqueduct of Sylvius and fourth ventricle are often dilated and distended with fluid. No exudation is found at the base of the brain.

§ 837. *Prognosis.*—The prognosis is always unfavourable, although a few cases recover either partially or completely.

§ 838. The *diagnosis* will be discussed in connection with tubercular meningitis, and the treatment of the two affections is the same.

2. Tubercular Meningitis (*Acute Hydrocephalus*).

§ 839. *Etiology.*—Most of those who suffer from tubercular meningitis belong to families in which the tubercular diathesis is distinctly marked. The influence which improper nourishment, want of pure air and light, exposure to cold and damp, and neglected hygiene exerts in the production of tubercular affections generally is well known. It is not surprising, therefore, to find that the largest proportion of cases of tubercular meningitis should occur in crowded populations and large cities, and amongst the poorest and most neglected part of the population. Season does not appear to exert any influence in the production of the disease. Tubercular meningitis may set in at any age, but it is much more frequent between the ages of two and seven years. The numbers diminish from the seventh to the tenth, and in still greater proportion from the tenth to the fifteenth. It is most common in adults between the ages of twenty and forty, and occurs very exceptionally after the forty-fifth year. The male sex appears to be more frequently affected than the female. In adults the proportion is 51·5 men to 48·5 women (Huguenin); and the proportion of males affected in children is still greater.

§ 840. *Symptoms.*—Various premonitory symptoms manifest themselves for a variable period of weeks or months before the development of the distinctive phenomena of tubercular meningitis. The most constant precursor of the affection is a gradual loss of flesh without any perceptible cause, and this is more noticeable in the trunk and limbs than in the face. There is loss of appetite, the bowels are constipated, or diarrhœa may

alternate with constipation, and the patient complains of weariness and an undefined feeling of illness.

The premonitory symptoms are better marked in the case of children. The child loses his vivacity, becomes sad, fretful, taciturn, irritable, and wants to be left alone, and in the midst of play leaves his companions in order to give vent to his distress in tears. The child sleeps with eyes half open, starts, and cries out; he grinds his teeth, and his sleep is disturbed by muscular twitches and horrible dreams.

Headache is generally present, and after a time a slight febrile accession is observed towards evening without obvious cause. In some cases the disease begins with head symptoms of an inflammatory nature in the midst of apparently good health. In these cases it may be presumed that cheesy foci exist in some part of the body in a latent condition, and that the vessels of the pia mater become rapidly and suddenly invaded at the time of the outbreak of acute symptoms.

Premonitory symptoms may also be absent when the membranes of the brain become secondarily affected in persons who are already suffering from acute general tuberculosis, or from local tuberculosis of the lungs, peritoneum, or other organ. Under these circumstances the patient may have been sleepless, restless, feverish, or delirious, before the pia mater is implicated, so that the brain symptoms make their appearance without any warning. The first symptoms which indicate an affection of the pia mater in these cases may be violent headache, vomiting, or facial paralysis, followed quickly by unconsciousness.

As the invasion approaches, all the premonitory symptoms, when present, become more pronounced. Some cases are ushered in by a chill, accompanied by a rapid rise of temperature, while in others there are distinct shiverings, violent headache, attacks of giddiness and vomiting. The disease itself presents (*a*) an initial period in which the symptoms may be referred to excitation of the cortex; (*b*) a period in which symptoms caused by excitation are mixed with those due to progressive pressure caused by effusion and to lesions at the base of the brain; and (*c*) a final period in which symptoms due to pressure caused by the ventricular effusion greatly predominate.

(*a*) *The Period of Invasion.*—Vomiting is the most common special symptom of the period of invasion. It varies greatly in frequency, but as a rule occurs only twice in twenty-four hours during the first two or three days of the disease. In some few cases, however, the child vomits incessantly, whether food is taken or not. When once the tendency to vomit has ceased for twenty-four hours it does not ordinarily recur. Constipation is present as a rule throughout the whole course of the disease. Although there are occasional exceptions the constipation is not often obstinate, and it is generally easy to procure action of the bowels by ordinary means.

Headache is another important symptom of the first stage of the disease, and may be of a dull, heavy, or lancinating character. The headache, although continuous, is subject to paroxysmal exacerbations. It is sometimes referred to the summit of the frontal bone, but more frequently the whole head is the seat of pain. Headache is usually an urgent symptom while consciousness is retained, and its temporary exacerbations are made known by moaning or shrieks; while even after unconsciousness has set in the patient puts his hand to his head, wrinkles his forehead, and distorts his face as if from pain. Vertigo is always present. Patients feel as if they were falling in bed, or as if surrounding objects were revolving round them, and the gait is often reeling and unsteady; but the rapid development of severe symptoms soon prevents all attempts at station and locomotion.

Motor disturbances are almost always present in this stage of the affection. Spasmodic movements occur in the form of partial convulsions, giving rise to tremor and conjugate deviation of the eyeballs, strong convergent and divergent squints, grinning contortions of the muscles of the face, grinding of the teeth, and rotation of the head and neck. In children epileptiform convulsions are not uncommon, but they are rare in adults. It is very probable that what Trousseau has described as the hydrocephalic cry, which so commonly occurs during this period of the disease, is due to spasmodic action of the respiratory and laryngeal muscles, and is quite independent of consciousness. "It is a single, violent cry," says Trousseau, "resembling the cry of a person suddenly exposed to great danger; the

expression of the face is not that of suffering. Any period of the disease may be attended by this cry, which may occur every hour, half-hour, or even every five minutes." Besides spasmodic movements, spastic rigidity of one or more groups of muscles may occur, the most important of these being stiffness of the muscles of the nape of the neck and back, and retraction of the abdominal muscles. Slight paralysis of some of the facial and ocular muscles may occur, consisting of inequality in the pupils, ptosis, strabismus, or slight facial paralysis.

Sensory disturbances are not so well marked, and they are usually soon obscured by loss of consciousness. At times a general hyperæsthesia of the whole surface of the body may be observed at the beginning of the disease, while at other times this condition may be limited. General or partial anæsthesia is not an unfrequent symptom at an advanced period of the disease, and it has occasionally been observed as a premonitory symptom (Dreyfous). Intolerance of sound and light is a prominent symptom: the child is impatient of the slightest noise, and avoids the light by lying with the face buried in the pillow or turned towards the wall, keeping the eyelids firmly closed. In this, the usual attitude of the first period of the disease, the knees are drawn up towards the abdomen, and Dreyfous believes that it is not voluntarily assumed in order to avoid the light, but corresponds to the forced attitudes of animals arising from experimental injury of the brain.

Psychical disturbances are not always present in the early period of the affection. The patient cannot, however, form consecutive trains of ideas, and children soon become somnolent, lie with their eyes closed, and reply to questions curtly or merely by a nod. When raised up, they complain much, knit their brows, throw back their heads, and slip down in bed; they cannot bear the slightest disturbance, and will clench their teeth against food. Delirium is of frequent occurrence when the patient is half asleep; and, in the case of children, the hydrocephalic cry is not unfrequently heard at this time, being accompanied by starting up in terror. At other times the patient may spring from bed, or make defensive movements, in consequence of hallucinations of sight and of hearing. The somnolence soon increases, the eyes stare without expression

into vacancy, and the patient ceases to speak to his attendants; but he talks senselessly with himself, or laughs, sings, whistles, or shouts, and performs meaningless movements, such as plucking at imaginary objects, picking the bed-clothes, blowing, spitting, and grimacing. After a time these disturbances cease, and the mental faculties may again become completely or partially clear; but after a longer or shorter period, new and more profound disturbances supervene, which increase until the fatal termination.

Examination of the retina may throw light on the nature of the affection. When general miliary tuberculosis exists tubercles of the choroid are frequently found, but they are absent in tuberculosis affecting the pia mater alone. Marked stasis is often found even at this early period in the veins of the disc. So long as the patients are tolerably conscious derangement of vision is generally absent.

The pulse is, as a rule, slow and full (from 60—70 beats per minute) during the early stage of the affection, but it is subject to rapid variations during muscular exertion of every kind and psychical excitement, especially in the case of children. The pulse is often decidedly irregular even at this stage of the disease. The temperature is very inconstant, and does not conform to any type.

The skin is usually dry, and even in those cases where the tubercular meningitis is associated with phthisis the sweats of the latter disease usually cease when the meningitis manifests itself. It has also been observed that the cough, dyspnœa, and expectoration of phthisis cease, and in the case of intestinal tuberculosis the diarrhœa is arrested. The injection of the face and conjunctivæ varies frequently without obvious cause; lividity of the face belongs to a later period or is dependent on lung complications.

The average duration of the first stage is about eight days, but it varies from two days to two or three weeks, and is not unfrequently followed by a remission which leads the friends of the patient to hope for a favourable termination.

(*b*) *The second stage* is now ushered in with the evidences of loss of functional activity. The signs of excitement become less frequent, and somnolence and mental torpor more prominent.

At times a profound coma is rapidly developed, which in children is frequently ushered in by a convulsion, and which generally continues unbroken until the fatal termination, but in some cases there are short clear intervals. The hydrocephalic cry in the case of children is most frequent at this time, and adults in the midst of sopor give evidence of severe headache by groans and gestures.

Spasmodic movements and muscular rigidity are more frequent than in the first stage. Contractions of the ocular muscles give rise to combined movements of the eyeballs (nystagmus) and inequality of the pupils, while slight convulsive movements occur in the facial muscles and in those of the extremities. The automatic movements of chewing, winking, whistling, or grinding of the teeth are also frequent; while tremors of the extremities or of the whole body often occur. A case is recorded by Dr. Hughlings-Jackson in which the movements of the extremities and face corresponded exactly to those of acute chorea. At other times the muscles are maintained in a state of cataleptic rigidity, and the extremities may then assume various forced attitudes.

The stiffness of the muscles of the nape of the neck becomes more intense, so that the head is drawn back and thrust into the pillow. The rigidity may also extend to the muscles of the trunk, so that the body is maintained in a condition of tetanic rigidity, the opisthotonos either appearing in paroxysms and lasting only a few minutes at a time, or remaining continuous until death. Paralyses of various extent and distribution now make their appearance. Paralysis of the oculo-motor nerve is common, and gives rise to divergent squint, ptosis, dilatation, and fixity of one pupil. The trochlear nerve may be paralysed along with the third, but is never affected alone. Paralysis of the abducens may, however, occur as a separate affection. The paralytic form of conjugate deviation of the eyes and rotation of the head and neck may appear at this period of the disease.

Facial paralysis is manifested by the usual signs of paralysis of cerebral origin. In some cases total paralysis of the facial has been observed, and in these the affection is due to interference with the nerve by effusion at the base of the brain. In other cases the ocular and frontal branches were affected,

while the oral and nasal branches were spared, and in these cases it is doubtful whether the symptoms are to be attributed to cortical injury or to partial pressure on the nerve at the base of the brain.

Paralysis of the hypoglossal nerve is manifested by the tongue turning towards the paralysed side on protrusion, but this nerve is not so frequently affected as the facial. Hemiplegia and hemiparesis are rare, but monoplegiæ are frequent. At times one arm or one leg is paretic or completely paralysed; while occasionally the paralysis assumes the paraplegic form, or a considerable diminution of strength may occur in all four extremities.

Sensory paralysis also supervenes at this stage of the affection. At times there is a general diminution of all forms of cutaneous sensibility, at other times anæsthesia may be limited to one or other extremity, or to the region of distribution of one of the branches of the trigeminus. After a time every form of sensibility is much weakened or abolished.

The retinæ undergo decided changes in this stage of the affection. The veins are dilated and stand out distinctly on the swelled papilla. Extravasations of blood occasionally occur in the retina, but they are usually slight in extent. Symptoms of neuro-retinitis appear either along with the phenomena of stasis or apart from them.

Vomiting may occur occasionally in this stage, constipation continues, the urine is generally passed in bed or there is complete retention.

The pulse becomes less frequent, and may sink to 60 or still lower, and is at the same time fuller; but the slightest excitement may raise the number of beats over 100 per minute, and it is apt to become irregular. The temperature varies.

The respiration is sometimes normal, at other times irregular, occasionally assuming the character of the Cheyne-Stokes respiration. Neuro-paralytic œdema of the lungs is liable to occur at this period of the disease. This period may last about a week, and then the third stage of the disease is ushered in by the appearance of persistent coma.

The Third Stage.—The patient is now completely insensible to most external excitants, although he may still respond to a

loud voice close to the ear, and most reflex actions are extinguished.

Isolated paralyses are not so readily observed, as there is general relaxation of the whole muscular system. Rigidity of the masticatory muscles and of the muscles of the back and extremities is not unfrequently observed. Convulsions are rare in adults; but in children partial convulsive spasms still occur, and immediately before death the patient may be seized with general convulsions. The electric excitability of the muscles to both constant and induction currents is normal. Every form of sensibility is now completely abolished. The pupils are dilated and fixed, the upper lids fall inert and paralysed over the eyeballs, and an ophthalmoscopic examination reveals signs of marked stasis and œdema of the optic discs, with neuro-retinitis. In the case of young children, the pulsations of the fontanelle become weaker and weaker, and may disappear before death.

The pulse during this period becomes more and more frequent and may be irregular.

The respiration is irregular, and at times so superficial and feeble that it may escape observation, while at other times it is forced and deep. Rapid emaciation occurs, especially in the case of children; the skin is generally dry and rough, and may become slightly livid with the decreasing force of the heart; occasionally a bed-sore appears. The temperature during this period varies greatly. In some cases it is below normal, and immediately before death may sink as low as 95° F. or even 93° F. In another series of cases a state of moderate fever continues up to the time of death; while in a third group the fever greatly increases before death, and may even continue to increase for some time afterwards.

The urine is generally scanty, with high specific gravity; its quantity may be increased, normal, or diminished; the proportion of chlorides varies greatly, and the phosphates are said to be increased. A small quantity of albumen is frequently present, but sugar is rare. The duration of the paralytic stage is generally three or four days, and seldom reaches a week.

§ 841. *Course, Duration, and Terminations.*—The course of tubercular meningitis varies greatly. It has already been

mentioned that when the affection of the pia mater is secondary to phthisis or tubercular peritonitis the disease begins without any premonitory symptoms, and the symptoms of the first stage are obscure or entirely wanting. Old affections of the brain, such as tubercular tumours, also render the course of the disease variable. Under the latter circumstances tubercular meningitis is superadded to a long-existing affection of the brain as a terminal phenomenon, and it is then difficult to diagnosticate anything beyond a meningitis of unknown cause.

In other cases tubercular meningitis runs a rapid course, and may terminate in five or six days. In some of these cases ventricular effusion is wanting, and death is due to some acute process in the brain itself. Some cases, on the other hand, have an exceedingly protracted course; the invasion is slow and insidious, the symptoms are gradually developed, and the disease may last from thirty to fifty or even sixty days.

In all cases in which the disease began by sudden paralysis, or paralysis combined with aphasia, the miliary tubercles have been found limited to the area of distribution of the Sylvian artery, while the choroid plexuses have been free from tubercles, and great effusion absent.

The disease is ushered in by depression of spirits, anxiety, anguish, hallucinations of hearing, self-accusations, and attempts at escape from punishment. After about forty-eight hours, unconsciousness supervenes, ptosis and facial paralysis soon follow with all the other signs of tubercular meningitis.

Sometimes the course of the disease is very similar to that of typhoid fever. In other cases, especially in children, unconsciousness comes on at an early period of the disease, and constitutes throughout the most prominent symptom of the affection. In these cases large ventricular effusion is found after death, and the brain has been doubtless subjected to pressure at an early period.

Some cases are recorded in which every time the patient attempted to sit up attacks of tetanic rigidity supervened (Biermer, Gerhardt).

§ 842. *Morbid Anatomy.*—The morbid anatomy of tubercular meningitis varies with respect both to the appearances

found in the brain itself and to morbid changes found in other organs. Various changes may be found in the cranial bones, and caries of the petrous portion of the temporal bone is frequently the primary cause of the disease. On removing the calvaria tubercles may be found in the dura mater, and would probably be found more frequently if a careful search were made. They are sometimes found between the two layers close to small branches of the middle meningeal artery, while others appear to be situated in the inner lamella of the dura mater (Huguenin).

The changes in the pia mater are, (*a*) those which are directly connected with the formation of miliary tubercles, (*b*) those caused by the inflammation surrounding them, and (*c*) those which arise from the effusion into the ventricles.

(*a*) The pia mater is studded with miliary tubercles. They appear as greyish-white granulations, varying from a size scarcely visible to that of a millet seed, while masses as large as a pea may be produced by aggregation.

The tubercles are always distributed in the neighbourhood of vessels. In some cases the whole length of an artery from its origin in the circle of Willis is covered with numerous tubercles, while in rare cases the granulations are chiefly situated on the peripheral branches of the vessels. At times all the arteries given off from the circle of Willis are studded with tubercles, while at other times particular portions of the surface of the brain are either exclusively affected or affected to a much greater extent than the remaining portions.

The territory supplied by the Sylvian arteries is particularly liable to be affected, and tubercles are also commonly found at the bottom of the great longitudinal fissure along the vessels which supply the corpus callosum. At other times the tubercles are most abundant in the pia mater covering the upper and under surfaces of the cerebellum, or on the median surfaces and posterior lobes of the cerebral hemispheres.

The number of tubercles present varies greatly. At times they may be so limited that a careful search is necessary to find them, while in other cases they are numbered by thousands. The granulations may be isolated, or collected in dense groups; while occasionally they form, along with the inflammatory

products of the pia mater, thick masses which exert pressure on the brain.

The tubercles are found in all stages of development. At times all of them consist of the small, grey, miliary granulations, but at other times these are mixed with larger tubercles, which are yellow at their centres from fatty degeneration. Callosities mainly composed of connective tissue and enclosing old tubercles are found on various parts on the surface of the brain.

(*b*) The *inflammatory* changes in the pia mater may or may not be well marked, according to circumstances. When the pressure caused by the effusion has been great, the surface of the pia mater may be dry and its vessels empty. The convolutions are flattened and the sulci more or less narrowed. A moderate hyperæmia of the pia mater is, however, frequently present, especially at the base, where the vessels are less liable to be subjected to pressure.

Evidences of suppuration are generally found at the base of the brain. Under these circumstances the whole tissue is swollen and yellowish, and cloudy streaks of exudation may be observed passing along the vessels. The convexity is not so liable to be the seat of suppuration. In some cases, however, a sero-purulent effusion is observed in the pia mater of the convexity, while at other times the evidences of suppuration on the convexity are still better marked. In the latter case the pia has lost its delicacy and transparency, is inelastic and easily torn, and is everywhere infiltrated with a sero-fibrinous, yellowish exudation.

The exudation often extends backwards over the anterior surface of the pons and medulla oblongata, creeps upwards to the upper surface of the medulla, and implicates the pia of the entire cerebellum. The suppurative process may extend from the chiasma forwards to the under and internal surfaces of the anterior lobe, along the olfactory lobe and the artery of the corpus callosum.

The changes are not always symmetrical, but in general it may be said that the greatest suppuration will be found where the tubercles are most numerous.

The inflammation may extend along the processes of pia mater

which enter the descending cornua of the lateral ventricles, and may also be transmitted through the great transverse fissure to the velum interpositum. At times the choroid plexuses and velum interpositum may be covered with a yellowish purulent exudation.

Extravasations of blood, varying in size from minute specks to a patch an inch or more in diameter, may be found in the meshes of the pia mater. The growth of tubercle in the walls of a vessel presses upon the media and intima, and thus diminishes its lumen. The vessel is thus partially obstructed, and either a thrombus may form at this point, followed by softening of the coats of the vessel and extravasation, or the internal and middle coats of the vessel are perforated directly by the tubercle.

(*c*) The ventricles are generally distended with serous fluid, but effusion is absent in about 20 per cent of all cases. The septum lucidum is frequently broken down; while the third ventricle is distended, but to a less degree than the lateral ventricles, owing to the resistance offered by the optic thalami. The soft commissure is generally more or less torn and speckled with capillary hæmorrhages, and the anterior portion of the ventricle may be so distended that the pia mater covering the lamina cinerea is exposed. The aqueduct of Sylvius is frequently dilated and the fourth ventricle distended. The fluid is at times purely serous, while at other times it is cloudy from the presence of epithelial cells and white blood corpuscles, and purulent effusions are occasionally observed. In some cases the fluid is tinged with blood derived from rupture of small vessels in the choroid plexuses. The choroid plexuses are hyperæmic, and miliary tubercles may be found in them, though never in large numbers; small extravasations of blood are not uncommon. The ependyma of the ventricles is sometimes dense and opaque, and when viewed by a side light its surface looks as if sprinkled with fine dust. At other times larger granulations may be observed intermediate in size between the fine dust and miliary tubercles.

The distribution of tubercles, inflammatory changes, and effusion may be combined in various ways. In the majority of cases miliary tubercles are distributed over the entire pia mater

and choroid plexuses, there is a large ventricular effusion, and the base of the brain is often covered by a purulent exudation, which in some cases extends to the convexity of the hemispheres. In a few cases miliary tubercles are scattered in small numbers over the pia mater, the choroid plexuses are unaffected, ventricular effusion is absent, and no pus is visible to the naked eye either over the base or convexity of the brain. In other cases the tubercles are limited to the region of distribution of one or more of the arteries of the brain, the territories of the Sylvian arteries being specially liable to be affected. The base of the brain and the vascular region in which the tubercles are developed are covered by a purulent exudation, and there is a moderate ventricular effusion. In some cases the evidences of recent tuberculosis are accompanied by circumscribed thickenings and laminæ of caseous connective tissue, in which old miliary and caseous tubercles are embedded.

The brain itself undergoes many important changes. If the effusion be large the cortex and neighbouring white substance are dry and anæmic, but when effusion is absent these parts are congested and œdematous. The cortex is often studded by punctiform hæmorrhages, caused, according to Rindfleisch, by tubercular degeneration of the nutritive arteries. The vessels of the cortex are surrounded by clusters of white and red blood corpuscles. When the pia mater is stripped off, portions of the substance of the brain will be found clinging to the vessels of the cortex, and consequently the surface of the brain assumes a rough appearance. White softening of the substance of the hemispheres is often observed. It may involve portions only of the fornix and corpus callosum, or may extend into the centrum ovale and basal ganglia.

The *cranial* nerves may all be affected in greater or less degree in tubercular meningitis. The inflammatory process set up at the base of the brain may extend to the sheaths of the nerves and gives rise to neuritis, and when effusion takes place they are injuriously affected by pressure.

The spinal cord is not unfrequently affected in tubercular meningitis. The inflammatory affection of the pia mater passes down a varying distance into the spinal canal, and tubercles

are found in the spinal pia mater. Tuberculosis of the lungs, pleuræ, pericardium, peritoneum, liver, spleen, lymphatic glands, and kidneys is frequently associated with the affection of the cerebral membranes. Cheesy degeneration of the mesenteric or retro-peritoneal glands, or of the bronchial, cervical, or axillary glands, is almost always found associated with the cerebral affection. Suppurations of the vertebræ, of the bones of the extremities, and pelvis, affections of the periosteum and joints, caries of the nasal bones from syphilis, caries of the clavicle, sternum, and petrous portion of the temporal bone are some of the most frequent causes of tubercular meningitis.

§ 843. *Morbid Physiology.*—Miliary tubercles act as foreign bodies and produce an attack of meningitis. In the early stage of inflammation the symptoms are mainly those of irritation of the cortex. It is manifest that the initial symptoms of the disease must largely depend upon the distribution of the tubercles and resulting inflammation. If the tubercular infiltration be mainly limited to one or more of the Sylvian arteries, the disease will be ushered in by symptoms of motor irritation, such as slight spasms, unilateral, or even general convulsions. In those cases which begin with aphasia the lesion is situated, as a rule, along the left Sylvian artery, the branch which supplies the posterior end of the third frontal convolution being specially implicated. In the recorded cases it is not mentioned whether the loss of speech was preceded by any evidence of irritation of Broca's convolution. Difficulties in the articulation of words may occur when there is no aphasia. When the area of distribution of the posterior cerebral artery is chiefly affected, the initial symptoms will be sensory disturbances, as hallucinations of sight and hearing; while the motor disorders consist of associated movements, as those of defence against threatened blows, or attempts at escape from apprehended punishment. When the anterior cerebral arteries are mainly affected, the disease begins by sopor alternating with slight delirious excitement, and coma usually supervenes at an early period.

When the cerebellar arteries are affected, stiffness of the muscles of the nape of the neck and back, and tetanic seizures are prominent symptoms. The various irregularities of gait

which are observed during the first stage of the affection may often be explained by reference to implication of the cerebellum. Dreyfous attributes great importance to implication of the pons varolii in the production of forced attitudes and various sensory disturbances, but it does not appear to me that his reasoning is conclusive.

Motor paralysis occurs mainly in the second stage, but is also observed at an earlier period. In the first stage the paralyses are probably caused by irritation and subsequent exhaustion of a motor centre, but in the second stage they are caused by destructive changes in the motor area of the cortex. Peripheral paralysis may be produced by the cranial nerves becoming implicated in the exudation as they pass along the base of the brain. The sensory portion of the fifth and the nerves of special sense as well as the motor nerves may be injured by the exudation.

The final stage of general paralysis of sensory and motor functions is explained by the gradual compression of the brain, due to increasing ventricular effusion.

§ 844. *Diagnosis.*—Tubercular meningitis is liable to be mistaken for cerebro-spinal meningitis, simple purulent meningitis, hyperæmia of the brain, the terminal stage of abscess, tumours of the brain, thrombosis of the sinuses, and leptomeningitis infantum, but the diagnosis between it and these affections have either been or will be herafter considered.

Typhoid fever with severe brain symptoms may closely simulate tubercular meningitis. Difficulties of diagnosis only arise in the case of aberrant forms of typhoid fever in which the bowels are confined and empty, the abdominal muscles retracted, and the spots absent. On the other hand, it must be remembered that diarrhœa may be present in tubercular meningitis, that hæmorrhage may occur from the bowels in cases of general tuberculosis, and that a roseolar rash may appear on the surface of the body.

The course of the temperature in typhoid fever is often variable, and not always to be relied upon as a means of diagnosis. The diagnosis must be made from a consideration of all the circumstances of the case.

Gastric derangement in young children may cause symptoms almost identical with those of the earlier periods of tubercular meningitis; but the diagnosis is soon cleared up by the progress of the case.

It should also be remembered that many acute diseases are attended by cerebral symptoms closely resembling those of the onset of tubercular meningitis.

§ 845. *Prognosis.*—The prognosis is in every instance exceedingly grave. Many presumed instances of recovery are recorded, but these cases are probably examples of leptomeningitis infantum or other affection, and not genuine tuberculosis of the pia mater.

§ 846. *Treatment.*—Prophylactic treatment is of the utmost importance, since the prospects of recovery are so unfavourable when once the disease is established. The children of scrofulous parents should be most carefully reared. Mothers of strongly marked tubercular diathesis should not suckle their children, and this applies all the more to the case of those in whom evidences of tubercular or scrofulous diseases are already apparent. The children should be sent to the country, fed with good milk, and the greatest care taken in attending to the condition of the digestive organs; the slightest diarrhœa should receive immediate attention. Change of climate to a mountainous district or to the seaside is sometimes attended by the most decided benefit. With regard to medicines, iron, iodide of iron, and cod liver oil must be administered according to the circumstances of the case.

The children should also be specially guarded from the infectious diseases to which they are liable; because an attack of measles or whooping-cough, or indeed any acute disease, is apt to lead to irritation of the glands and subsequent cheesy degeneration, and the degenerated glands in their turn may be the source of tuberculosis.

When the symptoms of meningitis have once appeared, the grave nature of the prognosis should not prevent the attendant from adopting appropriate treatment. There is a possibility in most cases that the meningitis may not be tubercular,

and at any rate attempts should be made to allay inflammatory action. Local blood-letting often relieves the severe headache and gives at least temporary relief. The head should be shaved and ice applied persistently. I have never seen the slightest good result from counter irritation, and its use should be abandoned. Smart purgatives may be of some use in relieving symptoms, and senna in conjunction with sulphate of magnesia or the compound jalap powder answers the purpose well. Preparations of mercury and iodine, and a large number of specific remedies have been used in the treatment of the affection, but with questionable success.

When once the progress of the case has rendered the diagnosis of tubercular meningitis undoubted, the less energetic treatment the better. During the second and third stages cold to the head may be exchanged for warm applications. I have seen delirium and restlessness much diminished by the use of a warm fomentation to the head, and one great aim of treatment is to soothe the sufferings of the patient as much as possible. With this view, when there is jactitation, delirium, and screaming, small doses of opium or chloral should be administered; such stimulants as ammonia or even small quantities of wine may be of use.

Chronic Hydrocephalus.

§ 847. *Definition.*—Chronic hydrocephalus consists of an abundant serous accumulation within the cranium, occupying the general ventricular cavity. A chronic accumulation of fluid into the sac of the arachnoid has been described under the name of external hydrocephalus, but it is doubtful whether the condition has any real existence.

§ 848. *Etiology.*—Chronic intracranial effusions in adult life are probably always the result of intracranial tumours, or of occlusion of one or both of the lateral sinuses, or prolonged venous congestion; while in old age it may be compensatory to the cerebral atrophy occurring after hæmorrhage and encephalitis. These conditions have, however, been already sufficiently considered, and we shall here deal exclusively with the chronic hydrocephalus which is congenital or acquired soon after birth.

The etiology of congenital hydrocephalus is not well known; but hereditary predisposition appears to exert some influence in its production, for more than one child may be affected in the same family. Congenital syphilis is probably the most important predisposing cause, and it is possible that too much importance has been attributed to rickets in its production. Of the exciting causes little is known. Chronic hydrocephalus is sometimes preceded by an attack resembling acute hydrocephalus.

§ 849. *Symptoms.*—Chronic hydrocephalus is generally congenital, and cerebral symptoms, such as daily recurring convulsions, strabismus, or rolling of the eyeballs, are apparent from the infant's birth, while in a few days or weeks the head is observed to undergo progressive enlargement.

Impairment of the general nutrition is one of the first symptoms; the child may seem eager for food and suck well, yet it loses flesh and strength, and the skin hangs in loose folds on its attenuated limbs. The bowels are generally constipated, or diarrhœa may alternate with constipation, and the evacuations are always unhealthy. The child is restless and may be drowsy during the day, but wakeful and fretful during the night. The fontanelles and sutures are now unusually open, the anterior fontanelle is tense and pulsates strongly, and the child is subject to paroxysms of restlessness, during which there is increased heat of the head.

The sutures become gradually wider with the increase of effusion, the fontanelles increase in size, the head assumes a globular form, and the physiognomy of the child soon acquires the characteristic features of chronic hydrocephalus. As the fluid accumulates within the cranium, it presses equally in all directions, and the cavity of the skull must enlarge in the direction of least resistance. According to West, the great increase in the size of the head is effected chiefly by enlargement of the anterior fontanelle and by widening of the sagittal suture, these being the points which are the last to be ossified, and at which the bones of the skull are less firmly fixed. The frontal bones are consequently pushed forwards, rendering the forehead round and prominent, the parietal bones are pressed backwards

and outwards, and the occipital bone downwards and backwards, sometimes even so far that it assumes an almost horizontal position. The head is, as a rule, globular and flat at the top, although it occasionally assumes a conical form. Its size varies, and it has been known to measure two or even three feet in circumference. The orbital plates of the frontal bones are pushed from the horizontal to an oblique or it may be almost vertical position, and thus encroach upon the cavities of the orbits. The eyeballs are consequently pressed forwards and rendered prominent; they are at the same time rotated downwards, so that the white sclerotica appears below the upper lids, while the pupils are half hidden beneath the lower lids. On placing the hand over the open fontanelles and sutures they are felt tense and fluctuating. The hair grows scantily over the head, the skin is tense and shining, differing in this respect from the wrinkled condition of that of the rest of the body, distended veins are seen to ramify over the scalp, and the enlarged head offers a remarkable contrast to the small face, which, according to West, retains for a long time its infantile dimensions. The child has a dull and stupid expression; he cannot hold his head up, and is therefore obliged to maintain the recumbent position or to assume a half-sitting posture, while his head is supported by his hands or propped up with pillows. The cerebral symptoms which appear during the progress of the case are variable, the most usual being convulsions, attacks of laryngismus stridulus, paralyses, of varying distribution, with contractures, strabismus, rolling of the eyeballs, and amblyopia progressing to amaurosis. Hearing, as a rule, remains unaffected until near the fatal termination.

§ 850. *Course, Duration, and Terminations.*—Almost every case of chronic hydrocephalus proves fatal. A large number die from some intercurrent disease, others from laryngismus stridulus, or general convulsions. The disease usually lasts one to two years, occasionally longer; exceptionally, patients affected with chronic hydrocephalus have been known to live to thirty (Bright) or even to seventy-eight years of age (Trousseau).

§ 851. *Morbid Anatomy.*—In chronic hydrocephalus the general ventricular cavity of the encephalon is distended with serous fluid, which varies in quantity from a few ounces to many pounds. The ventricular cavities are consequently greatly enlarged, the openings by which they communicate with one another are dilated, and the septum lucidum, commissures, fornix, and corpus callosum are stretched or torn, while the surrounding cerebral substance may be softened, of normal consistence, or unusually dense. The cerebral hemispheres are compressed and flattened; the convolutions are pressed out and the sulci disappear, the white and grey substances being scarcely distinguishable. The basal ganglia are pressed downwards, the cerebral peduncles are separated, the optic commissure is compressed, the pons varolii and corpora quadrigemina are distorted, the superior surface of the cerebellum is flattened, and the nerves at the base of the brain are compressed.

The membranes of the brain are rendered thin and softened, but the ependyma of the ventricles is sometimes found thickened, rough, and in a granular condition. The bones of the skull are generally thin and transparent, but in some cases they are of normal thickness, while in a few cases they are thicker than normal, being then unusually dense and resisting.

§ 852. *Morbid Physiology.*—Many pathologists believe that the effusion of chronic hydrocephalus is a passive dropsy, but Rokitansky and others are of the opinion that it results from a chronic inflammation of the ependyma of the ventricles and the choroid plexuses. The symptoms are partly due to displacement of the cranial bones, and partly to the compression of the substance of the encephalon.

§ 853. *Diagnosis.*—Congenital hydrocephalus may be mistaken for encephalocele, but in the latter affection the swelling is local; it is doughy and elastic instead of being fluctuating, and is not transparent. Fungus of the dura mater, that has perforated at birth, also forms a local tumour, which appears over one of the bones perforated by it, and not over the sutures or fontanelles, while the mass feels doughy, and when pressed upon symptoms of irritation are produced.

The enlargement of the head observed in rickets may be mistaken for chronic hydrocephalus. In the former the enlargement of the head follows other evidences of the general affection, while in hydrocephalus rickets is either absent or develops subsequently to the enlargement of the head, and this is never so uniform in rickets as in hydrocephalus.

The enlargement of the head which occurs in hypertrophy of the brain may be mistaken for hydrocephalus, but in the former affection the head enlarges at first without producing symptoms, and when these appear the disease pursues an acute course and terminates rapidly in death.

§ 854. *Prognosis.*—The prognosis is always grave, although isolated cases have been known to live to old age. The prognosis with regard to the restoration of the mental faculties is even worse than that as to life.

§ 855. *Treatment.*—Great attention should be paid to the general health, but it is needless to expect to obtain absorption of the fluid by means of internal remedies or the application of counter-irritants. Methodical compression of the head by means of adhesive plaster has been recommended, but has not been productive of any good. The treatment by puncture or aspiration, advised by Couquet and others, affords a chance of partial success. The puncture should be made by a fine trochar, or by the needle of an aspirator, which should be introduced perpendicularly. "The best spot for puncturing the skull," says Ramskill, "is about an inch or an inch and a half from the anterior fontanelle, near the edge of the coronal suture, taking care to avoid the longitudinal sinus and some of the large veins which empty themselves into it." Only a few ounces of fluid should be slowly withdrawn at a time, the skull being at the same time supported by bandages.

3. Simple Meningitis of the Base of the Brain. *(Basilar Meningitis.)*

§ 856. *Etiology.*—Inflammatory processes at the base of the brain are frequently caused by fissures, tumours, abscesses, and other lesions near the base of the brain, but cases of this kind

have already been sufficiently considered. That form of basal meningitis only is to be considered at present which arises spontaneously or from unknown causes. Nearly all those affected are from 16 to 30 years of age, and in most of them hereditary predisposition to tuberculosis is wanting.

§ 857. *Symptoms.*—When primary basilar meningitis is diffused and general, the affection begins by languor, mental depression, chilliness or even rigor, thirst, and the usual symptoms of fever. The patient complains of intense cephalalgia and giddiness, and these are followed by severe attacks of vomiting.

Motor disturbances may be completely absent throughout the whole course of the affection. When present, they consist of spasmodic rigidity of the muscles of the back of the neck, with retraction of the head, and rarely of rigidity or clonic twitchings of certain groups of the muscles of the extremities. The patient grinds his teeth during sleep; and in the later stages of the affection, trismus and hiccough have been observed. Paralysis of the abducens is not uncommon, but paralysis of the oculo-motor nerve is rare. Paresis of the facial or hypoglossal nerves may occur temporarily during the course of the affection and subsequently disappear, but complete paralysis of them has not been observed. The power of deglutition may be impaired during the course of the affection and be afterwards regained, and this may increase to complete dysphagia before death. Paresis of the extremities is occasionally observed, but never complete paralysis.

The sensory disturbances consist of cutaneous hyperæsthesia, especially in the region of distribution of the fifth nerves, ringing in the ears, scintillations before the eyes, and occasionally hallucinations. Anæsthesia and dysæsthesiæ have not been observed. The psychical disturbances are more variable than in any other form of meningitis. In some cases the mental faculties are unaffected throughout the whole course of the disease, while in others they are early involved. The mental symptoms usually consist of a mild delirium; but in exceptional cases this may be more active, the patient being restless, quarrelsome, capricious, and irascible. Active delirium

is usually temporary and soon gives place to a milder form; the patient after a time becomes somnolent, but may be temporarily aroused by a loud question addressed to him; in a short time he falls into a state of complete insensibility, during which the urine and fæces are passed involuntarily. Vomiting continues to distress the patient throughout the course of the disease, the bowels are constipated, but the abdomen is not retracted as in tubercular meningitis.

The temperature curve is very irregular. In the initial period it may rise as high as 104° F. in the evening and approach to the normal in the morning. In the later stages of the disease the temperature remains low, being sometimes subnormal. The pulse, as a rule, follows the temperature, being very frequent in the initial period, and sinking in the course of the disease to below 60 beats in the minute. Towards the end of life it again increases, and becomes very frequent, irregular, and intermittent; the patient is covered with bedsores, much emaciated, and dies in a state of marasmus.

The chronic forms of basilar meningitis may give rise to localised inflammatory products at the base of the brain, which cause symptoms scarcely to be distinguished from those of tumours occupying the same situation. The symptoms are variable in such cases, the most characteristic being paralyses of the various cranial nerves. In addition to the headache and dizziness, there are anosmia, amaurosis, or hemianopsia, ptosis, paralysis of the motor nerves of the eyeball, sensory disturbances in the region of distribution of the fifth nerve, masticatory paralysis, paresis of the seventh nerve, and occasionally paresis of one or more of the extremities. If the inflammation extend to the lower end of the pons, bulbar paralysis, dysphagia, and dyspnœa may be present.

§ 858. *Course, Duration, and Terminations.* — Basilar meningitis generally begins suddenly without premonitory symptoms. The course of the affection is variable, being interrupted by remissions and exacerbations. The duration of the disease extends from a period of seventeen to sixty-four days. In chronic cases it may be prolonged over a period of months; the symptoms are in such cases more or less similar to those of

tumour situated at the base of the skull. The affection generally terminates in death.

§ 859. *Morbid Anatomy.*—The changes found at the base of the brain vary according to the rapidity of the process. In the most acute cases purulent infiltration of the pia mater of the base from the chiasma to the posterior margin of the pons has been found. This infiltration may extend along the fissure of Sylvius for some distance, but does not reach the convexity of the brain; on the other hand, it often extends along the whole transverse diameter of the hemispheres, frequently involving the choroid plexuses and the ependyma of the ventricles. The ventricles are generally distended with fluid, while the convolutions are flattened and the sulci pressed together. In less acute cases the inflammatory exudation induces various degrees of thickening of the pia mater. The choroid plexuses are increased in size, indurated, and may at times be covered with pus.

§ 860. *Diagnosis.*—Basilar meningitis may run a course so similar to typhoid fever that the two affections can only be distinguished by long-continued observation. When the temperature curves of the two affections are similar, the points to be relied on in forming a diagnosis are the presence in typhoid form of diarrhœa, rose-coloured spots over the abdomen, and enlargement of the spleen.

§ 861. *Prognosis.*—Most cases end in death, but some are recorded in which the symptoms corresponded closely with those of basilar meningitis and which ended in recovery.

§ 862. *Treatment.*—Counter irritation in various forms has been employed with good effect, but this remedy should be reserved for the later stage of the disease. Quinine, mercury, and iodine have been employed, but with doubtful success. The headache must be allayed by narcotics. If syphilis be present, mercurial inunction and iodide of potassium should be employed.

4. Meningitis of the Convexity of the Brain.

This affection may either be primary or secondary, acute or chronic.

§ 863. *Etiology.*—Simple primary meningitis of the convexity is a rare affection. It may occur at all ages, but chiefly attacks infants under two years of age, and occurs with decreasing frequency from this age to puberty, when it becomes more frequent. Acute meningitis is rare in advanced age, but the chronic form is frequent. Of adults, men are more liable to the disease than women. The exciting causes are not well known.

Secondary meningitis of the convexity may be produced by inflammation of the bones of the skull, the usual causes of the latter being external injury, scrofula, and syphilis. Gummata may also give rise to this inflammation. Otorrhœa, especially when complicated by caries of the temporal bone, is one of the most frequent causes of purulent meningitis, and the affection may result from puriform softening of a thrombus in one of the sinuses, erysipelas of the head leading to osteo-phlebitis of the bones of the skull, carbuncles of the face and neck, suppuration of the eyeball, and old intracranial diseases like tumours, abscesses, or necrotic softening.

§ 864. *Symptoms.*—The course of acute meningitis may be divided into three stages: (1) The period of excitement; (2) The period of transition; and (3) The stage of collapse.

(1) *The Period of Excitement.*—Obscure premonitory symptoms are sometimes observed, consisting usually of a feeling of heaviness in the head along with paroxysms of violent cephalalgia, sleeplessness, irritability of temper, and general malaise. As a rule, however, the disease begins suddenly by a well-marked rigor, intense headache, vomiting, fever, and delirium, while in children it is ushered in by an attack of general convulsions.

The headache may be diffused, or referred to the forehead, temples, vertex, or occiput; it is intensified by light and sound, and by all movements of the head. The patient consequently shuns the light, and holds his head between his hands, in order to prevent it from moving. The headache is continuous, but is marked by exacerbations of intense severity, during

which the patient, especially if a child, may utter a loud and piercing cry.

Vomiting is a very constant symptom of meningitis, and is, like the vomiting symptomatic of other cerebral diseases, unattended by nausea, and epigastric pain or tenderness. It recurs frequently during the first forty-eight hours, and may then cease or occur at intervals throughout the course of the affection.

The motor disturbances in this stage are not well marked, if the general convulsions which usher in the disease in children, and which may also frequently recur in the course of the affection, be excepted. The patient staggers like a person drunk when he attempts to walk, and when confined to bed he is restless and keeps changing his position. Strabismus, slight twitching of the muscles of the face and limbs, and tonic spasms of those of the neck and back may also be observed. The pupils are usually contracted or unequal during this stage, but react readily to light.

The sensory disturbances consist of buzzing in the ears, flashes before the eyes, and intolerance of light and sound. Cutaneous hyperæsthesia is not unfrequently present, so that the slightest touch on the skin may cause pain, and the reflex excitability is increased.

The psychical disturbances are well marked from the first. The patient is extremely irritable, and fierce delirium is apt to occur, the patient shouting and violently struggling with his attendants. At other times he is morose, and buries his head under the bed-clothes, obstinately refusing to answer questions. The temperature of the body is elevated, the pulse beats from 120 to 140 or more, and the respirations are increased to 30 or 40 in the minute.

(2) *The Period of Transition.*—During this stage the furious delirium of the first stage becomes quieter, the patient lies on his back, with his fingers picking at the bed-clothes or catching at imaginary flies in the air.

More pronounced motor disturbances now make their appearance, consisting of partial or general convulsions, followed by paralysis. The muscles most commonly affected by partial convulsions are those of the eyeballs, producing strabismus; the same muscles in conjunction with those of the neck causing

conjugate deviation of the eyes and rotation of the head; the muscles of the face; those of the jaws causing grinding of the teeth and trismus; those of the tongue causing various distortions of the organ; the small muscles of the hand causing jerking movement of the fingers, subsultus, and tremor of the hands; and, lastly, the larger muscles of the extremities giving rise to various convulsive movements of the limbs. It is probable that the loud cry which the patient continues to utter occasionally in this stage is not a voluntary action, but caused by spasmodic contraction of the associated muscles of vocalisation. The muscles of the neck and back are liable to be affected by tonic spasm, causing retraction of the head with attacks of opisthotonos.

These convulsive symptoms are followed by paralysis, which is very variable in its distribution, some groups of muscles being paralysed while others continue convulsed.

The sensory disturbances consist of dimness of vision and of hearing, ending in blindness and deafness, while the general cutaneous hyperæsthesia of the first stage is replaced by anæsthesia. The bowels are constipated throughout, and the abdominal walls are often retracted as in tubercular meningitis. The respirations are irregular, the pulse frequent and thready, and there is retention of urine.

(3) *The Stage of Collapse.*—The third stage of the affection now becomes established; the convulsive phenomena give place everywhere to paralysis, and the patient passes into a profound and fatal coma.

Symptoms of Secondary Meningitis.—The symptoms of secondary meningitis differ considerably according to the cause of the inflammation; but, inasmuch as inflammation from caries of the petrous bone is the most usual form of the affection, it will be useful to describe it first.

The affection may be ushered in by chilliness or a distinct rigor and feverish symptoms; but intense headache, either continuous or marked by remissions and exacerbations, is the first symptom to direct attention to the brain. The headache may be fixed to a point in the vicinity of the diseased ear, or shoot from one ear to another, while at other times it is diffused over the whole head. If the local affection be attended by pain,

the commencement of the meningitis is marked by a great increase of its intensity, and the onset of the latter may sometimes be completely masked by an increase of the local inflammation in the ear. Attacks of dizziness now supervene, accompanied by nausea and vomiting; the patient complains of noises in the head, general painful sensations diffused over the body, and obscuration of the spinal senses.

After a paroxysm of intense cephalalgia, the patient begins to wander, or becomes actively delirious; these symptoms may, however, disappear temporarily. The initial symptoms are accompanied or preceded by the signs of local disease, consisting of transitory phenomena of irritation followed by those of depression. The signs of motor irritation are rigidity of the muscles of the nape of the neck, convulsive twitching of the facial muscles on the affected side, trismus, grinding of the teeth, and occasionally spasms of the extremities.

The depressive symptoms consist of paralysis of the facial, hypoglossal, and glosso-pharyngeal nerves on the same side as the lesion; while, if the inflammation extend forwards along the base of the skull, the third, fourth, sixth, and probably the fifth nerves may become involved in inflammation. The state of the pupils is variable and liable to frequent changes during the course of the disease, being generally contracted or unequal at first, and dilated and fixed when effusion has taken place. Paralysis of the extremities is rare, but the patient has an unsteady, staggering gait.

The *sensory disturbances* consist of marked hyperæsthesia of the skin, joints, bones, and muscles, so that every movement is painful.

Vomiting generally continues throughout the whole course of the disease, the bowels are constipated, and the abdominal muscles are tender to the touch and retracted. The temperature in acute cases is usually high, but remits in the morning, although it remains constantly high in some cases.

The pulse as a rule rises and falls in frequency along with the temperature, except in the cases where symptoms of compression of the brain occur during the first days of the disease.

The urine is often albuminous, and this may or may not be associated with amyloid disease of the liver, spleen, and kidneys.

The optic discs usually present the same appearances as those observed in tubercular meningitis. The psychical symptoms are very variable, consisting of jactitation, restlessness, and confusion of ideas, especially towards the evening when the temperature rises. After a time the patient falls into a somnolent condition, from which he can at first be readily roused by a loud question, but this state soon gives place to profound and fatal coma.

§ 865. *Course, Duration, and Terminations.*—The duration of simple purulent meningitis is variable, but as a rule the progress of the case is rapid. The disease may terminate in adults within a week, and in infants in a still shorter period, but sometimes it may assume a more or less chronic form, death resulting after weeks or months.

The duration of purulent meningitis, secondary to disease of the temporal bone, varies from a period of twenty-four hours to two or three weeks, and the affection is usually fatal.

§ 866. *Morbid Anatomy.*—The pia mater is infiltrated with a fibro-purulent exudation, the convexity being usually involved to a greater extent than the base, although the latter is generally more or less implicated, and the exudation may even extend over the pia mater of the cerebellum and medulla oblongata. The effusion into the ventricles varies in quantity, and is generally sero-purulent in character. Pus may sometimes be found in the tissue of the choroid plexuses. The pia mater is usually adherent to the cortex, and on being stripped off, portions of the latter are torn off with it. Small capillary extravasations are found in the cortex, or the cortex may be rendered anæmic by the intra-ventricular pressure.

On microscopic examination, the protoplasm of the ganglion cells is found to be granular and the cells themselves deformed, while the vessels are surrounded by emigrant white and red blood corpuscles. Secondary meningitis presents the same general morbid appearances as the primary variety, although its distribution is not always the same.

In the meningitis which results from caries of the petrous bone, the changes may at times be limited to the pons and

neighbouring parts, while at other times the base and the convexity of one or of both hemispheres are implicated. The inflammation generally begins on the inferior surface of the temporal lobe, and extends to the superior and inferior surfaces of the cerebellum, the anterior surface of the pons, and even into the vertebral canal.

If the meningitis be caused by thrombosis of the sinuses, the morbid appearances characteristic of the latter affection are found in addition to those indicative of meningitis.

§ 867. *Diagnosis.*—Simple purulent meningitis is a rare disease and occurs most frequently in youth and manhood, while the tubercular variety is much more common and occurs most frequently between the second and seventh years of age. The prodromata in simple meningitis are not well marked; the disease is suddenly developed in apparently healthy persons, and its onset is marked by rigors, while in the tubercular form the patient has been losing flesh for weeks before the commencement of the attack, and the disease is developed more gradually.

The delirium is, as a rule, more violent in primary than in tubercular meningitis, while the paralytic symptoms are on the other hand more pronounced in the latter. Partial convulsions are more characteristic of tubercular, and general convulsions of simple purulent meningitis, while rigidity of the muscles of the neck, and tetanic spasms of the muscles of the trunk are equally common in both affections, but retraction of the abdominal muscles is not so marked in simple as in tubercular meningitis. Cutaneous hyperæsthesia is more commonly observed in the simple than in the tubercular form.

Meningitis arising from caries of the petrous bone can hardly be distinguished from tubercular meningitis associated with disease of the same bone. Meningitis from thrombosis of the sinuses must be distinguished by the signs of the latter already described; and the diagnosis between purulent meningitis and abscess of the brain has already been considered.

§ 868. *Prognosis.*—Several recorded cases appear to show that recovery may take place in the early stage of simple puru-

lent meningitis, but in such cases the diagnosis must always remain doubtful; when the disease is once fully developed, recovery is probably no longer possible. The prognosis of acute secondary meningitis is always unfavourable; in a few fortunate cases, where the affection is secondary to an abscess, the contents of the latter may escape and recovery ensue.

§ 869. *Treatment.*—In the first stage of the disease the usual antiphlogistic treatment must be adopted, consisting of leeching, purgation, and cold applied to the shaven scalp. When the cephalalgia is intense, narcotics may be cautiously administered, the best being a small dose of morphia, subcutaneously injected. Chloral hydrate, either alone or in combination with bromide of potassium, is useful when there is much restlessness and mental excitement. Mercury and iodide of potassium have been given with the view of promoting absorption, but the affection appears to be much too acute for the action of these drugs.

5. Metastatic Meningitis.

Metastatic meningitis comprises certain varieties of the affection, which occur as terminal phenomena in the course of acute diseases.

§ 870. *Etiology.*—The diseases with which meningitis is most frequently associated are pneumonia, ulcerative endocarditis, acute rheumatism, purulent pleurisy, pericarditis, diphtheria, and the acute exanthemata. Although chronic Bright's disease is liable to be complicated by inflammation of the serous membranes, meningitis is rare.

§ 871. *Symptoms.*—The extent and intensity of the inflammation vary greatly in different cases; in some there is little or no effusion into the ventricles, and the symptoms of compression are absent; the inflammation is sometimes limited to the convexity, and at other times extends to the base and upper part of the spinal cord; and in the meningitis of acute febrile diseases the symptoms are obscured by the cerebral disturbance usually observed in all grave acute affections.

§ 872. *Varieties of Metastatic Meningitis.*

Meningitis with Pneumonia.—Meningitis may appear in the course of pneumonia from the third to the eighth day or even later. The most usual symptoms are chilliness, intense headache, rapidly developed and mild, or occasionally furious, delirium, a fresh accession of fever, and hyperpyrexia before death. The delirium gives place at an early period to somnolency, ending in coma. A slight degree of rigidity and pain in the neck is always a valuable sign of meningitis, and vomiting is a frequent occurrence. The pupils are generally contracted at first, and may subsequently become unequal. If the base of the brain be affected, paralysis of the ocular motor and other nerves at the base of the skull render the nature of the complication more apparent.

Meningitis with Ulcerative Endocarditis.—The cerebral symptoms in ulcerative endocarditis are caused by multiple hæmorrhagic infarctions of the cortex of the brain or of the pia mater, and the symptoms produced are more or less like those of pyæmic encephalitis.

A *rheumatic meningitis* has been described, but post-mortem evidence of its existence is wanting.

§ 873. *Morbid Anatomy*.—The amount of blood in the vessels of the pia mater and brain is variable. The exudation on the pia mater is usually purulent, and varies in quantity from a few specks, scarcely appreciable to the naked eye, to a layer extensively distributed over the surface of the brain. The layer of pus may be limited to the convexity, or extend to the base of the brain. The effusion into the ventricles also varies greatly both in quantity and quality. The ependyma and plexuses are not much changed. It is probable that the substance of the brain, more especially that of the cortex, is involved in the inflammatory process.

§ 874. *Diagnosis, Prognosis, and Treatment.*—The diagnosis must be made from the presence of symptoms which indicate a meningitis supervening, in the course of such diseases, as pneumonia, or ulcerative endocarditis. The prognosis is always

unfavourable, and the treatment must be conducted on the same general principles as that of other forms of meningitis.

6. Traumatic Meningitis.

In traumatic as in other forms of meningitis the cortex of the brain is involved in the inflammatory action, so that the condition would be more accurately described as a meningo-encephalitis.

§ 875. *Etiology.*—This form of meningitis may appear during the period of reaction from concussion, or follow a contusion of the brain. Injury of the scalp, with subsequent inflammation of the bones of the skull and dura mater, may also give rise to inflammation of the pia mater and brain. At other times the inflammatory process is set up by a perforating injury of the skull either with or without extravasation of blood between the dura mater and the bone, the effects in such cases being intensified by the admission of air containing germs into the open wound. In other cases the meningitis is a secondary result of osteitis, thrombosis of the sinuses probably playing an important part in its production in such cases. The meningitis at other times may result after necrosis of the bone has taken place. Hutchinson thinks that in fractures of the petrous portion of the temporal bone the inflammation extends along the sheath of the seventh nerve, and in this way gains access to the subarachnoidal spaces.

§ 876. *Symptoms.*—This affection may be divided into two varieties: (*a*) *acute,* and (*b*) *chronic or subacute* traumatic meningo-encephalitis (Erichsen).

(*a*) *Acute Traumatic Meningo-Encephalitis.*—The symptoms of the onset of the acute form of the affection are modified by the fact that the inflammatory process usually attacks a patient already suffering from the symptoms of concussion, compression, or contusion of the brain. It is impossible to distinguish between the symptoms of the reaction period of concussion and those of the early or congestive stage of true inflammation, so that we are unable to determine when the symptoms of the former terminate and those of the latter

begin. At the onset of the inflammatory attack the patient complains of severe and continuous cephalalgia; the carotids beat forcibly; the face is suffused and the scalp hot; the pupils are contracted; there are intolerance of light and sound, spectral illusions, noises in the ears, and general hyperæsthesia to external impressions. The patient likewise suffers from the usual symptoms of pyrexia; the pulse is full and bounding, and there is restlessness and wakefulness with delirium of a violent character. These symptoms may, under proper treatment, gradually subside until health is re-established; but more commonly the symptoms of the stage of irritation develop into those of the stage of compression.

During the stage of transition between the early stage of excitement and the stage of compression of the brain the symptoms of a localised disease may make their appearance. Clonic or tonic spasms, followed by paralysis, may occur in particular groups of muscles. Rigidity of the muscles of the nape of the neck with retraction of the head is usually present at this period, and may also extend to the muscles of the back and give rise to tetanic seizures. Hemiplegia of the side opposite to the injury is, according to Hutchinson, a constant symptom of direct traumatic meningo-encephalitis, or, as he terms it, arachnitis. The abdominal muscles are usually retracted and the bowels constipated. When the meningitis is situated at the base of the brain, the cranial nerves in their passage along the base of the skull may become implicated. The most usual symptoms produced are ptosis, strabismus, paralysis of the facial muscles or of half of the tongue, and difficulty of deglutition.

The symptoms of compression of the brain now become rapidly developed, the delirium is replaced by stupor, from which the patient is roused with difficulty; the pupils are dilated and insensible to light; the breathing is slow and stertorous; the pulse, retarded at first, becomes feeble and frequent towards the end; the skin is hot and bathed in perspiration; convulsive twitchings or jerkings of the limbs are observed, but these soon give place to general muscular relaxation, and the patient dies in profound coma.

(*b*) *Subacute Traumatic Meningo-Encephalitis.*—This form

of meningo-encephalitis may come on a few days after the injury or not until months have elapsed (Erichsen). The patient has often apparently recovered from the original injury, but as a rule some of the consequences of concussion remain. The patient in the interval has complained of headache, impairment of sight and hearing, confusion of ideas, or mental irritability. The symptoms of the inflammatory attack may be ushered in by an aggravation of the symptoms, which have persisted during the interval, or by an epileptic attack. The pupils are contracted, dilated, or unequal; there are intolerance of light and sound, convulsive twitchings of the limbs and face, strabismus, delirium, and the ordinary phenomena of symptomatic fever. After a time the symptoms of compression supervene, and the patient dies comatose.

§ 877. *Course, Duration, and Terminations.*—The symptoms of acute traumatic meningo-encephalitis sometimes begin a day or two after the injury, at other times not until several days have elapsed. In the subacute form of the affection weeks or months may intervene between the injury and the meningitis.

The duration of the disease varies within wide limits, the average being eight to fourteen days.

§ 878. *Morbid Anatomy and Physiology.*—The surfaces of the visceral and parietal layers of the arachnoid are covered with adherent lymph of a greenish-yellow and purulent appearance. A little fluid pus may sometimes be found between the two layers (Hutchinson). The visceral layer of the arachnoid is congested, or thickened and semi-opaque. The pia mater is usually much congested, but it does not appear to be infiltrated with pus or covered by a layer of lymph like the arachnoid. The substance of the brain is congested and has a rosy hue, while the red points are increased in number. The ventricles are filled with a turbid serum, of a reddish colour. Fracture of the petrous bone gives rise, according to Hutchinson, to inflammation of the subarachnoid spaces at the base of the brain. In such cases the inflammation spreads along the sheath of the seventh nerve, and in this way gains direct access to the subarachnoid spaces.

The effusion begins at the base of the brain, and may extend upwards over the hemispheres or through the transverse fissure into the ventricles, and may also extend down the spinal cord for a considerable distance. The effusion is always underneath the visceral layer of the arachnoid.

§ 879. *Diagnosis and Prognosis.*—Traumatic meningo-encephalitis may be distinguished from the reactive stage of concussion, and from simple congestion of the brain by the elevation of the temperature and the persistence and severity of the symptoms generally. The prognosis is always grave; and in the cases which are said to recover it is doubtful whether anything more than intense congestion of the brain was present. It is, however, important to remember that all the symptoms of the first stage of traumatic meningo-encephalitis may have been present, and yet that the patient may make a good recovery.

§ 880. *Treatment.* — The treatment of acute traumatic meningo-encephalitis is the same as that of the other acute forms of meningitis and encephalitis. The patient should be confined in a quiet and darkened room and removed from all causes of excitement. The head should be shaved and ice applied to it. Erichsen recommends bleeding from the arm, leeches or cupping, free purgation, abstinence, and the administration of calomel so as to produce salivation. As the case assumes a more chronic form the treatment must be less energetic, but the patient must be kept for a long time in a state of complete quietude.

PART V.—DISEASES OF THE ENCEPHALO-SPINAL SYSTEM.

CHAPTER I.

PARALYSIS AGITANS, AND MULTIPLE SCLEROSIS.

(I.) PARALYSIS AGITANS

(Shaking Palsy—Parkinson's Disease).

PARALYSIS agitans is a chronic nervous disease characterised by continuous tremor of the voluntary muscles, and peculiar alterations in the attitudes of the body.

§ 881. *Etiology.*—Paralysis agitans occurs in advanced life, being rarely observed under forty years of age. Duchenne met with the disease in a youth of sixteen, and Meschede in a boy twelve years old as the result of having been kicked by a horse in the face. Men are more frequently attacked than women; hereditary predisposition does not appear to be an important factor in its production.

Exposure to damp and cold seems to be an exciting cause of paralysis agitans, the disease being frequently met with amongst persons living in damp and unhealthy situations. It is sometimes caused by great emotional disturbance, and many cases follow wounds and other injuries.

§ 882. *Symptoms.*—The clinical history of paralysis agitans may be divided into three stages: (1) The period of invasion; (2) the stationary period; and (3) the terminal period.

(1) *The Period of Invasion.*—Paralysis agitans, as a rule, sets in somewhat slowly and progressively, but occasionally in

an abrupt manner. In the *slow* mode of invasion the development of the disease may be preceded for some time by premonitory symptoms, such as sleeplessness, mental irritability, and a transitory feeling of weakness in the limbs. The disease itself is ushered in by a trembling in one of the extremities, usually beginning in the small muscles of the hand or forearm. At this stage the tremors are arrested, on the patient making a voluntary effort. The tremors may, indeed, cease when the voluntary effort is unconnected with the members affected; thus the act of walking may arrest tremor of the upper extremities. Even at this early period the tremor presents characteristic features. If the hand be affected, "the patient," to quote Charcot, "closes the fingers on the thumb, as though in the act of spinning wool; at the same moment the wrist is bent by rapid jerks on the forearm, and the forearm on the arm." The tremor increases in intensity, and, instead of being as at first occasional, it gradually becomes persistent, and invades by degrees parts which have hitherto remained sound. The order in which the various muscles are invaded by the tremor is somewhat variable. The most usual mode of invasion is that which Charcot has called the *hemiplegic type.* In this form the tremor usually begins in the right hand, and after months or years the lower extremity on the same side becomes affected, and after another variable period the left hand and foot are successively invaded. In other cases both the lower extremities are first affected, forming the *paraplegic type;* while in a few cases the upper extremity of one side, generally the right, is first invaded, and then the lower extremity of the opposite side, forming what Charcot calls the *decussated mode of invasion.* Charcot states that the muscles of the neck and head are nearly always unaffected by tremor at every stage of the disease, a fact which deserves particular notice inasmuch as it serves to distinguish paralysis agitans from the cerebro-spinal form of disseminated sclerosis. The tremor may be preceded by a remarkable feeling of fatigue, or by rheumatoid or neuralgic pains, and in such cases it is not uncommon to find that the disease has been occasioned by an injury.

In the *abrupt* mode of invasion the tremor appears suddenly either in one extremity or in all the limbs at once, and

the disease then usually results from great emotional disturbance. The tremor may soon diminish or disappear, but it recurs, and, after a series of alternate exacerbations and remissions, becomes permanent. Whatever be the mode of invasion, the duration of the initial stage varies from one to two or three years.

(2) *Stationary Period.*—When the disease is fully developed the trembling becomes almost incessant, although it varies in intensity. It is aggravated by emotional excitement, cold, and voluntary effort; while, on the other hand, it becomes less during repose, and ceases during sleep.

The different segments of the hands and fingers undergo involuntary and rhythmical oscillations, which closely resemble complicated voluntary movements. "Thus, in some patients," says Charcot, "the thumb moves over the fingers, as when a pencil or paper-ball is rolled between them; in others, the movements are more complicated, and resemble what takes place in crumbling a piece of bread." The handwriting now assumes special characteristics. At an early stage of the disease the writing at the first glance presents little change; but, when examined with a magnifying glass, inequalities are perceived, some parts being thicker and heavier than others. As the disease advances the up strokes become markedly tremulous, probably owing to the lumbricales and interossei muscles being most profoundly affected by the tremor.

The muscles of the head and neck, as already stated, usually remain unaffected. The muscles of the eyeballs are also exempt from tremor, and consequently nystagmus, which is so prominent a symptom of disseminated sclerosis, has no existence in paralysis agitans. The movements of the eyeballs are, however, often executed with great slowness (Debove). The muscles of the face instead of trembling are motionless, the features become fixed, and the face assumes a mournful, stolid, or vacant expression. The utterance is slow, jerky, and accomplished with great apparent effort, soon inducing weariness, and if the tremor of the body be intense it becomes tremulous and broken, while in old-standing cases the saliva may dribble from the mouth to some extent.

After a longer or shorter time the muscular power becomes

gradually weakened. In many cases, however, motor weakness is more apparent than real, the phenomena depending upon the great slowness with which voluntary movements are executed, the immense effort which all voluntary actions, even speaking, entail, and the readiness with which fatigue is induced. But although the muscular power, when measured by the dynamometer, is often retained much longer than might be expected, yet after a time motor power becomes gradually diminished, and paralytic symptoms supervene (Charcot). The paralysis, however, almost always remains partial, and is irregularly developed in different groups of muscles; and, as in various other forms of paralysis, the extensors of the limbs are affected to a greater extent than the flexors. The trembling often abates in the muscles as paralysis increases. The bladder and rectum are only very exceptionally involved in the paralysis. The muscles react normally to both the faradic and galvanic currents.

After a time the muscles of the extremities, trunk, and neck become the subjects of *rigidity*, at first temporary, but ultimately becoming permanent, the flexors being affected to a greater extent than the extensors. The rigidity of the muscles produces characteristic alterations in the attitudes of the body. The rigidity of the anterior cervical muscles causes the head to be strongly bent forwards, and the patient cannot raise it or turn it to either side without great difficulty. The body is also inclined forwards when the patient is standing. The elbows are habitually held somewhat removed from the chest, the forearms are slightly flexed on the arms, and the hands are sometimes flexed, sometimes slightly extended on the forearms, and rest on the epigastrium. The fingers are flexed at the metacarpo-phalangeal articulations, the index and middle fingers are extended, but the remaining fingers are slightly flexed, at the phalangeal articulations, all of them are slightly inclined to the ulnar border of the hand, and the thumb is extended and opposed to the index finger, so that the attitude of the hand and fingers closely resembles that assumed by them in holding a pen (Charcot—*Fig.* 281). In some cases the fingers are alternately flexed and extended at their several articulations so as to resemble the distortions of *arthritis deformans* (*Fig.* 282).

In paralysis agitans, however, the joints are not swollen and stiff, and passive movement of the articulations does not give rise to the creaking sounds observed in the former. The rigidity of the muscles of the lower extremities is sometimes so great as to resemble paraplegia with contracture. The spasm of the adductors of the thighs and muscles of the calf predominate over their antagonists so that the knees are drawn

FIG. 281.

FIG. 281 (After Charcot). *Attitude of the Hand in Paralysis Agitans.*

inwards, the leg is slightly flexed on the thigh, and the foot assumes the well-known position of *talipes equino-varus.* The toes are extended at the metatarso-phalangeal and flexed at the phalangeal articulations (*Griffe des Orteils*).

FIG. 282.

FIG. 282 (After Charcot). *Attitude of the Hand in Paralysis Agitans simulating that of Arthritis Deformans.*

In the advanced stage of the disease the patients move all of a piece, as if their joints were, to use Charcot's expression, soldered together; the head and body are kept inclined forwards, a position which no doubt largely contributes to produce that tendency to fall forwards manifested when walking (Plate IV., 3 and 4).

The gait of the patient is now characteristic. The patient gets up slowly and with difficulty from his seat, and hesitates for a few moments before starting; when once he has begun to walk,

he is compelled to run forwards, in order to save himself from falling. In the language of Trousseau, he looks as if pursuing his own centre of gravity. This gait has been called *paralysis festinans or procursoria*, or simply *propulsion.*

The forward running or propulsion is the usual gait of paralysis agitans, but some patients manifest a strong tendency to run or to fall backwards, although their bodies are inclined forwards, a tendency which has been named *retropulsion.* Graves mentions the case of a patient who, if arrested in his forward movement, immediately began to run backwards; and Charcot could excite the impulse to move backwards in a female patient by slightly pulling her back by the dress when she was standing. It has already been mentioned that a few cases of paralysis agitans are ushered in by rheumatoid or neuralgic pains, but in the majority of cases pains are absent. The patient is, however, distressed by disagreeable sensations, as cramps and sensations of tension and traction in the muscles, along with a general feeling of weariness and discomfort. These sensations render the patient restless, and cause him to seek frequent changes of posture. He complains of a constant sensation of *excessive heat*, although the thermometer shows that the temperature of the body is normal. In order to relieve this feeling, the patient throws off the bed-clothes at night, and only retains the lightest garments in the day-time (Charcot). This sensation of heat is especially felt in the epigastrium and back, but may affect the face and limbs. It is liable to remissions and exacerbations, and seems to attain its maximum after a paroxysm of trembling; it is often accompanied by profuse perspiration.

(3) *Terminal Period.*—The course of the disease is very protracted, and may extend over a period of many years. As the tremors and muscular rigidity increase in intensity, the patients are compelled to remain the whole day seated on a chair, or confined to bed. The general nutrition suffers, the muscles become atrophied, and the paresis of the second stage of the disease gives place to pronounced paralysis. The memory and intellect are weakened, general prostration sets in, the urine and fæces are passed unconsciously, bed-sores appear on the sacrum, and the patient dies from nervous exhaustion and

marasmus. The tremors disappear entirely a few days before death, and paralytic symptoms become predominant. In the majority of cases, however, death results from some intercurrent disease, such as pneumonia or pleurisy.

§ 883. *Morbid Anatomy.*—In a very considerable number of cases post-mortem examination has not revealed any lesion of the nervous system. Charcot examined three well-marked cases of the disease in which no lesion of the nervous system could be found, and individual cases have been examined by Petraeus, Olivier, Th. Simon, and Kühne also with negative results. Cohn found in one case well-marked cerebral atrophy, but the brains of old people are frequently found in this condition in the absence of paralysis agitans. Meschede, Bamberger, Lebert, Marshall Hall, and Skoda found sclerotic patches in various parts of the cord and medulla oblongata, pons, and walls of the ventricles, but these were, doubtless, cases not of true paralysis agitans, but of disseminated sclerosis. Parkinson and Oppolzer found induration of the pons, medulla, and cervical portion of the cord, but these also were probably not cases of genuine paralysis agitans. Cayley and Murchison found sclerosis of the posterior part of the spinal cord, thickening of the septa, enlargement of the central canal, and aggregation of leucocytes in spots. In three cases of paralysis agitans recently observed by Charcot and Joffroy, a microscopical examination revealed changes in the spinal cord, consisting of obliteration of the central canal by increase of the epithelium of the ependyma, and pigmentation of the ganglion cells, especially of the columns of Clarke. In one there was a sclerotic spot on the posterior surface of the medulla oblongata.

Dowse and Kesteven found pigmentary degeneration of the nerve-cells near the decussation of the anterior pyramids, of the cells of the olivary body, nucleus of the ninth nerve, laminæ and corpus dentatum of the cerebellum, and anterior cornua of the spinal cord, along with cortical sclerosis of the right lateral column of the cord, and miliary changes in the white matter of the corpus striatum and hemispheres.

§ 884. *Morbid Physiology.*—As just observed, not much light has hitherto been thrown upon the pathology of paralysis agitans by morbid anatomy. The results obtained, however, favour the idea that the morbid changes are due to a chronic degeneration. If the changes begin, as the observations of Charcot and Joffroy seem to indicate, around the central canal of the spinal cord, the small cells of the accessory system may be expected to suffer to a greater extent than the large cells of the fundamental system; and if the lesion consist in part of a thickening of the connective tissue septa of the cord, as was observed in Murchison's case, the small fibres of the accessory system, which lie near the vessels, will be injured by the usual cicatricial contraction of the new growth to a much greater extent than the larger and more resisting fibres of the fundamental system. Even aggregations of leucocytes in the neighbourhood of the vessels, such as were found in Murchison's case, would damage the accessory cells and fibres to a greater extent than the fundamental. The results obtained by Dowse and Kesteven, however, appear to show that the morbid changes in paralysis agitans are not limited to the spinal cord, but are widely diffused in the cerebellum and cerebral hemispheres, as indeed might be expected when it is considered that the disease occurs almost exclusively during the degenerative period of life. It is worthy of notice that Dowse and Kesteven make special mention of the white matter of the corpus striatum, probably the internal capsule, as having undergone morbid changes.

Turning to the clinical history of the disease, the most prominent symptoms are the tremors, the slowness in the execution of movements, and the peculiar alteration in the attitude of the body with its associated muscular rigidity. The causes of tremor have already been discussed (§§ 68 and 73). It is probably caused in this affection by a diminution in the conductivity of the fibres of the pyramidal tract, which prevents impulses from the cortex reaching the muscles in sufficiently close proximity to produce a continuous contraction. According to this view, the tremors and the slowness in the execution of movements are merely the first indications of the more pronounced paralysis which supervenes in the

terminal period. Another view, which might be adopted with regard to the origin of the tremors, is that they are caused by a loss of the balance normally existing between the regulative functions of the cerebrum and cerebellum. The attitude of paralysis agitans is, as has been pointed out by Hughlings-Jackson, the opposite of that of tetanus. During a tetanic seizure the actions of the extensors of the trunk and lower extremities predominate, and the body is arched backwards; in paralysis agitans the action of the flexors predominates, so that the different segments of the trunk and lower extremities are flexed upon one another. In tetanus the muscles, the actions of which must have gradually predominated in the course of evolution, in the attainment of the erect posture, are excited to increased activity; while in paralysis agitans the same muscles, speaking broadly, become relatively paralysed, and there is a gradual reduction of the human to the animal posture. A patient suffering from paralysis agitans during the stage of propulsion is, in his attitude, very similar to a dog attempting to walk on his hind legs. If, then, paralysis agitans be a disease in which the accessory portion of the nervous motor apparatus suffers to a greater extent than the fundamental part, it may be asked how it is that the facial and ocular muscles escape tremor. I am unable to give a satisfactory answer to this question; but it must be remembered that, although these muscles do not suffer from tremor, yet they are affected with comparative immobility and rigidity at a comparatively early period of the disease.

The phenomena of propulsion are caused partly by the forced attitude of the patient and partly by the great slowness with which his movements are executed. When the heel is once raised from the ground by contraction of the muscles of the calf, the patient must in walking balance himself on the ball of the foot, but in walking the forward inclination of the body tends to make the line of gravity pass in front of the active leg. The position is to some extent the same as that assumed by a person running. In the latter the centre of gravity is held well forwards, so that the line of gravity falls in front of the foot of the active leg during the greater portion of the time it is maintained on the ground. A healthy runner, however, is able to take a rapid

and long stride with the passive leg, so that by the time it is brought to the ground, in order to become active, it is in front of the line of gravity, and the body is thus kept from falling. But it is different in paralysis agitans. The rigidity of the muscles prevents the patient from taking a long stride, while the great slowness with which all his movements are executed renders it impossible for him to plant the leg about to become active in advance of the line of gravity. In retropulsion the line of gravity must ever tend to fall behind the point of the foot of the active leg, while the other leg cannot be moved backwards with sufficient celerity to enable the patient to plant it far enough behind the retreating centre of gravity in order to arrest the backward movement.

§ 885. *Diagnosis.*—Paralysis agitans is most likely to be mistaken for senile or toxic tremor and disseminated sclerosis. It may be distinguished from senile tremor by the facts that it occurs before senescence, and that its tremor is of greater intensity; the gait and expression of paralysis agitans are also characteristic. The tremor of mercurial poisoning resembles paralysis agitans more closely than that of any other form of toxic tremor, and in distinguishing between them the history of the case will be of much value. The diagnosis between paralysis agitans and disseminated sclerosis will be described when the latter disease is under consideration.

§ 886. *Prognosis.* — As far as recovery is concerned the prognosis is absolutely unfavourable, but the patient may live for a very long period. The disease may, indeed, last thirty years, and the symptoms of the third or terminal period may linger on for four or five years. The sooner the muscular rigidity and paralysis supervene the more unfavourable does the prognosis become.

§ 887. *Treatment.*—The treatment of paralysis agitans can only be palliative. Carbonate of iron, chloride of barium, hyoscyamus, and the use of the constant current or Pulvermacher's chain, and warm baths are the agents which have been vaunted in the treatment of the disease. Strychnine has been

praised by Trousseau, but Charcot thinks that, instead of calming, it aggravates the tremor. Ergot of rye and belladonna have also been tried, but without success. Morphia and other narcotics are necessary adjuncts of the treatment in the later stages of the affection when the patient is harassed by restlessness and sleeplessness, and both chloral and bromide of potassium may be of use; none of these remedies appears to produce any action beyond palliating the symptoms. Eulenburg recommends the subcutaneous injection of Fowler's solution, and I must say that I should have more faith in arsenic than in any other remedy with probably the exception of phosphorus. Quinine, zinc, nitrate of silver, and chloride of gold have all been tried, but without producing any marked effect upon the disease.

(II.) MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD

(Disseminated or Insular Sclerosis).

§ 888. *Definition.*—Multiple sclerosis is, as its name implies, a chronic induration disseminated in patches in various parts of the nervous system; the affection is characterised clinically by the presence, in greater or lesser integrity, of a group of symptoms, the most constant of which are muscular weakness and tremor on voluntary effort.

§ 889. *History.*—Disseminated sclerosis was first described by Cruveilhier in his "Atlas de l'Anatomie Pathologique," 1835—1842. The clinical histories of two cases of the disease are there given, which, along with the accompanying plates of the lesions found, leave no doubt as to the nature of the affection described. Carswell in 1838 accurately represented in his "Atlas" the lesions observed, while Marshall Hall in 1841 described an undoubted example of this affection. It was that of a man, aged 28 years, suffering from tremor of the right arm and leg, who had a peculiar rocking motion of the eyes, and a degree of stammering and defective articulation. In Germany, the disease was studied clinically by Frerichs (1849), Valentiner (1856), and Türck (1856); while its pathological anatomy was examined by Rokitansky, Leyden, Rindfleisch, and Zenker. But this singular affection has been studied with the greatest success in the Salpêtrière by Charcot and his scholars, and our present accurate knowledge of the disease is mainly owing to their labours. A valuable paper on the subject has been contributed by Moxon in this country, and individual cases have been recorded by many others.

§ 890. *Etiology.*—Hereditary transmission has been traced in several recorded cases of multiple sclerosis, although it is always indirect rather than direct. Duchenne and Erb have reported cases of the kind. Frerichs observed the affection in a brother and sister, and Dreschfeld described two exquisite examples in brothers.

According to Charcot the female is more frequently affected than the male sex, but the statistics of others do not bear out this conclusion. Multiple sclerosis is commonly observed in youth and middle age, and usually makes its appearance during the second and third decades of life, and probably never after 45 years of age. In a large proportion of the cases recorded in England, children under ten years of age have been the subjects.

The exciting causes are exposure to cold and damp, excessive mental or bodily exertion, profound emotional disturbances, and traumatic influences, as blows on the head and concussion from railway accidents.

§ .891 *Symptoms.*—Multiple sclerosis has been divided by Charcot into three varieties: (1) The *cerebro-spinal,* (2) the *cerebral,* and (3) the *spinal* form. Of these the cerebro-spinal form is by far the most frequent and important.

(1) *The Cerebro-Spinal Form.*— This form of multiple sclerosis, as a rule, develops gradually and insidiously, but occasionally abruptly. In cases the development of which is gradual the initial symptoms are very obscure, and may be referred either to the spinal cord or brain. The spinal symptoms which usher in the disease consist of paresis of the lower extremities with a slow and trembling gait, or ataxia with various paræsthesiæ, neuralgic pains, other disturbances of sensibility in the limbs, and cardialgic attacks accompanied by urgent vomiting. The more usual cerebral symptoms observed in the beginning of the disease are vertigo, headache, staggering gait, tremors on voluntary effort, impairment of speech, vision or hearing, paresis of the muscles supplied by one or more of the cranial motor nerves, and various psychical disturbances. When the disease begins abruptly, the symptoms are ushered

in by a convulsive or apoplectiform attack, followed by diplopia, amblyopia, or nystagmus, and disturbances of speech.

The first motor symptom to attract attention is usually paresis or paralysis of certain muscular groups. Weakness generally begins in one leg, and subsequently extends to the other leg and to the arms, but the order in which the paralysis of the different muscular groups is developed presents every imaginable combination.

The gait is usually of the spastic variety, muscular contractures set in, and the legs are held like rigid bars in the position of extension and adduction, just as in primary lateral sclerosis. In the later stages of the disease flexion of the different segments of the lower extremities may predominate over extension. The paralysis rarely becomes so well marked in the upper as in the lower extremities. When the upper extremities are, however, affected with paralysis and contracture, they are maintained in a position of forced extension, and closely applied to the sides of the body. The affection sometimes begins with ataxia, but in these cases it may often be noticed that characteristic symptoms of true locomotor ataxia are absent, while others are present which do not usually belong to it. In a patient under my care at present, for instance, the symptoms when I first saw him, upwards of two years ago, were paralysis of both sixth nerves, and an ataxic gait. But the gait, differing from that of locomotor ataxia, was somewhat reeling, although not sufficiently so to be attributed to cerebellar disease, there were no lancinating pains in the extremities, and the patellar-tendon reflex was exaggerated in both legs. The patient is now suffering from the same ataxic gait, the characteristic tremors of multiple sclerosis, slight nystagmus, and scanning speech, while the paralysis of the external recti muscles has disappeared.

Intermittent muscular tremor constitutes one of the most characteristic symptoms of this affection, although it has been found absent in a few isolated cases. This tremor appears almost exclusively during voluntary movements, and disappears during repose. So long as the patient remains seated quietly the tremor is either entirely absent, or at most there is only a trifling shaking movement of the head, or a slight oscillation of the trunk. As soon, however, as he attempts to seize anything

with his hand the tremor begins, and increases in violence in proportion to the effort made to execute the movement. Several devices may be used in order to bring into prominence the characteristic tremor of multiple sclerosis, such as asking the patient to seize small objects with his fingers or to stretch out his arms horizontally before him.

The tremor of this affection differs from that of paralysis agitans, not only in being intermittent instead of continuous, but also in having a much wider sweep than that of the latter affection. It holds an intermediate position between the extensive jerking movements of chorea, and the small and frequent oscillations of paralysis agitans. The true characteristics of the tremor of multiple sclerosis is best elicited by asking the patient to convey a glass of water to his mouth. As the glass is being carried to the mouth it oscillates from side to side in the patient's hand, these oscillations appearing to increase in extent and frequency as the mouth is approached. In aggravated cases the contents of the glass are spilt in every direction; but in milder cases the patient is able, moving his head downwards in order to meet the glass, to apply it to his lips, and then the trunk, head, and arms begin to tremble violently, so that the edge of the glass rattles against the teeth, and the contents are spluttered over the patient's face. When the patient rises and attempts to walk the tremor involves the entire body, which may be shaken with such violence that he is unable to proceed or even to remain standing. As soon as the voluntary effort is relaxed the tremor diminishes, and as long as the patient is in the recumbent posture no trace of it can usually be detected; occasionally the tremor has been known to persist during repose.

The *sensory* disturbances are somewhat variable and not always well marked. They may assume the form of facial neuralgia, lancinating or diffused pains in the extremities, hyperæsthesia or anæsthesia of variable distribution, girdle pains, and various paræsthesiæ felt in different parts of the body, the latter being the most frequent of all the sensory disorders.

The *reflex* actions are variously affected in different cases. The cutaneous reflexes remain for a long time unaffected;

but the deep reflexes are usually exaggerated, especially in the lower extremities. In consequence of the increase of the tendon-reflexes in the lower extremities, the knee-phenomenon and ankle clonus are usually exaggerated, and the limbs may be thrown into the state of trembling named *spinal epilepsy*. This condition must, however, be carefully distinguished from the characteristic tremor of multiple sclerosis.

Trophic disturbances are generally wanting for a long time, but in the later stages various nutritive disorders usually make their appearance. The sclerotic nodules may encroach on the anterior grey horns of the spinal cord, and then muscular atrophy results as in progressive muscular atrophy. Muscular atrophy may present itself in the upper or lower extremities, neck, face, tongue, or indeed in any part of the body. The electrical reactions of the nerves and muscles remain normal until the muscular atrophy begins, and then the electric irritability of both becomes gradually diminished.

During the terminal period of the disease bed-sores appear over the sacrum and other parts subjected to pressure, and general nutrition fails.

The *bladder and rectum*, as a rule, remain unaffected for a comparatively long time, but their functions are ultimately interfered with as in chronic myelitis. The disorders of the sexual functions are somewhat variable. In some patients sexual desire appears to be increased at an early period of the disease, while in others it is completely abolished. In the majority of cases the sexual functions remain normal for a comparatively long time.

Bulbar Symptoms.—Some of the phenomena caused by implication of the pons and medulla oblongata in the morbid process are amongst the most important and characteristic symptoms of the disease. The speech is slow and hesitating, while each syllable is separately pronounced, presenting a mode of articulation which has been named the *syllabic* or *scanning*. The voice is weak, low, sometimes whispering, and monotonous, while it breaks readily when forced efforts are made. Laryngoscopic examination shows that the vocal cords move normally, but their tension is diminished and frequently changes (Leube). The acts of laughing and crying are often represented by peculiar

noisy inspirations. After a time symptoms of true bulbar paralysis supervene, the movements of the lips and tongue are impaired, and by-and-by mastication and deglutition become increasingly difficult, the velum palati is paralysed, speech becomes inarticulate, and the mouth remains permanently paralysed, while the saliva dribbles out.

Diplopiá with strabismus is a not unfrequent symptom, although it may subsequently disappear as in locomotor ataxia.

Nystagmus is, however, the most important of all the ocular symptoms, being present, according to Charcot, in about half the cases. The movements of the eyeballs may be persistent or occur only during forced accommodation, or when movements are performed by the extremities. At other times the nystagmus may not be apparent during the ordinary movements of the eyeballs, but when the patient is asked to look upwards and outwards so as to strain the ocular muscles, slight oscillatory movements may be observed.

Amblyopia is not unfrequently observed. It consists of a progressive weakness of sight, accompanied by colour blindness and restriction of the field of vision, and may increase to complete blindness. The development of amblyopia is sometimes preceded by photopsia; the optic discs may be normal, partially diseased, or the subjects of white atrophy.

The senses of smell, taste, and hearing are impaired in some cases, but these disorders are rare.

Psychical disturbances are always observed in multiple cerebro-spinal sclerosis. They consist of mental irritability, emotional excitability causing the patient to laugh or to shed tears without apparent motive, and impairment of memory and intelligence. At other times the mental disorder assumes the form of distinct unsoundness of mind. In such cases there may be melancholia, monomania, with ideas of persecution or of grandeur, and the patient may fall into a state of complete dementia.

In the course of the disease, the patient suffers from attacks of vertigo. This symptom usually comes on at an early period, and continues to distress the patient throughout. The patients feel as if they themselves were being turned round, or as if

surrounding objects were whirling round them. They suffer greatly from sleeplessness and violent headache.

Apoplectiform or epileptiform seizures have been observed in a small number of cases; they are apparently analogous to the apoplectiform attacks, which occur in general paralysis of the insane. They are characterised by the development of grave cerebral symptoms, and are accompanied by a considerable elevation of temperature. After slight premonitory symptoms, such as a feeling of pressure in the head, there is a partial loss of consciousness, which in a few hours may develop into coma. The face is red and hot, the pulse is quick, and the temperature of the body rises to 104° F. or 105° F. In some cases the loss of consciousness is accompanied by unilateral convulsions—epileptiform attacks; while in other cases there are no convulsions—apoplectiform attacks. In most cases hemiplegia with muscular flaccidity, and on rare occasions rigidity, is present from the outset of the seizure. After one or two days the temperature falls, the patient sinks into a quiet sleep from which he may be readily roused, and he feels, on awaking, comparatively well. Hemiplegia, however, persists for a few days longer and then gradually disappears. These attacks may be repeated several times in the course of the disease, recurring in some cases every few months; but each is followed by an aggravation of the general symptoms, and death sometimes occurs during an attack.

(2) *Cerebral Multiple Sclerosis.*—In this form of the disease, which is rarely observed, the psychical disturbances are predominant. The tremor is said to precede the paralytic manifestations, but in other respects the course of the affection does not differ greatly from the cerebro-spinal variety.

(3) *Spinal Multiple Sclerosis.*—The spinal form of the affection is characterised by the absence of the cerebral symptoms, particularly nystagmus, tremor on voluntary effort, vertigo, apoplectiform attacks, and psychical disturbances. The symptoms of the spinal form of multiple sclerosis often simulate those of primary lateral sclerosis, although in the former some additional symptoms are usually present. In other cases they simulate locomotor ataxia, but in multiple sclerosis symptoms are usually present which form no part of the former.

§ 892. *Course, Duration, and Terminations.*—The course of multiple sclerosis is divided by Charcot into three stages:—

The *first* stage extends from the beginning of the disease up to the appearance of marked paralysis with contractures. This stage may last from two to six years, or longer. Its development is generally slow. It sometimes begins with cephalic symptoms, as headache, vertigo, and unsteady gait; but more usually with spinal symptoms, as paresis of the lower extremities, and in such cases the nature of the disease remains doubtful until the appearance of the characteristic tremor clears up the diagnosis.

In other cases the development of the disease is more rapid. It begins by an apoplectiform attack, or gastralgic disturbances, while paralyses, disorders of co-ordination, tremor, and other symptoms are superadded in quick succession. The progress of this stage is often interrupted by remissions or improvements, but the nature of the disease is essentially progressive. The patients become more and more helpless, complete paraplegia is developed, the legs being maintained in a condition of rigid extension and adduction; tremor deprives them of the use of the hands, and the intellectual power becomes more and more impaired.

The *second* stage of the disease is now developed; it lasts from four to six or more years. During this period the disease remains more or less stationary; the general nutrition is but little impaired.

The *third* stage is characterised by impairment of general nutrition and the appearance of symptoms indicative of exhaustion. The patient loses his appetite and becomes emaciated; the bladder is paralysed, and cystitis and bed-sores occur, leading to pyæmia, marasmus, and death.

The increasing bulbar symptoms may threaten life in another way, while it is not unfrequently cut short by intercurrent disease, as pneumonia, pleurisy, or pulmonary consumption.

Some cases have terminated fatally in from one to two years from the commencement, but such cases are rare. The average duration is from five to ten years, but individual cases have lived much longer.

The termination of the disease is always in death. During

the first stage a partial amelioration of the symptoms may occur, either spontaneously or under treatment, which may lead the patient and his friends to hope for recovery. The improvement is, however, deceptive, for the symptoms always return and ultimately prove fatal.

§ 893. *Morbid Anatomy.*—The morbid alterations in multiple sclerosis appear in more or less numerous spots or nodules, which are scattered in greater or lesser number throughout the spinal cord, medulla oblongata, pons varolii, cerebellum, and cerebrum.

The individual nodules, when near the surface of the spinal cord, may be seen through the pia mater as brown or amber stains, and in aggravated cases the entire surface of the cord may be studded with greyish spots. Each spot is, as a rule, sharply defined from the surrounding tissues and slightly elevated above the surface of the cord, but it is occasionally atrophic and depressed or on a level with the normal portions. On transverse section the nodules appear grey or greyish-yellow, and when exposed to the air change to a salmon colour; they are translucent or opaque, irregular or oval in shape, generally isolated and circumscribed, but occasionally confluent, and are in consistence dense, tough, even cartilaginous, but rarely semi-fluid and gelatinous. These nodules vary from the size of a hemp-seed to that of a bean in the spinal cord, but they often become confluent and consequently appear to attain a much larger size in the brain. The distribution of the nodules in the spinal cord is subject to great variations. On making successive transverse sections of the cord the nodules will appear in one or both of the lateral columns at one level, in the posterior columns at another, and in the grey substance at a third, while the nodule occupies the greater part of the area of the section at certain levels. The number of nodules which are present is variable, a few only being observed in some cases, while in others hundreds may be counted.

The cerebral hemispheres usually contain a large number of nodules, their favourite sites being the white substance of the centrum ovale, septum lucidum, corpus callosum, basal ganglia, and walls of the lateral ventricles. The cerebellum

generally contains a few of them, these being generally found in the central white substance. The convolutions of the cortex of the brain, and the cortex of the cerebellum, are usually exempted. A considerable number of nodules are generally found in the pons, medulla oblongata, and peduncles of the cerebrum and cerebellum.

The nerves themselves may be affected by patches of sclerosis; the cranial nerves in their passage along the base of the skull are specially liable to be affected. The anterior and posterior roots of the spinal nerves have been found diseased.

The membranes of the brain and spinal cord are frequently normal, but at other times they present evidences of hyperæmia and chronic inflammation. The cerebro-spinal fluid is generally increased, often cloudy, and the ventricles are dilated. Bed-sores, pyelo-nephritis, and evidences of pyæmia or of an intercurrent disease are usually observed at the autopsy.

On microscopical examination the sclerosed patch is found to present the usual appearances of chronic interstitial myelitis. The trabeculæ of the neuroglia are thickened, the nuclei are swollen and increased in number, while Deiter's cells are numerous, large, and sharply-defined. The medullary sheath of the nerve-fibres is gradually destroyed, but the axis-cylinder persists for a long time. Ultimately the nodule consists of a wavy fibrillated connective tissue, in which all trace of nerve structure is lost. The walls of the vessels are thickened and the lumen is diminished, while the adventitia becomes blended with the connective tissue, and the surrounding lymph-channels are obliterated. An infiltration of fat into the lymph-channels surrounding the vessels has been described, but this appearance is probably produced by methods of histological preparation. If the sclerosis extend into the grey substance, the ganglion cells become degenerated and atrophied.

§ 894. *Morbid Physiology.*—Multiple sclerosis is a compound affection, and the implication of several of the functional systems of the spinal cord affords a ready explanation of a large number of the symptoms.

The paresis, contractures, excess of the tendon-reflexes, and the phenomena grouped under the name of *spinal epilepsy* are

caused by implication of the lateral columns; the ataxic symptoms are produced by the formation of nodules in the posterior columns; muscular atrophy by invasion of the anterior cornua; while impairment of speech, disturbances of respiration, difficulty of deglutition, and other bulbar symptoms are caused by disease of the nuclei in the medulla oblongata and pons. The nystagmus is caused probably by the presence of nodules in the corpora quadrigemina or peduncles of the cerebellum; whilst impairment of smell and taste, diplopia, facial and other paralyses, and amblyopia are often produced by sclerotic patches on the cranial nerves themselves as they pass along the base of the skull. Vertigo may be occasionally due to an existing diplopia, but it is generally the result of nodules in the cerebellum.

The *psychical* disturbances are doubtless caused by the development of nodules in the hemispheres of the brain.

The apoplectiform attacks are difficult to explain, but the most usual explanation is that they are occasioned by attacks of cerebral congestion. This opinion is, however, opposed by Charcot, who was unable to discover any evidence of congestion or œdema of the brain in cases which terminated fatally. He thinks that these attacks are only observed in cases in which the pons and medulla oblongata are diseased.

The cause of the characteristic tremor of multiple sclerosis is very obscure. Charcot attributes it to the long persistence of the axis-cylinders in the nodules of sclerosis. Conduction through these may still take place, although when once the medullary sheath is destroyed the conduction will be so retarded that the impulses from the cortex do not pass in a sufficiently quick succession to cause a continuous contraction. On the other hand, it is asserted that in purely spinal cases the characteristic tremors are absent (Hammond, Ebstein), and that they are never present unless the pons and the parts of the brain situated in front of it are affected (Ordenstein). Erb examined twenty-two recent cases with the view of deciding this question. In all the cases in which the tremors were present during life, the pons, medulla oblongata, crura cerebri, and other parts of the brain were involved in the sclerosis; while in the cases in which there were no tremors the nodules were absent or only

present in small numbers in the pons, medulla oblongata, and cerebellum, although other parts of the brain were affected.

§ 895. *Diagnosis*.—Multiple cerebro-spinal sclerosis and paralysis agitans have only been distinguished from one another in recent years. The tremor of paralysis consists of fine rapid oscillations; it persists during repose, may be temporarily arrested by a voluntary effort, and never implicates the muscles of the head; while the tremor of multiple sclerosis is more extensive, ceases during rest, is excited or aggravated by voluntary movements, and invariably implicates the muscles of the head. Paralysis agitans is a disease of advanced age, and multiple sclerosis of youth and middle age. In the former paralysis is not developed until long after the appearance of tremor; while in multiple sclerosis the paralysis precedes or soon follows the tremor. The cerebral symptoms of multiple sclerosis already described are wanting in paralysis agitans.

The spinal form of multiple sclerosis may be mistaken for locomotor ataxia; but in the former disease the ataxic gait may be associated with excess of tendon-reflex, tremor, early appearance of paralysis, scanning speech, nystagmus, or other symptoms which do not belong to locomotor ataxia.

The cases of hereditary ataxia described by Friedreich are, owing to the presence of nystagmus, very liable to be mistaken for multiple sclerosis; but in the latter early paralysis, contractures, excess of tendon-reflex, scanning speech and other bulbar symptoms, and apoplectiform attacks and other cerebral disturbances are absent. The spinal form of multiple sclerosis is most liable to be mistaken for primary lateral sclerosis, and in some cases a diagnosis is impossible. If, in addition to the well-known and classical symptoms of lateral sclerosis, other symptoms, as scanning speech and tremor, be present, then multiple sclerosis ought to be suspected.

When the grey substance of the spinal cord and medulla oblongata is involved, multiple sclerosis may be mistaken for progressive muscular atrophy, progressive bulbar paralysis, or amyotrophic lateral sclerosis; but the course and symptoms of multiple sclerosis render it easily distinguishable from the other affections.

Certain affections accompanied by trembling, as senile, mercurial, and saturnine tremor, bear some resemblance to multiple sclerosis; but the diagnosis, as a rule, presents no difficulty. I have, however, seen a case of mercurial tremor which could only be distinguished from a moderately advanced case of multiple sclerosis, by the history of the case and by the beneficial effects of treatment. Tremor in hysterical subjects may likewise be mistaken for that of multiple sclerosis; in the former the tremor persists during repose provided the patient be conscious of being observed, it may disappear for days or weeks and then recur, and general hysterical symptoms are usually present. The disorderly movements of chorea differ considerably from the tremor of multiple sclerosis, but the diagnosis is not always easy when, as may occasionally happen, choreiform movements complicate those proper to multiple sclerosis.

§ 896. *Prognosis.*—The prognosis is always unfavourable, but it must be remembered that the course of the disease is often interrupted by remissions and partial ameliorations of the symptoms. The prognosis in each case will depend upon the presence or absence of bulbar symptoms, apoplectiform attacks, cystitis, bed-sores, and other symptoms which are known to threaten life.

§ 897. *Treatment.*—The treatment of multiple sclerosis is essentially the same as that of chronic myelitis, our therapeutic means being even more limited in the former than in the latter disease. Arsenic, belladonna, bromide of potassium, ergot, and strychnine have proved useless in the hands of Charcot, while the disease was aggravated by chloride of gold and phosphate of zinc. Nitrate of silver appeared to produce a favourable effect, which, however, was maintained only for a short period. Hammond recommends three-quarters of a grain of chloride of barium three times a day. The most promising treatment appears to be the persistent application of the galvanic current to the spine, hydropathy, nitrate of silver, phosphorus, cod-liver oil, and nourishing but unstimulating diet.

CHAPTER II.

CHOREA, AND MÉNIÈRE'S DISEASE.

(I.) CHOREA.

Two forms of chorea are often described, named respectively *chorea major* and *chorea minor*, but the former is only an aggravated form of hysteria, and consequently the latter alone will here be described under the name of *chorea*. It is a disease which chiefly attacks children, and is characterised by irregular clonic spasms of certain groups of voluntary muscles.

§ 898. *Etiology.*—Heredity plays an important part in the production of chorea, but the transmission is probably always indirect. The patient may inherit either a susceptible nervous system, or the rheumatic diathesis—rheumatism being one of the most frequent and important causes of the disease. Age is an important predisposing cause of chorea, the disease generally occurring during the period of bodily development. Isolated cases of the affection have been observed in infants at the breast, while it is not uncommon in young women. Sée states that three-fourths of the cases observed in the Children's Hospital in Paris occurred in girls.

Everything which augments the excitability of the nervous system during the period of sexual development, as premature excitement of the sexual passion, onanism, or any undue emotional disturbance, increases the tendency to chorea. The most usual predisposing causes of the affection in adults are pregnancy, menstrual disorders, and chlorosis.

Very little is known with regard to the geographical distribution of the disease, or the influence exerted by the different

seasons and atmospheric changes in its production. That some causal relationship exists between articular rheumatism and chorea has been known since the beginning of the century, but the true nature of this relationship is not yet accurately ascertained. The frequent occurrence of cardiac murmurs in chorea was noticed by Addison and subsequently by Todd. Out of 299 cases collected by Hughes and Brown, there were 104 whose history could be carefully ascertained, and of these only 15 had not suffered from rheumatism or had not developed cardiac murmur. Out of 128 patients suffering from chorea Sée found 64 who had suffered from articular rheumatism. Chorea occurs frequently after scarlatina, a fact which may probably be explained by the frequency with which the latter is followed by rheumatism.

The relationship between pregnancy and chorea is very obscure, inasmuch as it is only in a small number of cases that the attack has been preceded by rheumatism or endocarditis. Chorea occurs most frequently during first pregnancies, although it is sometimes repeated in the same patient in subsequent pregnancies, and the majority of those affected are from twenty to twenty-three years of age. It appears more frequently during the first than the second half of pregnancy, but sometimes it begins in the later months and may continue up to the time of delivery or even beyond it.

Of the exciting causes of chorea the most frequent and important are emotional disturbances, such as fright, sorrow, and discontent. Hysterical girls and those who are strongly predisposed to chorea, or who have already suffered from an attack, may acquire the disease by imitation of those suffering from it.

§ 899. *Symptoms.*—The development of the characteristic phenomena of chorea is generally preceded by various premonitory symptoms for a variable period of days or weeks. The most usual of these are afforded by changes in the character and disposition of the patient, who becomes forgetful, inattentive, fretful, and discontented or apathetic, while the intellectual powers are impaired. The spasmodic movements may in some cases be preceded by paralytic phenomena. A case under my

care at the Manchester Southern Hospital was admitted as one of hemiplegia, and it was only two days after admission that the characteristic choreic movements made their appearance. The irregular spasmodic muscular contractions of chorea are, indeed, not unfrequently ushered in by a slight dragging of one of the lower extremities, with a tendency to walk in a curved line and liability to let objects fall from the hand. These manifestations of the approaching disease are probably due, in some degree, to irregular muscular contractions, but are largely dependent upon muscular weakness. The characteristic choreic movements generally begin in the small muscles of the face and in those of a hand. They consist at first of grimaces and other contortions of the face, and slight jerking movements of the fingers and at the wrist joint, with pronation of the forearm, when the patient is conscious of being observed or is excited from any other cause; these soon increase in intensity and persist during repose.

The irregular contractions soon extend so as to involve all the voluntary muscles, when the affection may be called *general chorea,* or they remain more or less limited to the muscles of one-half of the body, when the disease is called *unilateral chorea* or *hemichorea.*

General Chorea.—When once the disease is fully established the symptoms are quite characteristic, and it would be difficult to find phrases more expressive of the disorderly muscular movements than "insanity of the muscles," adopted by Bellingham, and "folie musculaire" by Bouillaud.

The features undergo every variety of contortion. The brow is knit and immediately expanded; the eyebrows are elevated and the next moment depressed, or one may be elevated while the other is lowered; the eyelids open and close alternately; the eyeballs are quickly rotated in different directions; the labial commissures are suddenly drawn outwards, and as quickly retracted. These opposite movements succeed one another with such rapidity that the face presents in quick succession the most contradictory expressions, such as those of delight, vexation, and anger.

The tongue is thrust out of the mouth, and quickly retracted and rolled about from side to side; the jaws are separated and

closed, it may be with so much violence that teeth are broken, or the tongue and cheeks are severely bitten; lateral displacements of the lower jaw are frequently observed, and the head is jerked suddenly from one side to the other, while the facial grimaces by which the movements of the jaws, tongue, and head are accompanied add to the comical appearance presented by the patient.

The superior extremities execute every variety of movement, the shoulders are elevated, then lowered, and immediately afterwards drawn backwards or forwards; the arm and forearm are moved at the shoulder and elbow joint in every possible direction; the hand is alternately pronated and supinated, flexed and extended; and the fingers are at one moment extended and spread apart and at the next flexed. These movements are combined in such varied ways that a gesticulatory agitation is produced which defies description.

The muscles of the trunk are implicated, and their unequal disorderly contractions produce sudden lateral and antero-posterior deviations of the vertebral column, which in certain cases may be so violent that the patient is thrown from his chair or out of bed. The muscles of the lower extremities also undergo irregular contractions, causing eversion and inversion of the foot and various contortions of the toes, as well as movements at the larger articulations. Choreic movements cease as a rule during sleep and under the influence of chloroform, but in aggravated cases they may continue during sleep; the pupils are usually dilated, and their reaction to light is diminished. The respiratory rhythm becomes irregular and jerky, and on laryngoscopic examination the vocal cords have been observed to act in an irregular and disorderly manner (von Ziemssen).

Most of the irregular movements just described may occur during repose, although they are much exaggerated when the patient is under observation or excited in any way. When, however, the patient endeavours to execute a voluntary movement, the motor disorder becomes, as a rule, greatly increased. A distinction has been drawn by Dr. Gowers between the choreic movements that occur during repose, and the motor inco-ordination observed during attempts at voluntary movements which may be called choreic ataxia; but whether this

distinction be valid or not, it is undoubted that great motor co-ordination during attempts at voluntary movement may be present in cases in which the choreic movements of repose are slight; and, conversely, the voluntary inco-ordination may be slight in cases in which the choreic movements of repose are excessive. In cases of moderate intensity delicate manual operations, such as those required for writing, sewing, and playing upon musical instruments, alone become impossible; while operations, like eating, requiring less complicated adjustments for their performance, are still effected, although in an imperfect and round-about manner, and after frequent interruptions from the involuntary contraction of antagonistic muscles.

In aggravated cases it becomes impossible to execute almost any intended movement. When the patient endeavours to carry anything to his mouth, such as a glass of water, the progress of his arm is arrested by a series of jerks and contradictory movements which may scatter the contents of the glass in every direction; the patient cannot button and unbutton his clothes; the maintenance of the erect posture is difficult or impossible; and even in the recumbent posture he is not free from the danger of being thrust out of bed; his clothes and linen become worn out by constant rubbing; and the skin over the prominent bones becomes erythematous and may ulcerate.

On the patient being asked to show the tongue he protrudes it with a jerk, the mouth being opened to an unnecessary extent; the tongue is immediately withdrawn, while the mouth and jaws close upon it with violence. When the patient endeavours to speak the convulsive action of the facial muscles becomes aggravated; his articulation is irregular, jerky, drawling, or stammering; his voice is monotonous; and in aggravated cases his speech is so disordered as to be almost if not entirely unintelligible. Spasmodic contraction extends to the muscles of mastication and deglutition, and consequently these functions are performed imperfectly and with difficulty.

Hemichorea.—The spasmodic phenomena are sometimes limited to the muscles of one-half the body, the unilateral variety occurring in about one-fifth of all cases. Some authors state that the left and others that the right is more frequently

affected, but there does not appear to be a great difference between their relative liability. Broadbent asserts that the muscles bilaterally associated in their actions, and which are comparatively spared in hemiplegia, are affected to some extent on both sides in hemichorea.

The other symptoms of hemichorea are the same as those of general chorea, and do not require separate description.

Although spasmodic motor disturbance constitutes the most characteristic feature of chorea, it must not be forgotten that a certain degree of muscular weakness is always present, this being easy of recognition in cases of hemichorea. Indeed, towards the termination of the affection or during its course, the choreic movements may be replaced by a more or less complete hemiplegia or paraplegia, and we have already seen that paralytic symptoms may precede the development of the characteristic movements.

The electric excitability of the nerves and muscles is said to be increased to both currents, a fact more readily proved in hemichorea than in the bilateral variety (Rosenthal, Gowers).

The reflex excitability is said by some authors to be increased and by others to be diminished.

Sensory disturbances are not frequently observed in chorea. Painful points have been found at times in the course of the nerve trunks of the affected region, while tenderness on pressure over the spinous processes of some of the vertebræ is occasionally met with. At other times cutaneous hyperæsthesia or hyperalgesia distributed over half or the whole of the body has been observed, but anæsthesia of like distribution is more common.

Vaso-motor and secretory disturbances are wanting, there are no special trophic changes, and the general health does not suffer, except in aggravated and chronic cases, in which the constant agitation and want of sleep induces a condition of anæmia and general marasmus.

Psychical disturbances are invariably observed in chorea. The mental depression and irritability with which the disease begins usually increase during its course. The patient is obstinate, taciturn, and even violent towards parents and attendants. He suffers from impairment of memory, incapacity for thinking, and general intellectual weakness. At times there may be

hallucinations of sight, especially at night, succeeded by a maniacal delirium, and according to the observations of Marcé, half of the cases, in which this delirium supervenes, terminate fatally.

The pulse may be irregular, and the patient suffers from palpitation, while a physical examination of the heart generally reveals the presence of endocardial murmurs, either arising from disease of the valves or of functional origin. Chorea is not usually accompanied by pyrexia, but in severe cases, where there is violent muscular action, elevation of temperature is not uncommon. When it is associated with acute rheumatism, more or less fever is necessarily present.

§ 900. *Course, Duration, and Results.*—Chorea, as a rule, runs a chronic course, lasting in the majority of cases from six to eight weeks; while aggravated cases may continue for four to five months. The disease may, indeed, last many years, and it is probable that such cases are the result of permanent anatomical lesions in the nervous system.

The course of the disease is seldom uniform, and relapses are frequent; slight emotional disturbances often aggravate the symptoms or induce a relapse during convalescence.

Chorea frequently recurs at varying intervals. The recurrent attack may be induced by emotional disturbance, by pregnancy, or by the presence of an acute disease. Most of the relapses occur during puberty, but among persons who were choreic at or before this period they may appear at from twenty to thirty years of age or later.

The disease generally terminates in complete recovery, but a nervous fidgetty manner, exhibiting itself in slight grimaces, needless haste, and want of precision in executing certain movements, often remains for years.

Termination in imperfect recovery is rare. Sometimes, however, a few symptoms of chorea may persist, paralysis may develop in the half of the body which has been most affected, or permanent mental disease, such as mania, melancholia, or general paralysis of the insane become established. Death is extremely rare in children in uncomplicated cases, but when the disease is complicated with rheumatism and endocarditis it

is not unfrequently fatal. Sée found a mortality of 5·7 per cent in the chorea of children; whilst the statistics of Wenzel give a mortality of 27·3 per cent in the chorea of pregnant women. The cause of death in chorea is generally to be assigned to various complications, but occasionally to the intensity of the disease itself. In the latter cases the symptoms are unusually acute and violent from the first, and attain excessive severity in a few days; the choreic movements cease either suddenly or gradually and collapse sets in, along with complete muscular relaxation and involuntary evacuations, and death follows preceded by coma.

§ 901. *Morbid Anatomy.*—In the old observations of Cruveilhier, Romberg, and others, foci of softening were found in various parts of the brain, but the absence of a careful microscopical examination greatly diminishes the value of these records. Brown-Séquard and Gendron observed softening of the spinal cord. Tuckwell in 1867 found, at the post-mortem examination of a patient dead of chorea, fibrinous vegetations on the valves of the heart, a branch of the middle cerebral and another of the posterior cerebral artery occluded by emboli, and foci of red softening in the cortex of the brain corresponding to the distribution of the occluded vessels. It may be noticed in passing that Kirkes had suggested in 1850 and again in 1863 that the well-known relation between rheumatism and chorea would be found in the endocarditis caused by the former, giving rise in its turn to multiple embolism of the vessels of the brain. Ogle published in 1868 an analysis of ninety-six cases of chorea. Sixteen of these were fatal, and a post-mortem examination of them was obtained. Cardiac lesions, consisting of fibrinous deposits on the valves, were found in ten cases only. In six congestion of the nervous centres was noted. In a girl of seventeen who died from maniacal chorea during pregnancy, hyperæmia of the surface and softening of other parts of the brain were observed. The anterior column of the spinal cord in the lower dorsal region, on a level with the ninth dorsal nerves, was swollen and softened. A microscopical examination, conducted by Lockhart Clarke, revealed softening of the white substance and extravasations of blood, with granular exudation.

Steiner, in 1868, published the results of the post-mortem examination of three cases of chorea. He found cerebro-spinal anæmia, effusion of serum into the spinal canal, and proliferation of the connective tissue in the upper half of the spinal cord in one case, and in another hyperæmia of the spinal cord and brain, including their membranes, and an accumulation of fluid blood about the roots of the cervical and upper dorsal nerves. Evidences of endocarditis were found only in one of these cases. Fatal cases of the chorea of pregnancy, in which no signs of endocarditis were observed, have been recorded by Wilks, Lawson Tait, and Barnes. Aitken found the specific gravity of the corpora striata and optic thalami of a person dead of chorea much less than that of other parts of the same brain, and of the same parts in healthy brains. Numerous changes were found in the brain and spinal cord by Meynert in chorea. The main changes consisted of hyaline swelling with molecular degeneration of the protoplasm of the cells of the cortex of the brain, partial sclerosis of the cells of the cortex of the island of Reil and of the basal ganglia, and multiplication of the nuclei of the nerve cells. He also found great multiplication of the nuclei of the neuroglia and swelling of Deiter's cells in the spinal cord. Elischer found, in a pregnant woman dead of chorea, nuclear proliferation, hyperplasia of the connective tissue, and thickening of the tunica adventitia of the small vessels in the corpus striatum, and division of the nuclei of the nerve cells in the claustrum. The spinal cord presented thickening and nuclear proliferation in the walls of the vessels, thickening of the ependyma of the central canal, and nuclear proliferation of the connective tissue around the nerve cells of the grey matter. The cells themselves presented a dull appearance, were destitute of nuclei, and filled with pigment. The white substance was hyperæmic, and the lateral and posterior columns contained a fibrillated tissue with abundant nuclei. The fibres in the peripheral nerves were diminished in number.

A valuable paper on "The Pathology of Chorea" has recently been contributed by Dr. Dickinson, in which the results of the post-mortem examination of seven fatal cases of chorea are recorded. The changes described consist of dilatation of the medium-sized arteries and veins throughout the substance of

the brain and spinal cord, exudations or small hæmorrhages indicated sometimes by the presence of blood crystals, into the tissues surrounding the distended vessels, and in chronic cases patches of sclerosis in the neighbourhood of the vessels. These changes were most pronounced in the corpora striata and optic thalami, the anterior perforated spaces, and at the junction of the posterior grey horns and central columns of the spinal cord, especially in the upper dorsal and cervical regions. In one case the central canal of the dorsal region of the spinal canal was greatly distended by bloody serum. "Speaking generally," says Dr. Dickinson, "the chosen seats of the choreic changes are the parts of the brain which lie between the beginning of the middle cerebral arteries and the corpora striata—the partes perforatæ; and in the cord the central portion of each lateral mass of grey matter comprising the root of each posterior horn." It will be evident how these observations of Dr. Dickinson bear out the theory advanced in these pages as to the great pathological importance of the parts of the nervous system which I have termed accessory.

A microscopical examination of the nervous system in a case

FIG. 283.

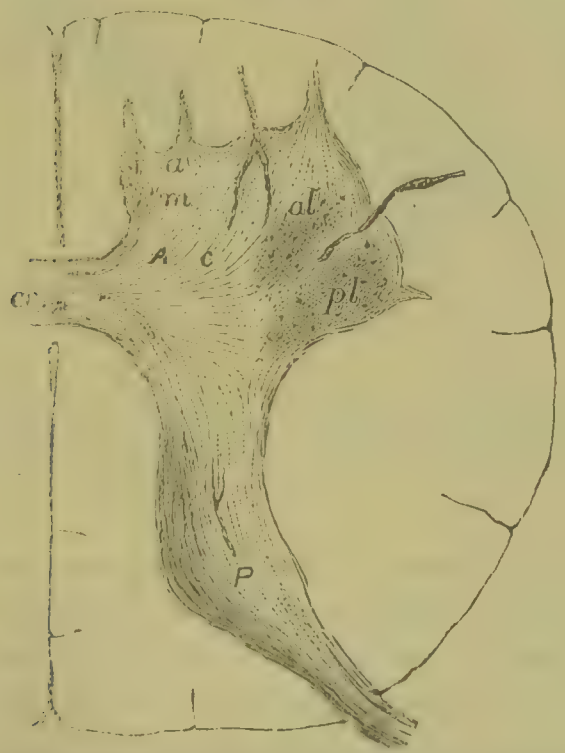


FIG. 283 (Young). *Section of the Cervical Region of the Spinal Cord from a case of Chorea.*—*cc*, Central canal; A and P, Anterior and Posterior horns respectively.

of fatal chorea, which I had the opportunity of making, enables me to confirm to a large extent the statements of Dr. Dickinson. In the cases he describes periarterial erosions and hæmorrhages occurred around the central artery and its primary branches, while in my case the most pronounced changes were found in the anterior and antero-lateral arteries. All the vessels of the cord were more or less distended with red blood corpuscles, but in some sections a fibrinous plug was observed in the anterior or antero-lateral arteries, the vessel being distended by it (*Fig.* 283). Spots of necrotic softening were observed in the corpora striata. A section of the spinal cord from a case of chorea was exhibited by Dr. Bury at the Manchester Microscopical Society, in which the periarterial exudations and hæmorrhages round the branches of the central artery were distinctly shown (*Fig.* 284),

Fig. 284.

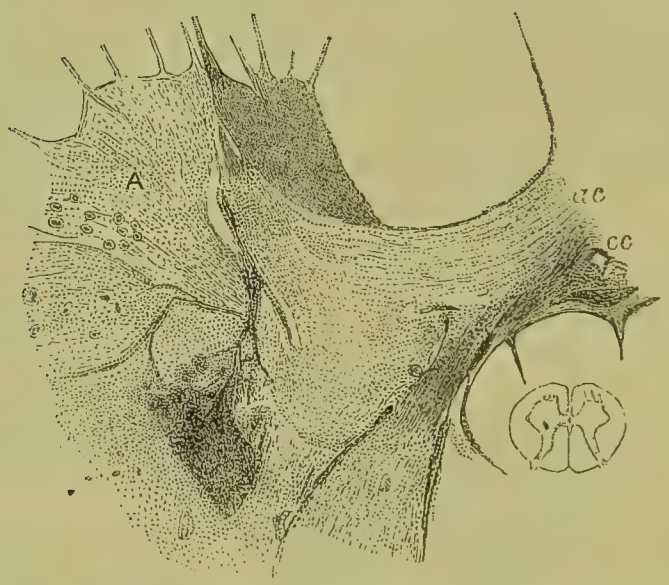


Fig. 284 (Bury). *Section of the Anterior Grey Horn of the Cervical Enlargement of the Spinal Cord from a case of Chorea that died on the fourth day of Scarlet Fever.—cc,* Central canal ; *ac,* Anterior commissure ; A, Anterior horn.

and corresponded accurately to the description and drawings of Dr. Dickinson. But although no decided changes were observed in the tissues near the central artery of the spinal cord in the case examined by me, I was struck with the alterations presented by the accessory cells of the anterior grey horns in comparison with the fundamental cells. The accessory cells could not be

seen with a low power, and are therefore not represented in the diagram; but with a higher power they appeared shrivelled, their protoplasm was granular, the nuclei were obscure, and many of the processes were indistinct or absent. The larger fundamental cells did not appear much altered. Of twenty-two fatal cases of chorea collected by Dr. Dickinson the heart was found healthy in five only, and of these one only was a child. As Dr. Dickinson remarks, endocarditis appears to be an almost invariable accompaniment of fatal chorea in children, while beading of the mitral valve with lymph is probably present in every instance of cardiac complication. In the case observed by me the free edges of the mitral valve were fringed by a row of fibrinous beads.

§ 902. *Morbid Physiology.* — In 1868, Broadbent and Hughlings-Jackson almost simultaneously advanced the hypothesis that the corpus striatum and optic thalamus are the main centres in which the lesions in chorea are localised. Hughlings-Jackson surmised that the convolutions of the cortex situated near the corpus striatum were also involved in the disease. The observations of Meynert, Dickinson, and others confirmed by the case examined by myself, prove that the lesions are widely distributed throughout the brain and spinal cord.

An endeavour has been made to determine the localisation of the lesion in chorea by experimental investigation. Chauveau divided the spinal cord close to the skull in a dog suffering from general chorea, and found that the choreic movements continued unabated until the death of the animal several hours after the operation. The convulsive movements of the tail and posterior extremities ceased immediately on the spinal cord being divided in the dorsal region. From the results of these experiments, Chauveau concluded that the spinal cord is the seat of the lesion. Similar experiments were conducted by Longet, Bert, and Carville, and the same conclusion arrived at.

Legros and Onimus found that irritation of the posterior columns of the exposed cord with the scalpel increased the twitchings. The choreic movements ceased on the cord being exposed to a current of cold air, and reappeared on its being subsequently moistened with warm water. Section of the posterior roots did not exert any influence on the choreic movements. Partial removal of the posterior cornua and columns weakened, and complete excision of them abolished the movements. An

ascending galvanic current through the cord increased the intensity and frequency of the contractions; while a descending current weakened them considerably. The authors conclude from these experiments that the morbid process in chorea implicates the nerve cells of the posterior grey cornua of the spinal cord, or the nerve fibres which unite them with the cells of the anterior cornua. Rosenthal injected fine flower seeds into the left carotid artery of a dog suffering from choreic movements of the right fore-leg. All voluntary movements were instantly arrested, but choreic movements became much stronger in the affected extremity, and involved the eyelids and tail, lasting until the animal died two days subsequently. The autopsy revealed encephalitis of the left anterior lobe, softening of the left corpus striatum, and embolism of the left Sylvian artery. A microscopic examination conducted by Dr. Scheiber showed spots of proliferated connective tissue in many parts of the brain substance. Canine chorea is by no means the same disease as that of the same name in man, and it would be hazardous to attach much importance to any of these experiments.

The nature of the lesion in chorea has been a subject of as much controversy as its localisation. The relation between rheumatism and chorea had been known for a long time, and Bright went so far as to assert that rheumatic pericarditis was the most frequent cause of chorea. In 1850, and again in 1863, Kirkes suggested that endocarditis was the causal link between rheumatism and chorea. According to this opinion, chorea was caused by embolic particles washed off from the inflamed endocardium and arrested in the vessels of the brain and spinal cord. Hughlings-Jackson adopted this view, and in 1868 he advanced the opinion that chorea was caused by multiple embolism of the nutritive arteries of the basal ganglia and convolutions of the cortex of the brain situated near to the corpus striatum. It cannot be doubted that embolism of the vessels of the nervous centres does occur at times in chorea, inasmuch as some of the vessels of the brain have been found actually occluded by an embolus in fatal cases, while the condition liable to occasion embolism is present in a very large proportion of fatal cases. On the other hand, in some fatal cases there has been an entire absence of cardiac complication. Again, of the large majority of cases which recover, although a cardiac complication is frequently present, yet this is by no means invariable. It may, therefore, be concluded that although chorea may be caused by multiple embolism of the vessels of the nervous

system, yet the affection may occur in the absence of embolism, and it is not, therefore, the essential condition upon which the disease depends. Similar reasoning applies to the opinion of the humoral pathologists, who believe that chorea is caused by the rheumatic diathesis, or by the poison of rheumatism circulating in the blood, producing irritation of the tissues of the nervous system. Chorea may occur in the absence of a history of active rheumatism. "We see in chorea," says Dr. Dickinson, "a widely distributed hyperæmia of the nervous centres, not due to any mechanical mischance, but produced mainly by causes of two kinds—one a morbid, probably a humoral, influence, which may affect the nervous centres as it affects other organs and tissues; the other, irritation in some mode, usually mental, but sometimes what is called reflex, which especially belongs to and disturbs the nervous system, and affects persons differently, according to the inherent mobility of their nature."

To turn to the first factor, it must be remembered that distension of the blood vessels of the nervous system after death by no means proves the existence of an active hyperæmia during life. The conditions usually present, such as cardiac disease, are such as to cause anæmia of the nervous system, and the phenomena of chorea are best explained on the supposition that the excess of irritability of the nervous centres is caused by defective nutrition of their tissues.

With regard to the second factor, the profound mental impression causing chorea is usually fright, one of the depressing passions, which is certainly more calculated to exhaust the irritability of the nervous system than to maintain it in a state of continuous activity through irritation. Reflex irritation is also more likely to act by causing anæmia rather than hyperæmia.

The third co-operating factor—inherent instability of the nervous centres—is a very important one. It is probable that the children in some families inherit an unstable nervous system, which renders them liable to be directly affected by chorea, but this has not been definitely proved as yet. It is not doubted, however, that an inherent tendency to develop the disease at a certain age exists. As Dr. Dickinson remarks, "Every period of life has its own regions of nervous susceptibility: in

childhood the motor; in adolescence the emotional; in advancing years the mental, and coevally, or nearly so, that part of the nervous mechanism which instigates glycosuria. Much the same mental impression may make a child choreic, a girl hysterical, or a man diabetic." In chorea the sensory and psychical functions of the nervous centres are disturbed, but the great weight of the disease falls upon the motor functions, and of the latter the last-developed co-ordinations of the muscles of the hand and of the face and tongue are implicated in a special degree.

We have already seen that the most pronounced lesions in chorea are found in what I have called the embryonic portions of the motor area of the cortex and corpus striatum, and of the motor grey matter of the cord, and consequently whether chorea be viewed functionally or structurally, it appears to be a widely distributed disease of the nervous centres, in which the cerebro-spinal motor functions and mechanisms are specially affected, the accessory functions and structures being affected at an earlier period and more profoundly than the fundamental functions and structures. I came to this conclusion after making a microscopical examination of the nervous system in the case above referred to, and was not aware at the time that Hughlings-Jackson, approaching the subject from the clinical standpoint, had come to practically the same conclusion upwards of ten years before. "Since the disorder in chorea," he says, "is of movements which are acquired, and which are probably only fully learned by a long apprenticeship, I used to suppose that the nervous arrangements for these movements were but partially developed in children, and that the motor processes involved, and their arterial integration, would be, so to speak, caught in a stage of incomplete development—in short, that their centres were diseased when *half*-educated." In the same paper he attributes the symptoms to "under-nutrition" of the tissues affected.

§ 903. *Diagnosis.*—The diagnosis of chorea can hardly present any difficulty. Paralysis agitans occurs in the later years of life, and the attitude of the patient and his mode of locomotion are so characteristic that this disease is no more

likely to be mistaken for chorea than is the tremor associated with old age, and with chronic poisoning by alcohol, lead, or mercury. The spasmodic movements occurring in groups of muscles supplied by certain nerves, such as convulsive tic, and those occurring in definite groups of muscles engaged in performing certain actions, as in the case of writer's cramp, are also separated from the movements of chorea by broad lines of demarcation.

§ 904. *Prognosis.*—The prognosis of chorea is, as a rule, favourable. It becomes grave, however, when the movements are so violent as to exhaust the patient, cause sleeplessness, and prevent sufficient food being taken, or when there is delirium. The chorea of pregnancy is much more fatal than that which occurs about and before puberty.

§ 905. *Treatment.*— In the treatment of chorea the diet should be carefully regulated; and any source of reflex irritation, such as intestinal worms, should be removed. If anæmia be present, iron may be given, either alone or along with cod liver oil; while if rheumatism complicate the case, salicylate of soda must be administered. The use of the hot vapour bath has been much praised in the treatment of chorea, and it is worth a trial in cases in which a rheumatic diathesis can be traced, even if there be no active rheumatism at the time of the attack. The child should be immediately removed from school, all intellectual work suspended, and even bodily exertion avoided in the early stage of the disease.

The medicines which appear to do most good are the nervine tonics, and of these arsenic is probably the best. Ziemssen recommends a dose of from five to eight drops of Fowler's solution for children, and eight to twelve drops for adults. Most practitioners will be inclined to begin with a smaller dose and gradually increase it. Iron may be combined with arsenic if the patient be anæmic and the stomach bears it well. If the arsenic has not produced a decided improvement in the symptoms within a period of a week or ten days, zinc may be substituted, the sulphate being the most convenient preparation and as successful as any other. A dose of two or three grains

may be given at first three times a day, but this dose must be gradually increased daily; if nausea or vomiting be produced, the dose should be slightly diminished for a few days until tolerance is established. After this it should be again gradually increased until the symptoms begin to improve, and then continued without alteration until improvement ceases or the disease subsides. The tolerance for the drug becomes so great after a short time that 15 to 20 grains may be given three times a day to a patient 15 years of age, without nausea or any other ill effects being produced. I have made a fair trial of the valerianate and the bromide of zinc, but have not found these salts to be in any way superior to the sulphate. Bromide of potassium does not appear to me to exert any favourable influence on the progress of the disease, but it may be usefully administered with or without chloral when psychical disturbance and sleeplessness are prominent symptoms.

(II.) MÉNIÈRE'S DISEASE.

(Auditory Vertigo.)

§ 906. *Definition.*—Ménière's disease is characterised by attacks of vertigo, associated with noises in one or both ears, and partial deafness.

§ 907. *Etiology.*—All the causes which produce disease of the peripheral organ of hearing may occasion auditory vertigo, and they need not, therefore, be enumerated here.

§ 908. *Symptoms.*—The characteristic symptoms of Ménière's disease are sometimes preceded by partial deafness, earache, and other indications of a local lesion of the peripheral organ of hearing. In other cases the patient is suddenly attacked with noises in one ear, and a feeling of giddiness, attended by faintness, nausea, and vomiting. The attack passes off in a few seconds or minutes, but recurs after a variable period, the paroxysms becoming more aggravated and more frequently repeated as the disease advances.

The noise is sometimes heard in both ears, but it is probably always more pronounced on one side than the other. It is

sometimes compared to the loud whistling of a steam engine, at other times to a succession of explosions, and it is often described as a continuous humming or buzzing. The noises in the ear cease with the attack of vertigo, in recent or slight cases; but in aggravated forms of the affection they continue to distress the patient during the intervals.

The attack of vertigo varies in duration and intensity. In slight cases it consists of a momentary feeling of swimming in the head, in severe cases each paroxysm may extend over a period of ten or more minutes, while in still more aggravated cases the feeling of uncertainty and giddiness is never absent during waking hours, and every effort on the part of the patient to assume the erect posture determines the vertigo along with nausea and vomiting. During the paroxysm the patient feels as if he were falling forwards, backwards, or laterally, or were rotating round a vertical or horizontal axis; he staggers and clutches at surrounding objects for support, or actually falls in a direction corresponding with his sensations. The patient during the paroxysm feels faint; the skin becomes pallid, cold, and covered with sweat; the pulse is feeble and flickering; and there is an intense feeling of nausea, which often terminates in vomiting, when the attack usually comes to an end. Actual syncope may occasionally occur, and there may be transitory loss of consciousness, but this is exceptional. The patient, however, during the paroxysm suffers from confusion of ideas, and in the worst cases any unusual intellectual effort may determine an attack of vertigo.

Oscillatory movements of the eyeballs have been observed by Schwabach and Hughlings-Jackson during the attack. In the case observed by Schwabach, as quoted by Hughlings-Jackson, the eyes were turned towards the affected side and slightly downwards; while in the case observed by Hughlings-Jackson himself, "each eye was partially and very slightly rotated to the right in frequent jerks from left to right," the left ear being the diseased one. During the attacks there was an apparent displacement of objects with reference to the patient; and in the case described by Hughlings-Jackson, objects were said to revolve, contrary to what might have been expected, from

left to right, or in the same direction as the rotation of the eyes. An instructive case of this disease is recorded by Mr. Lewis Mackenzie and quoted by Hughlings-Jackson, in which the patient—a medical man—was much distressed by continuous noises in the right ear, following the discharge of a heavily-loaded gun near it, and, along with the other more usual symptoms of auditory vertigo, there was manifested a constant tendency to walk to the left.

§ 909. *Course, Duration, and Terminations.*—The paroxysms of vertigo come on at first at irregular intervals; they increase gradually in frequency and intensity, and in aggravated cases the patient suffers continuously from some degree of vertigo, while he is liable to paroxysmal exacerbations of great severity. The noises in the ears may cease at first during the intervals, but after a time become constant. The sense of hearing becomes gradually diminished, and ultimately complete deafness of the affected ear is established, when, fortunately, the paroxysms of vertigo and all the distressing symptoms of the disease cease.

§ 910. *Morbid Anatomy and Physiology.*—Several post-mortem examinations have revealed the presence of inflammatory exudation in the semicircular canals. The symptoms under consideration are, however, often associated with disease of the middle or external ear, but in such cases some indirect influence is probably exerted on the labyrinth.

§ 911. *Diagnosis and Prognosis.*—Auditory vertigo is liable to be mistaken for the vertigo associated with gastric disease or sexual excess, the diagnosis being rendered more difficult by the fact that a considerable degree of deafness and noises in the ears may be present in the latter. In auditory vertigo the noises are unilateral, the feeling of vertigo is very intense, and is accompanied by a sensation as if the body had undergone actual displacement; these symptoms never occur to the same degree in the vertigo of dyspepsia or sexual excess. Paroxysms of auditory vertigo may be mistaken for epileptic attacks, and can only be distinguished by a careful examination of the

symptoms. The prognosis is grave as far as ultimate recovery is concerned, but the symptoms disappear when complete deafness is established.

§ 912. *Treatment.*—If the symptoms depend upon disease of the external or middle ear, the patient should be placed under the care of the specialist, and when the local disease is not accessible to treatment, relief may be obtained by rest in the recumbent posture. The administration of four or five grains of quinine three times a day appears to have produced great amelioration of the symptoms in several cases (Charcot, Hughlings-Jackson).

CHAPTER III.

EPIDEMIC CEREBRO-SPINAL MENINGITIS, TETANUS, AND HYDROPHOBIA.

(I.) EPIDEMIC CEREBRO-SPINAL MENINGITIS.

§ 913. *Definition.*—Epidemic cerebro-spinal meningitis is an acute epidemic fever, characteristic symptoms of which are caused by a purulent inflammation of the spinal and cerebral pia mater.

§ 914. *History.* — This disease probably prevailed in ancient times, but we have no reliable accounts of it before the present century. The first epidemic prevailed in Geneva, in the early months of 1805. Then followed epidemics in Grenoble (1814), Vesoul (1822), and Dorsten on the Lippe (1833). The disease appeared in Southern France in 1837, and remained confined for many years to the barracks. About the time that it attained its highest development in France the epidemic extended to Italy, and prevailed there from 1839 to the spring of 1845.

In 1844 a transient epidemic occurred in Gibraltar, the following year in Denmark, and in 1846 it appeared in the workhouses of Ireland, while a few cases were observed in Liverpool. The disease appeared in Sweden in 1854 and reappeared in 1861; there was an epidemic in Norway in 1859—1860.

The disease may be traced back in the United States to the beginning of the century, but it made active progress during the year 1842, and prevailed with great intensity during the civil war. In the last few years it has appeared in Canada, and it has never entirely disappeared from the United States, declaring itself either in sporadic cases or in small epidemics.

Sporadic cases have been observed in the United Kingdom; but the most fatal epidemic began in Ireland in March, 1866, and attained its chief development in the following winter. Its effects were almost entirely confined to Ireland, the worst cases occurring in Dublin. In 1876 a slight epidemic occurred in Birmingham.

§ 915. *Etiology.*

Predisposing Causes.—When the disease was first noticed in Southern France in 1837, and in the subsequent outbreaks up to 1849, it was almost entirely confined to soldiers. Raw recruits were specially liable to be affected, and this liability was probably largely due to great physical exertion and an overcrowded condition of the garrisons. In subsequent outbreaks, however, in France and elsewhere no special liability to the disease has been manifested amongst those engaged in particular employments.

In the earlier epidemics males were almost exclusively attacked; and, although subsequent observation has not confirmed the idea that the disease is peculiar to men, it is much more common in them. Age does not appear to have any particular influence in the production of the disease. In some epidemics children, in others young people, and still in others adults of from thirty to thirty-five have been attacked in greatest proportion.

The disease is especially prevalent in the cold months of the year, and, notwithstanding some apparent exceptions, it is undoubtedly more prevalent amongst the poor and ill-fed than the affluent classes. In a large number of epidemics it almost exclusively prevailed amongst the inmates of prisons, workhouses, and overcrowded garrisons.

§ 916. *Symptoms.*—Epidemic cerebro-spinal meningitis may be divided into four varieties: (1), the *simple;* (2) the *fulminant;* (3), the *purpuric;* and (4), the *abortive* forms.

(1) *Simple Epidemic Cerebro-Spinal Meningitis.*—Premonitory symptoms are sometimes observed, consisting of loss of appetite, lassitude, and neuralgic pains in the back and abdomen. As a rule, however, the patient is suddenly seized, while following his ordinary occupations, or at play, with shivering, vomiting, and headache (Burdon-Sanderson). Profuse and uncontrollable vomiting is almost a constant symptom, the ejected matters consisting at first of half-digested food and subsequently of mucus stained with bile. Delirium now supervenes, sometimes so violent in character that it is necessary to

place the patient under restraint, but he is usually apathetic and drowsy, and only talks of imaginary objects when roused.

The patient soon complains of an agonising pain in the occiput and nape of the neck, which may extend along the spine and is aggravated by movement and pressure. The abdominal muscles, as well as those of the back and loins, are acutely painful, and any movement rendering them tense occasions great pain. The skin becomes extremely sensitive, and severe pain is felt in the limbs, but it is difficult to determine how much is due to cutaneous or muscular hyperæsthesia. The head is retracted, partly from spasm of the muscles of the nape of the neck, but mainly as an instinctive means of relaxing the muscles in order to relieve pain. In no case coming under the observation of Dr. Burdon-Sanderson were the contractions of the muscles of the back of the neck of such a character as to be correctly called tetanic. "The patients," says Dr. Sanderson, "invariably lay on their sides, with their knees drawn up so as to relieve the abdominal muscles, and with the face looking towards the head of the bed, and excessive pain was produced whenever the body was moved in such a way as to extend the painful muscles, and more particularly when the patient was lifted in bed." The period of invasion lasts from one to three days, and is then followed by the stage of depression.

The mental confusion and low-muttering delirium, which is present in the stage of invasion, now gives place to stupor, which in fatal cases ends in profound coma. The patient lies in a somnolent condition, although often able to answer questions when roused. The symptoms are liable to undergo considerable fluctuations; at times the sopor predominates, at other times there are restlessness and nocturnal delirium, and the patient continues to complain of pains in the back of the neck and in the loins. Tremors are observed in the extremities, the pulse is slow, the face is livid, the pupils dilated or contracted, and strabismus, amblyopia, or deafness is not unusually present. In several children seen by Dr. Burdon-Sanderson the symptoms, shortly after the cessation of the initial period, were very similar to those of tubercular meningitis. An exanthematous eruption appears about the mouth, either like that of measles or scarlet fever, and occasionally herpetic in character;

it spreads upwards over the eyelids and ears, and downwards over the chin and neck.

The degree of pyrexia varies greatly in different cases, but the temperature usually ranges from 100° F. to 103° F., or in fatal cases to 105° F. or higher. The tongue may be clean, the bowels are usually constipated, and the abdomen is retracted. The urine is frequently albuminous, destitute of chlorides, and contains a relatively large proportion of urates, while polyuria and saccharine urine have occasionally been observed.

In unfavourable cases the coma increases and becomes associated with more pronounced paralytic symptoms, such as ptosis, strabismus, and paresis of the extremities. The pulse is feeble, and irregular or intermittent; the respiration is embarrassed, a slow and laboured inspiration being followed by a quick expiration and a long pause (Burdon-Sanderson); the skin is cyanotic, and covered with a cold sweat as in the algide stage of cholera; and the patient soon sinks.

The disease frequently terminates favourably, the amendment being indicated by a gradual subsidence of the nervous phenomena, restoration of the mental faculties, and a steady fall in temperature. If the progress towards recovery be uninterrupted, health is re-established in from three to four weeks. Convalescence is, however, often delayed for a long time by relapses, and in such cases recovery is often incomplete.

(2) *Fulminant Epidemic Cerebro-Spinal Meningitis.*—In this variety the patient falls without any premonitory symptom into a state of collapse, drowsiness rapidly supervenes, and is quickly followed by coma. Purpuric spots appear over the surface of the body generally; these soon change from a purple to a black colour, and are often confluent so as to form irregular patches. Death may ensue in less than five hours, or life may be prolonged for two or three days; recovery is not unknown even in this form.

(3) *Purpuric Epidemic Cerebro-Spinal Meningitis.*—In this variety the symptoms which characterise the simple and fulminant varieties are combined in various proportions. In the great majority of cases the disease follows at first the course of the simple variety; but in from one to four days from

the wounded soldiers, after the heat and fatigue of battle or long marching, are exposed to cold and other privations during the night.

Tetanus occurs more frequently in tropical countries than in the temperate or frigid zones, probably owing to the sudden alternations of temperature which are liable to occur in the former, sultry days being often followed by cold nights.

Rose has endeavoured to show that improper treatment of the wounded increases the proportion of tetanus, and there is no doubt that the proportion of cases of tetanus amongst the wounded is much less in the present day than in former times.

The male is more frequently affected with tetanus than the female sex; it is more frequent in youth and middle age than in advanced life, and the robust and muscular are said to be more frequently attacked than the feeble.

§ 923. *Symptoms.*—Premonitory symptoms are generally observed in tetanus consisting of shivering or a distinct rigor, sensation of dragging in the neck, stiffness in certain muscles, difficulty of articulation and deglutition, and yawning. In traumatic cases the wound may become sensitive, and the patient complain of shooting pains radiating from it. These symptoms may occur a few hours or even a few days before the characteristic tonic spasms make their appearance.

The spasms, as a rule, begin in the muscles of the jaw. At first the jaws can be separated, and the movements of chewing and swallowing be accomplished, although with difficulty. Soon, however, the jaws become firmly clenched, constituting the condition called *trismus;* spasm of the œsophagus renders swallowing of even a small quantity of fluid difficult and fatiguing; articulation is indistinct; and the voice is altered, partly from the difficulty of moving the tongue, and partly from implication of the muscles of the larynx in the spasm.

Spasm of the facial muscles gives to the countenance a characteristic expression. The angles of the mouth are drawn outwards, being at the same time depressed or elevated, and the lips are often drawn apart and expose the set teeth, so that the face assumes a sneering expression, named the "*risus sardonicus.*" The nostrils are dilated; the eyes are staring

and motionless; the pupils are generally contracted; the brows are wrinkled; and all the lines of the face become strongly marked, and give to the patient an aged appearance.

The spasm rapidly extends to the muscles of the back of the neck, causing retraction of the head; while the erectores spinæ soon become implicated, and the vertebral column is then arched backwards; the chest is projected forwards and rendered very broad, and the body rests on the back of the head and sacrum, constituting the condition called *opisthotonos.* The epigastrium is sunk, and the abdomen flattened, while the hardness assumed by the abdominal muscles is characteristic. On rare occasions the body is said to be bent forwards, the convexity of the arch being directed backwards, a condition named *emprosthotonos.* In a few cases the body is maintained in a rigid attitude without being curved in any direction, a condition named *orthotonos;* and in some rare cases it is curved laterally—*pleurosthotonos.*

The muscles of the extremities are usually not affected to so great an extent as those of the trunk, neck, face, and jaws.

Fig. 285.

Fig. 285 (From Spence's Surgery).—Taken from the original painting by Sir Charles Bell.

The muscles of the lower extremities are, however, generally implicated to a greater or lesser extent; and during the spasmodic attack extension, as a rule, predominates over flexion, although flexion at individual joints has occasionally been observed

the wounded soldiers, after the heat and fatigue of battle or long marching, are exposed to cold and other privations during the night.

Tetanus occurs more frequently in tropical countries than in the temperate or frigid zones, probably owing to the sudden alternations of temperature which are liable to occur in the former, sultry days being often followed by cold nights.

Rose has endeavoured to show that improper treatment of the wounded increases the proportion of tetanus, and there is no doubt that the proportion of cases of tetanus amongst the wounded is much less in the present day than in former times.

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The muscles of the lower extremities are, however, generally implicated to a greater or lesser extent; and during the spasmodic attack extension, as a rule, predominates over flexion, although flexion at individual joints has occasionally been observed

(Bauer). During the attack the various segments of the lower extremities are extended upon one another and upon the trunk, so that the patient rests on the head and heels; the toes are extended and spread out, and the feet are everted. Even in severe cases the muscles of the upper extremities may remain comparatively unaffected, but if passive motion of the forearm be attempted, increased muscular resistance is readily perceived. When the spasm extends to the muscles of the upper extremities, flexion predominates over extension; and during the paroxysms the arms are drawn close to the chest, the forearm is flexed upon the arm, the hand is flexed at the wrist, and the fist is closed, the palm being directed towards the upper arm (*Fig.* 285).

In some cases the spasm persists continuously from the beginning to the termination of the disease; but, as a rule, the spasmodic rigidity of the muscles occurs in paroxysms with intervals of comparative, but never complete muscular relaxation. Each paroxysm lasts from a few seconds to several minutes, or with slight remissions for hours; while the duration of the free interval varies from ten minutes to hours, but at other times the spasms recur and remit with such frequency that they assume a more or less clonic character. As the disease progresses, the paroxysms of spasm recur with greater frequency, and muscular contraction is sometimes so violent that teeth are broken, long bones, like those of the thigh, fractured, and large muscles, like the psoas and rectus femoris, torn across. The paroxysms recur spontaneously, but they are induced by the most trivial external cause, such as a draught of air, a sudden noise, or an attempt to swallow or to administer an injection. Attempts at swallowing may, indeed, provoke an attack so readily that the disease may bear a certain resemblance to hydrophobia. The spasm occasioned by the attempt may be in the pharynx, gullet, or in the cardiac end of the stomach, as the œsophagus passes through the diaphragm. But wherever it may be situated, all attempts at swallowing are rendered impossible, and fluids introduced are ejected through the nose and mouth, so that the patient can neither be fed in the usual way nor by the stomach pump. During the paroxysm the action of the inspiratory preponderates over that

of the expiratory muscles, so that the act of coughing is rendered impossible, and mucus accumulates in the bronchi. In severe paroxysms the chest becomes fixed; the countenance is livid; the eyes are suffused; the patient foams at the mouth; and is tormented with a feeling of dread and suffocation. Arrest of respiration may sometimes be caused by spasm of the glottis; but, as a rule, it is the result of spasm of the thoracic muscles and diaphragm. In the intervals respiration is only slightly changed in frequency, from twenty to twenty-four in the minute, but it is accompanied by a painful sensation of increased resistance, requiring effort. Motor paralysis is a rare symptom of tetanus. Rose observed paralysis of the muscles of one side of the face in a case in which the primary lesion was in the area of distribution of the facial nerve. General muscular weakness, and paralysis of certain groups of muscles are observed as terminal phenomena; strabismus is, according to Wunderlich, a precursor of death.

The *sensory* disturbances in tetanus are such as are usually produced by intense muscular cramp in the muscles of the calf. Some observers have noticed an increase of the sensibility to pain independently of the spasms, while at other times the acuteness of the senses of touch and temperature may be diminished. Paræsthesiæ, such as numbness and tingling, have occasionally been observed. Pain is sometimes absent during the tetanic seizures; and Blane mentions the case of a patient who only felt a pleasant sensation of tickling during the severest spasms. Pain at the epigastrium, piercing through the back, is, according to some authors, a pathognomonic symptom of tetanus. It is present during both the tetanic paroxysms and the intervals, and depends most probably upon spasm of the diaphragm.

Psychical disturbance is generally absent in tetanus. The mind is almost always clear from the beginning to the end of the disease, although delirium or coma may supervene a short time before death, often due to the remedies used. Sleeplessness is one of the most troublesome symptoms of acute cases of tetanus, and even in subacute cases sleep is only obtained at broken intervals. The spasms cease during sleep and the narcosis of opium or chloroform. The skin, in the paroxysms

and even intervals, is hot and bathed in perspiration, having a peculiar pungent smell, while the surface may be covered by sudamina as in other cases of profuse sweating. In the majority of cases the temperature ranges from 101° F. to 103° F., and may even rise suddenly to 105° F. in cases which recover, although it is not maintained long at this level only in fatal cases. In many cases there is hyperpyrexia immediately before death, the temperature rising to 108° F. or even 110° F., and it may continue to rise for some hours after death.

The pulse may remain normal during the first stage of tetanus, but there is a considerable increase in its frequency during the tetanic seizure, and in the last stage, especially when there is elevation of temperature, it may beat as often as 180 in a minute. Liston observed in a case of amputation during tetanus the vessels so contracted that not a drop of blood had escaped.

The daily quantity of urine passed in tetanus is usually below the average in health; the reaction is strongly acid, the specific gravity high, and there is generally an abundant deposit of urates on cooling. Sugar in the urine in tetanus was first discovered by Demme, and its presence has since been detected by others. Senator found that the excretion of nitrogen was not increased in tetanus as compared with the amount excreted by a person fasting. He also states that the creatinine is not increased. There may be retention of urine, caused probably by spasm of the sphincter, while at other times dribbling may occur during the paroxysm. The bladder is, however, never affected to so great an extent as in acute spinal meningitis. Spasm of the sphincter ani is often present, as is proved by the difficulty of introducing an enema pipe.

The general health of the patient suffers greatly during the course of the disease. The distorted position of the body, the persistent sleeplessness, the difficulty of respiration, and the impossibility of swallowing combine to render the state of the patient extremely distressing. The bowels are constipated, the tongue is generally coated, a tenacious viscid saliva accumulates in the mouth, and the patient may be excessively hungry and tormented with thirst, yet can neither swallow food nor drink.

§ 924. *Course, Duration, and Terminations.*—The time which elapses between the occurrence of an injury and the outbreak of tetanus varies greatly. The average interval is from five to ten days, but the spasms may begin a few hours after the injury, or weeks may intervene. Mr. Ward, of Manchester, has reported a case where the symptoms appeared ten weeks after the injury.

In tetanus neonatorum the disease appears from four to eight days after birth, but it may sometimes be delayed until the fourteenth day.

The intensity of the disease is liable to vary considerably. The symptoms in slight cases may consist only of trismus and some stiffness of the neck; in others they develop rapidly and prove fatal in a few days, or occasionally in a few hours from the commencement.

Death takes place in several ways. It frequently occurs during a paroxysm from asphyxia, caused by rigidity of the respiratory muscles. In other cases the spasms cease, and death follows during mild delirium associated with quick pulse, high temperature, and symptoms of asthenia. At other times the heart suddenly ceases to beat.

In cases of recovery the convulsive attacks become lighter and less frequent, and after a time entirely cease; if sleep return, it is a favourable sign. The rigidity continues for some time after the paroxysms cease and then gradually disappears, though not in definite order. Recovery takes place in from one to eight weeks or even longer, and a certain degree of weakness and stiffness may remain in the muscles for a long time.

§ 925. *Morbid Anatomy.*—Rigor mortis sets in almost immediately after death, probably caused by the strongly acid reaction in the previously active muscles. It has long been suspected that the morbid changes in tetanus are to be found in the central nervous system, and more especially in the spinal cord. In the earlier records, the changes most frequently mentioned are congestion and extravasations of blood into the cord and its membranes, and occasionally softening of the former. Rokitansky was the first to subject the spinal cord to micro-

scopic examination in cases of tetanus. He found an increase of nuclei, destruction of the medullary substance, agglomerations of fat granules, and amyloid bodies. He also observed similar changes in the peripheral nerves. Leyden, however, has since proved that the changes found by Rokitansky are not constant, being in great measure due to imperfect methods of preparation. Lockhart Clarke found great hyperæmia of the cord and its membranes, as well as centres of softening, in the grey and white substance, and similar changes have been described by Dickson, Allbutt, Coats, and other observers.

In the spinal cord of a patient dead of tetanus, I found marked softening in the lumbar region. A section from the middle of the lumbar region is shown in Plate V., fig. 6. A vessel from the anterior fissure, represented in Plate V., fig. 7, was seen to be surrounded by leucocytes, and the whole of both grey and white substances was densely infiltrated with the same, although not usually aggregated in the perivascular spaces and around the vessels as in hydrophobia. Another vessel from the grey substance, cut obliquely, is shown in Plate V., fig. 8, the lymph sheath of which is filled with leucocytes. The most interesting changes were observed in the ganglion cells of the anterior horns. A few cells of normal size were seen in the portion of the median group nearest the anterior fissure, the greater portion of the cells of the antero-lateral group and a few of those of the postero-lateral group were also of normal size; but most of the cells of the median group, and the marginal cells of the other groups, had apparently disappeared, when the section was examined with a low power. The disappearance of these cells was, however, not real; under a high power, they could be seen shrunk in their cavities. Similar changes, although less in extent, were found in other three cases examined by me. Shrinking of some of the ganglion cells of the anterior horns has been recently observed by Dr. Harris and Mr. Doran.

With respect to the medulla oblongata in the cases examined by me, the cells in the internal and anterior portions of the nucleus of the hypoglossal were considerably altered, while those of the body of the nucleus appeared normal. The cells of the nuclei of the spinal accessory, and pneumogastric, appeared

always to suffer; while the longitudinal vessel, which lies in the deepest part of the floor of the fourth ventricle, is, as remarked by Dr. Coats, usually surrounded by red blood corpuscles. The nucleus of the facial, the motor nucleus of the fifth, the nucleus of the abducens, and that of the third and fourth cranial nerves appeared normal. A large number of leucocytes were observed in the olivary bodies, the brachium of the pons, and between the fibres of the seventh nerves.

In the roots of the fifth nerve, proceeding from the cerebellum, large vessels were observed which were distended with red blood corpuscles, and the whole of the surrounding tissue was densely infiltrated with leucocytes. The corpus dentatum of the cerebellum and the white substance subjacent to the cortex, were also densely infiltrated with leucocytes and intersected with distended blood-vessels to an extent which it is impossible to regard as other than the result of disease. The cells of Purkinje were surrounded with leucocytes, but did not themselves present any decided morbid appearances.

In traumatic tetanus the older reports state that marked changes were frequently seen at the seat of the wound. Nerves were crushed and torn, foreign bodies buried in the nerve trunks, and inflammation and thickening were found about the injury. Lepelletier was the first to describe the occurrence of an ascending neuritis in a patient who died from tetanus. Froriep found red spots and swellings of the nerves alternating with parts which remained healthy, these changes extending from the seat of injury to the spinal cord.

Evidences of inflammation have been described by Aronssohn, Dupuy, and Andral in the sympathetic nerves, especially in the cervical and semilunar ganglia.

The voluntary muscles are generally of a pale colour, and ruptures of bundles of fibres with extravasations of blood have been found. Fatty degeneration of the muscles has also been observed.

§ 926. *Pathology.*—The pathology of tetanus is not very clear, but a few landmarks for future researches have been ascertained. Morbid alterations have been found in various portions of the cord and medulla oblongata. Whatever morbid

process these changes may indicate, they are doubtless accompanied by breaking down of the structure of the cord, and during the disintegrative process the molecules of the protoplasm of the cells or of the axis-cylinders, or of both, fall from an unstable to a stable position, the latent energy being rendered active. During this process the irritability of the grey matter and of the nerve fibres is increased, while there is a decrease of their resistance to conduction. In consequence of the increased irritability and diminished resistance, the slightest peripheral irritation will determine muscular spasms by setting free a relatively large amount of energy. Indeed, Romberg regards the *increased reflex irritability* as the chief element in tetanus, but it must be remembered that spasms may be caused by the pathological process going on in the cord independently of peripheral irritation.

An ascending neuritis has been found in some cases in the nerve leading from the wound to the cord; and it is possible that in all cases there is a progressive extension of the diseased process from the external wound towards the centre. But the morbid changes in tetanus are not limited to the peripheral nerves, spinal cord, and medulla oblongata, but probably extend to the cerebellum. I am inclined to believe with Dr. Hughlings-Jackson that discharges of nerve energy from the cortex of the cerebellum are the main cause of the paroxysms of spasm in tetanus, although these discharges may to some extent be determined by the instability of centres in the medulla oblongata and spinal cord.

§ 927. *Diagnosis.*—The diagnosis in well-marked cases presents no difficulty, but the obscure symptoms of the early stage may be overlooked or their importance under-estimated. In the tetanus of strychnia the masticatory muscles are rarely attacked first, and may possibly escape altogether; the symptoms are well marked at the commencement, and reach their full development in a few minutes; opisthotonos is a very early symptom; there are usually intervals of complete intermission; and death occurs commonly in less than three hours, or else recovery is very rapid.

Masticatory spasm induced by decayed teeth, hysteria,

and other causes, and stiffness of the jaws from tonsillitis, parotitis, and disease of the articulations of the jaw, may be mistaken for the first stage of tetanus; but even in slight cases of tetanus the cervical muscles are to some extent affected, and if attention be paid to the symptoms of the former diseases, they cannot well be mistaken for the latter.

Hysterical spasms may sometimes closely simulate tetanus, but in hysteria an interval of variable duration follows the paroxysm, in which the muscles are relaxed, and other symptoms indicative of hysteria are present.

§ 928. *Prognosis.*—The prognosis in tetanus is always grave. Most authors regard idiopathic tetanus as being less dangerous than the traumatic variety.

It may be laid down, as a general rule, that the longer the interval which elapses between the injury and the appearance of tetanus the more likely is the disease to become chronic and to end favourably. Acute cases, in which the spasms supervene soon after the injury, and recur with increasing violence and at decreasing intervals, are almost always fatal, death taking place in a few days, or even hours, from the commencement. The aphorism of Hippocrates, that tetanus ends in recovery if the patient survive the fourth day of the disease, may be accepted as practically true, although there are many exceptions.

Special symptoms are relied upon by some authors in forming a prognosis. The prognosis is said to be grave when attempts to swallow during the first few days induce suffocative attacks. Wunderlich regards the occurrence of strabismus as of fatal augury, and a frequent pulse and high temperature belong to the terminal phenomena of the affection.

§ 929. *Treatment.*—At one time tetanus was regarded as an inflammatory disease, and treated accordingly by so-called anti-phlogistics, blood-letting, and mercurials; but this method of treatment has been abandoned along with the theory upon which it was founded. It is needless to point out here how necessary it is in surgical practice to protect all wounds from unfavourable influences both in the local treatment of the wound and in the general surroundings of the patient.

Different expedients have from time to time been proposed with the view of arresting the peripheral irritation caused by the primary injury. Amputation of the limb and section of the nerves, at one time recommended, are now abandoned for the safer and apparently more successful operation of nerve stretching.

The next aim of treatment is to diminish the irritability of the cord. Chloral hydrate is probably superior to any other remedy for this purpose.

Cannabis Indica has been used, but it is too uncertain in its results, and its physiological action is not yet well ascertained. *Calabar bean* or *physostigmin* has been employed with occasional success. Tobacco or nicotine is highly recommended by Curling, but the depression it produces is sometimes alarming and may be dangerous. Bromide of potassium diminishes reflex irritability, and may be used either alone or in combination with chloral.

Curara, belladonna, and prussic acid have been employed in the treatment of tetanus with the view of controlling the spasms, but the course of the central disease is unaffected by arrest of the spasms.

The cold bath and cold douche may be used when there is a sudden elevation of temperature, and under these circumstances the patient may be placed in a bath of about 90° F., the temperature of which is rapidly reduced to 60° F. by the gradual addition of cold water.

In ordinary cases the *warm bath* is exceedingly soothing to the patient, and for this reason it forms a pleasant auxiliary to other treatment. The *vapour bath* has been used, but is inferior to the warm bath.

The patient should be protected from every source of irritation, such as noises, strong light, changes in temperature, and sudden touching of any part of the body. His strength should be well supported, and when spasm does not permit the patient to swallow liquids, food should be introduced by means of a tube passed through the nose after the patient has previously been brought under the influence of chloroform. Nutritive enemata may also be employed.

Constipation is a troublesome symptom, and may require

attention; but less irritation is caused by the contact of fæces in the bowels to which they are accustomed than by the irritation produced by powerful cathartics.

A few trials have been made with electricity in the treatment of tetanus, but the results have not been encouraging.

(III.) HYDROPHOBIA.

§ 930. *Definition.*—Hydrophobia is a disease caused by the inoculation of a specific animal poison contained in the saliva of animals under its influence, the most characteristic clinical features in man being excitement and spasms induced by attempts to swallow fluids.

§ 931. *Etiology.*—The cause of hydrophobia appears to be in all cases a specific virus contained in the secretions of the mouth of the infected animal, and the disease is communicated to man, and probably to other animals also, only by direct inoculation through a bite. There are no grounds for believing that canine rabies ever arises spontaneously, and it is probably in all instances communicated from one animal to another by means of a bite. The animals which are capable of inoculating man are the dog, wolf, fox, badger, marten, cat, horse, sheep, pig, and goat (Gamgee). Only a small proportion of human beings bitten by rabid animals become affected with hydrophobia, a proportion which has been variously estimated at from 5 to 50 per cent. The number which become subsequently affected with the disease is greater when the exposed parts of the body are bitten. It is probable that the teeth in passing through the clothes are often cleansed, so that the wound escapes inoculation. It is likely that a considerable proportion of those bitten are protected by the cauterisation and other local treatment to which the wound is usually subjected at the time. Some individuals appear to possess a relative or complete immunity from the disease, and it is transmitted irrespectively of age, sex, or constitution.

§ 932. *Symptoms.*—The period of incubation in hydrophobia is longer, and liable to greater variations in its duration, than that of any other specific disease. In the majority of cases the

symptoms manifest themselves from four to eight weeks after the injury, but they may occasionally appear at the end of the first week or not until nine months, and on rare occasions not until several years have elapsed.

The outbreak of the characteristic manifestations of hydrophobia is often preceded for a day or two by premonitory symptoms. The wound inflicted by the bite becomes intensely painful, and the pains generally extend along the nerves towards the trunk. If the wound has not healed, it assumes an unhealthy character; if it has closed, the cicatrix becomes red and irritable. During the period of invasion the patient suffers from chilliness and general malaise, the pulse is rapid, the respirations are quick and sighing, there is thirst and anorexia, and even in this stage the patient may manifest a disinclination to swallow fluids. The face is pale and has an anxious expression; the patient is fidgetty, restless, and peevish; while depression of spirits is so constant and marked a symptom that the period of invasion has been called the *melancholic stage* of the disease.

At the end of a few hours, or at most a day or two, the characteristic features of the disease become fully developed. The patient complains of stiffness about the head and neck, his mouth and fauces are congested, and he suffers from agonising thirst, while every attempt to swallow induces a violent spasm of the pharyngeal and respiratory muscles, which often extends to the muscles of the entire body, and throws the patient into a condition of intense excitement and alarm.

The patient at first may be able to swallow some liquid. In the cases that have come under my observation the patient in the early stage of the disease on being asked to drink took hold of the vessel, looked at it doubtfully for some time, and then, with horror depicted on his face, asked for it to be taken away. Soon afterwards he would summon up sufficient resolution to ask to drink again, and after a period of deliberation and apparent preparation for a supreme effort the vessel was carried rapidly to the mouth, generally with both hands, and part of its contents swallowed with the utmost precipitancy and agitation, the vessel being then almost flung from the hands. This period, however, is of short duration. Every attempt to swallow liquid

is found to induce a severe spasmodic paroxysm, and the abhorrence of water becomes so intense that the spasm may be excited by the sight of it, or even by the noise occasioned by pouring it from one vessel to another. A viscid saliva now accumulates in the mouth, which the patient is constantly attempting to spit out, giving rise to the sound which has often been described as a bark. During the course of the disease convulsive movements may occur in the face, jaw, neck, trunk, and extremities, the spasms being sometimes not unlike tetanic seizures.

The *sensory* disturbances consist of hyperæsthesia of general sensibility and the special senses. The patient often complains of the weight of the bed-clothes, he shuns the light or luminous objects, and is distressed by the slightest noise. Patients also complain of painful sensations in the head, neck, back, and epigastrium, as well as in the neighbourhood of the primary wound.

The *reflex* excitability is greatly increased, and a draught of cold air on the surface of the body, especially on the face, may determine a spasmodic attack of great severity. The slightest impression on the surface of the body occasions, as has been pointed out by Dr. Gowers, a spasm of the costo-superior respiratory muscles and erectores spinæ, very similar to that produced by the cold affusion in healthy individuals. The pulse is small and frequent from the beginning, and becomes thready, irregular, and uncountable towards the fatal termination; and the urine frequently contains albumen and sugar. Out of five cases, in the Manchester Royal Infirmary, and reported by Mr. Southam, the urine was saccharine in three, and highly albuminous in all of them.

Psychical disturbances are always present in hydrophobia, although they are much more prominent in some cases than in others. They vary from a certain emotional excitement that may be mistaken for ordinary hysteria to a state of acute maniacal delirium, the latter being specially liable to occur in children. In the majority of cases the patient has a wild and agitated look; his eyes are bright and restless; his brows wrinkled; and his countenance assumes an expression of extreme horror. The patient is restless, sleepless, often loquacious,

and all his movements are characterised by great precipitation. The intellect may remain more or less clear for a time, but towards the end he begins to wander, and hallucinations or attacks of violent maniacal excitement supervene, during which the patient may injure himself or the attendants. In children the symptoms of acute mania may predominate from the first. In the case of a child four and a half years of age under the care of Mr. Ewart, in St. Mary's Hospital, Manchester, the maniacal symptoms were early manifested. During the maniacal paroxysms, terror, caused probably by hallucinations of sight, appeared to be the predominant symptom, and the child screamed to his mother to save him from the "pussy" that was threatening to attack him (he had been bitten five weeks previously in the face by a rabid cat), and struggled violently to escape from his attendants.

As the disease progresses to a fatal termination all the symptoms are aggravated, and the pulse becomes rapid, irregular, and thready; tenacious mucus accumulates in the mouth and is expelled with difficulty, the voice becomes hoarse, and the spasmodic paroxysms increase in severity and frequency. An attack of convulsions or profound coma may precede the fatal termination, but it is rare to observe a case uninfluenced by narcotics. Death may take place suddenly from asphyxia during a convulsive attack, or from exhaustion. In some cases the spasms gradually diminish and may cease a few hours before death; the patient may even become able to drink, but this comparative calm is deceptive, and, instead of being a sign of recovery, is only the precursor of death.

§ 933. *Course, Duration, Terminations.*—When once the disease is fully established it pursues a rapid course, and terminates probably always fatally in from two to four days after the commencement of the symptoms.

§ 934. *Morbid Anatomy.*—In the older records of post-mortem examinations in cases of death from hydrophobia the chief alterations of the nervous system mentioned are congestion of the brain, medulla oblongata, and spinal cord and of their membranes. In 1869 Meynert made a microscopic examina-

tion of portions of the brain and spinal cord of two patients who had died of hydrophobia; but the changes observed by him, in addition to congestion of the spinal cord and brain, were somewhat indefinite. Soon afterwards Dr. Allbutt made some important observations. "In the cerebral convolutions the mesocephalon, the pons, medulla, and spine, the vessels," he says, "were seen in various degrees of distension, and in many places the walls were obviously thickened, and here and there in them were patches of incipient nuclear proliferation." These observations were confirmed by Hammond, who also found changes in the ganglion cells of the nuclei of origin of the pneumogastric and hypoglossal nerves, as well as in those of "the first and second" layers of the cortex of the cerebrum. The nuclear proliferation described by Dr. Allbutt as occurring in the walls of the distended vessels consists, as was subsequently pointed out by Benedikt, of migrated white blood corpuscles.

In the brains of dogs affected with rabies, Benedikt found the white blood corpuscles aggregated around the vessels to such an extent as to form what he termed a miliary abscess. He found similar appearances in the brain of a human subject who had died of hydrophobia. The spinal cord and medulla oblongata were not examined in these cases. These observations have been confirmed and extended by Coats and Gowers, who found accumulations of leucocytes around the smaller vessels of the medulla oblongata, spinal cord, basal ganglia, and cortex of the brain. Small extravasations of blood were occasionally observed in the neighbourhood of the distended vessels, while Coats found migrations of leucocytes in the salivary glands, mucous glands of the larynx, and kidneys. Marochetti observed pustules on the frenum linguæ during the first few days of the period of incubation. In the case of a dog that died of rabies I found miliary abscesses extensively distributed through the spinal cord, medulla oblongata, basal ganglia, and cortex of the brain. In addition to the aggregation of leucocytes around the vessels, Gowers describes intravascular changes, consisting of the presence of clots in some of the vessels, which he thinks must have formed during life.

The following description is derived from my own micro-

DESCRIPTION OF PLATE V.

(From Drawings by Dr. A. H. Young.)

Fig. 1.—Section of medulla oblongata from a case of hydrophobia, showing extensive infiltration of the tissues with leucocytes; all the cut vessels being surrounded by them. The nucleus of the hypoglossal nerve is infiltrated with leucocytes; but its cells are apparently healthy. Almost all the cells of the nucleus of the pneumogastric nerve have disappeared, and the nucleus of the spinal accessory nerve was similarly affected lower down the medulla.

Fig. 2.—Portion of the nucleus of the hypoglossal nerve from same section as Fig. 1, under a higher magnifying power. The ganglion cells appear healthy, although they are surrounded by leucocytes. The cut ends of the vessels may be seen surrounded by numerous leucocytes.

Fig. 3.—Portion of grey matter of olivary body from same section as Fig. 1, magnified, showing infiltration with leucocytes.

Fig. 4.—Section of the anterior horn of the grey substance of the cervical enlargement of the spinal cord in a case of hydrophobia. *m*, Median; *al*, Antero-lateral; *pl*, Postero-lateral; and *c*, Central group of ganglion cells. Shows infiltration of the tissues with leucocytes, and the cut vessels surrounded by them. The ganglion cells have apparently disappeared from the area which lies between the median and the antero-lateral groups, and only two or three of the cells of the central group are seen; while some cells have also apparently disappeared from the margins of the antero-lateral and postero-lateral groups. The central group was also infiltrated with red blood corpuscles.

Fig. 5.—Portion of the area from which the cells had apparently disappeared in Fig. 4, magnified. Shows that the cells are still present, although they are much shrunk, and some of their processes destroyed. The walls of the cavity surrounding them are infiltrated with leucocytes or covered by nuclei.

Fig. 6.—Tetanus. Section of the anterior horn of grey substance from the middle of the lumbar enlargement. Showing infiltration of the tissue with leucocytes, and apparent disappearance of the ganglion cells from the area lying between the few remaining cells of the median group and those of the antero-lateral group; also apparent disappearance of a considerable portion of the cells of the postero-lateral group.

Fig. 7.—Vessel from the anterior median fissure from the same section as Fig. 6, surrounded by numerous leucocytes.

Fig. 8.—Vessel cut obliquely from the grey substance from the same section a Fig. 6, showing its lymph sheath surrounded by leucocytes.

Fig. 1.

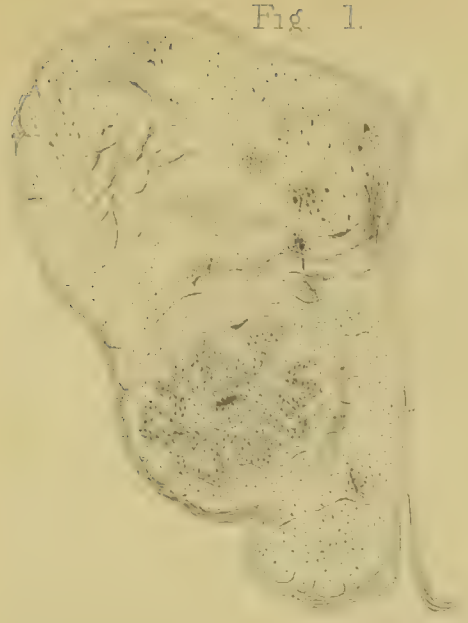


Fig. 2.

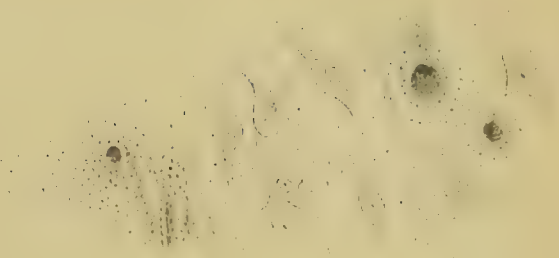


Fig. 3

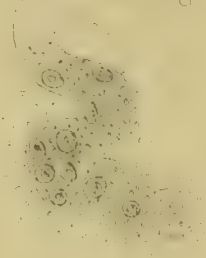


Fig 4

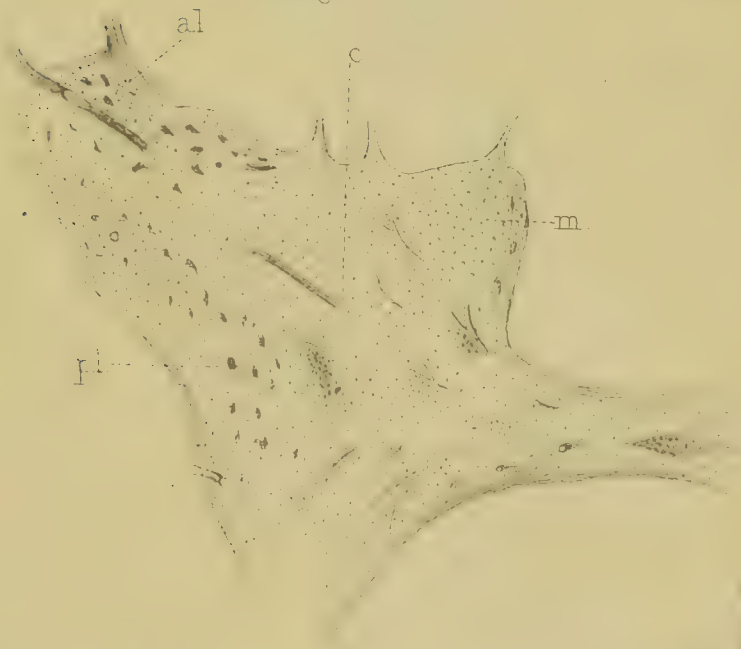


Fig 5

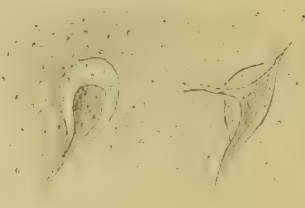


Fig 6

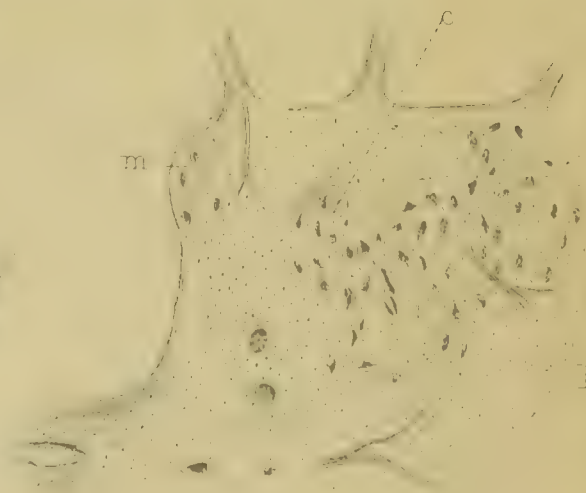
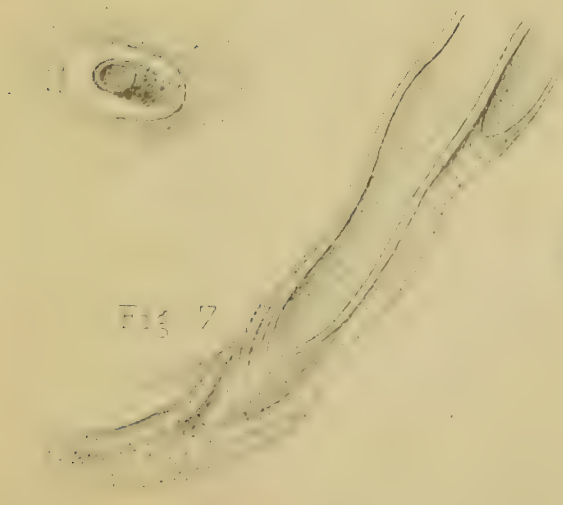


Fig 7



scopical examination of the nervous system from six cases of hydrophobia:—

The changes observed in the medulla oblongata in hydrophobia corresponded closely to the careful drawings and accurate description of Dr. Gowers; except, perhaps, with respect to his description of the clot within the blood-vessels. The distribution of these vascular and perivascular changes in the medulla oblongata was, in one of my cases, almost coextensive with the distribution of the vessels. The grey matter on the floor of the fourth ventricle was most affected, probably because it is the most vascular; but similar changes were found in the *formatio reticularis*, olivary bodies, restiform bodies, and to a less extent in the anterior pyramids. The condition of the medulla in this case is shown in Plate V., fig. 1. It will be observed that, although the nucleus of the hypoglossal is infiltrated with leucocytes, its own cells are not much affected, while scarcely a trace is left of those belonging to the spinal accessory and pneumogastric nuclei. The same condition is shown under a higher magnifying power in fig. 2, in which the cells of the nucleus of the hypoglossal appear healthy, although the tissue is densely infiltrated with leucocytes. Infiltration of the grey matter of the olivary bodies with leucocytes is shown in fig. 3. Similar vascular and perivascular changes were observed, though to a less extent, throughout the whole extent of the pons, in the grey substance surrounding the aqueduct of Sylvius, and in the corpora quadrigemina, as has already been described by Dr. Coats. The cortex of the brain and subjacent white substance were infiltrated with leucocytes, and the pyramidal cells of the fourth layer of the cortex were often partially filled with small yellow granules. The cortex of the cerebellum and subjacent white substances were also much infiltrated with leucocytes, the changes here being almost, if not quite, as marked in extent as in the brain, and the cells of Purkinje were very granular. It would appear, therefore, that hydrophobia is a disease of a very diffused character.

The changes found in the spinal cord in hydrophobia deserve special mention. They are not always well marked, and in three of my cases might very readily be overlooked, while in the other three, marked alterations were observed in the upper dorsal region and in the cervical enlargement. One of the anterior horns, from a section of the cervical enlargement, is represented by Dr. Young in fig. 4. The whole of the grey substance is infiltrated with leucocytes, and the vessels on each side of the central canal are surrounded by them. The branches of the anterior external and anterior lateral arteries distributed to the grey substance are surrounded by leucocytes. Three distinct miliary abscesses were observed in the external margin of the antero-lateral group of cells in the cord taken from the child, who died of hydrophobia, under the care of Mr. Ewart.

The most interesting changes probably have occurred in the ganglion cells themselves. Two or three cells, of normal size, may be observed in

the part of the median group which is nearest to the anterior fissure, and full-sized cells may also be observed in the central portion of the antero-lateral and postero-lateral groups. When examined under a low power, all the cells seem to have disappeared from the territory which lies between the few remaining cells of the median group (fig. 4, m) and the antero-lateral group (fig. 4, a l), while the cells of the central group (fig. 4, c) have also, with the exception of two or three, disappeared, and a considerable number are wanting also along the margins of the antero-lateral and postero-lateral groups. When the areas from which the cells have apparently disappeared are examined with a higher power, it is seen that remnants of the latter are still present. The cells are surrounded by a partially empty cavity, the walls of which are covered either by leucocytes or nuclei, while the cells themselves are shrunk to a very variable extent (fig. 5). The cell membrane appears to be shrunk around the nucleus and nucleolus, along with a small quantity of granular contents; but one or more processes can almost always be detected; the full-sized cells often contain yellow pigment granules, and sometimes appear to be increased instead of diminished in size. A number of red blood corpuscles are infiltrated into the central group of cells (fig. 4, c) and the cells themselves are much altered and diminished in size. The vesicular column of Clarke, especially in the upper dorsal and lower cervical regions, is usually much infiltrated with leucocytes, and its own cells are often deformed and shrunk. At the junction of the dorsal and lumbar regions, the cells of this group are frequently found healthy.

§ 935. *Morbid Physiology.*—The mode of origin of hydrophobia shows that the disease is due to a specific animal poison, and the anatomical lesions found after death appear to indicate that the toxic agent, whatever its nature, is conveyed in the blood to the nervous tissues. There is nothing specific in the miliary abscesses found in hydrophobia as they are observed in other diseases. The poison, as was first suggested by Dr. Allbutt, appears to act first on the nuclei of the medulla oblongata, and more especially on the respiratory centres; then on the grey matter of the spinal cord; and, lastly, on the cortex of the cerebrum. That the poison of hydrophobia should act in this manner on the nervous tissues is not more inscrutable than that the poison of scarlet fever should produce an eruption on the skin, which first appears on the chest, then extends to the forearms and lower part of the abdomen, and finally becomes general.

While examining the spinal cord in cases of hydrophobia I was much struck by the fact that the cells of the median

group and the marginal cells of the other groups of ganglion cells of the anterior horns were invariably altered, while the ganglion cells of the centres of the groups were apparently unaffected. It was in my power to take refuge in the supposition of a special affinity between the poison and these cells, but I was met by a similar vulnerability of the same cells in tetanus, in all central inflammations of the spinal cord, whether acute or chronic, and even in chorea. It was in this state of doubt that I came to recognise the significance of two other facts with regard to the cells in question. The ganglion cells which are most vulnerable in all affections of the grey substance of the spinal cord are, speaking broadly, smaller than those which are most resisting, and the former are developed at a much later period than the latter. The small size of the cells enables them to present a large surface to their environment in comparison with their bulk, and consequently they must absorb a proportionably larger quantity of nourishment. The cell-membranes of the last are also likely to be thinner than those of the first developed cells, and this will increase still further the capacity of the former for the absorption of nourishment. The ganglion cells which absorb a large quantity of nourishment in a relatively short time must necessarily suffer at an earlier period in states of active hyperæmia than the cells which absorb a less quantity, while they will be equally the first to suffer in conditions of anæmia, inasmuch as the want of nourishment must be first felt in those cells which are undergoing the most active changes. There are no grounds, therefore, for believing that the accessory manifest a greater affinity than the fundamental cells for the virus of hydrophobia, but the conditions under which nutrition is normally carried on in both are such that the former suffer in this disease to a greater degree than the latter.

§ 936. *Diagnosis.*—The history of a bite from a rabid animal is generally sufficient to direct attention to the true nature of the disease. But inasmuch as the animal is generally killed soon after inflicting the injury, we often cannot be sure that it was the subject of rabies. On the other hand, those who have been bitten by dogs or other animals are often haunted by the

dread of hydrophobia, and hysterical symptoms not unlike those of the actual disease may become developed in them. In cases of the kind, besides globus hystericus, other symptoms are generally present which render their nature apparent, and, inasmuch as hydrophobia is almost always fatal within four days from the commencement, its diagnosis from hysteria cannot long remain matter for doubt. It must also be remembered that tetanus may occur after the bite of an animal just as after other injuries, and it is not impossible that cases of reported recovery from hydrophobia have been of the nature of tetanus, In tetanus the patient is usually calm and the mental faculties are clear to the last; while there is complete absence of the emotional excitement, horror, and delirium observed in hydrophobia.

§ 937. *Treatment.*—Every effort should be made to prevent the development of the disease. The tissues surrounding the wound should be at once excised with the knife, or, if this be impossible, destroyed by the actual cautery or powerful caustics, as potassa fusa or nitric acid. Youatt placed the greatest reliance on the cauterisation of the wound with the solid nitrate of silver.

When the disease is established, every effort should be directed to soothe the sufferings of the patient. In the cases coming under my observation, subcutaneous injections of morphia and chloral appeared to be productive of most relief, inasmuch as several hours of quiet sleep were procured for the unfortunate patient. Hot-air and vapour baths have been recommended, and the latter is very soothing to the patient; but there are no grounds for believing that it can eliminate the virus.

CHAPTER IV.

HYSTERIA.

HYSTERIA is a functional disease of the nervous system characterised by paroxysms of convulsions with apparent loss of consciousness, along with various sensory, motor, vaso-motor, and psychical disturbances, which may be combined in such manifold ways that the grouping of the symptoms may simulate any one of the numerous organic diseases to which the nervous system is liable.

§ 938. *Etiology.*—Hereditary predisposition exerts a powerful influence in the production of hysteria. The transmission of the disease is sometimes direct, the mother transmitting it to the daughter, and at other times indirect, the patient inheriting a neurotic constitution which manifests itself in one member of a family as hysteria, and in the others as neuralgia, epilepsy chorea, or insanity.

Hysteria occurs with preponderating frequency, although by no means exclusively in the female sex. Briquet states that one out of four of all females are affected with decided hysteria, and that one-half present an undue impressionability which differs very little from it, a proportion much too high for this country. Out of 1,000 cases collected by Briquet, one male was affected with hysteria in proportion to twenty females.

The disease usually begins in females about the age of puberty, the first symptoms being manifested in more than half the cases collected by Briquet between twelve and twenty, and in a third of them between fifteen and twenty years of age. The establishment of menstruation does not appear to favour

the development of hysteria, although the affection is frequently associated with derangement of the function.

Hysteria occurs in children of both sexes under ten years of age. Briquet states that the first symptoms appeared in children under ten years in about one-eighth of his cases, a proportion which is much too high so far as this country is concerned. Boys from ten to fourteen years of age are not unfrequently the subjects of hysteria; cases of the kind have been recorded by Wilks and Roberts, and I have myself seen several examples.

All causes which lower the nutrition of the nervous system, as hæmorrhages, insufficient nourishment, impaired digestion, and anæmia, predispose to hysteria.

Hysteria affects the females of the higher classes of society more frequently than those of the working classes. Young ladies often lead an utterly aimless existence, periods of idleness alternating with the unhealthy excitement afforded by balls and theatres. Under such circumstances too little exercise is apt to be taken, the bowels become constipated, digestion is impaired, and anæmia results; all of these are conditions tending to diminish the nutrition of the nervous system. The mental energies not being expended on any healthy occupation are apt to be directed inwards to the contemplation of the patient's own thoughts and feelings; and under these circumstances the emotions connected with the gratification of the sexual impulses are apt to occupy a predominant place in the thoughts. This mental condition, while strengthening the lower emotions, weakens the power of the will, and induces that excessive irritability of the nervous system which underlies hysteria.

The depressing passions, as fear, anxiety, jealousy, and remorse, frequently induce hysteria, while exhaustion from overwork when combined with anxiety, as in the case of those who have to nurse sick relatives, is one of the most fruitful causes of the disease.

All uterine derangements, whether structural or functional, are apt to be attended by hysteria, and the symptoms are liable to become aggravated during the menstrual periods in those who are already hysterical. The name hysteria was, indeed, given to the disease under the belief that it was always caused

by disease of the uterus and its appendages; but this exclusive view of the origin of the disease is now no longer maintained by anyone. Hysteria may be acquired by those who are predisposed to it by the imitation of others. Young susceptible girls are not unfrequently seized with hysteria after being witnesses of the disease in another. An actual epidemic of hysteria may occur in public institutions, like hospitals, factories, and girls' schools, and the convulsive epidemics of former ages appeared to spread in this manner.

§ 939. *Symptoms.*—The symptoms of hysteria may be divided for the purpose of description into: (1), those which characterise the intervals between the attacks; and (2), those which constitute the hysterical paroxysm.

(1) Interparoxysmal Symptoms.

(*a*) *Sensory Disturbances.*—*Hyperæsthesia* in some form or another is seldom absent during the course of hysteria. The acuteness of the perceptive faculties is sometimes increased, so that hysterical patients may see, hear, smell, or taste objects imperceptible to the healthy; and their sense of touch may also be preternaturally acute. But disorder of the common or emotional sensations is much more usually met with than disorder of the special or intellectual. Sensations which to others are indifferent or pleasant produce in the hysterical the highest degree of discomfort; and, conversely, sensations disagreeable to others may be enjoyed by them. Hysterical patients often complain urgently of neuralgic pains in various parts of the body in the absence of any recognisable cause.

The senses of touch and temperature are sometimes increased in acuteness. Some hysterical patients recognise persons and objects by the sense of touch with an accuracy unknown in the healthy, while they often complain of pulsation in different parts of the body, which can only be explained on the assumption of an increased sensibility.

Cutaneous or deep-seated hyperalgesia is rarely absent in hysteria, and it may or may not be accompanied by spontaneous pains in the affected parts. Cutaneous hyperalgesia is some-

times widely diffused, so that the slightest movement of the patient causes discomfort. In most cases, however, the hyperalgesia is limited to particular portions of the surface or to one extremity, a circumscribed portion of the trunk, or half the body, while irregularly circumscribed anæsthetic patches are sometimes found in the midst of a hyperæsthetic region.

The neuralgiform pains which occur in various parts of the body are described as of extreme severity. Neuralgia of the mammary gland is sometimes complained of, especially at the menstrual periods; while a fixed and severe pain in the left infra-mammary region—probably an intercostal neuralgia—is an all but constant symptom of hysteria. The skin over the mammæ may become so sensitive that the slightest touch or the contact of the dress may be unbearable, and great sensitiveness to pain is often observed in the neighbourhood of the ensiform cartilage; while pains of a dull, heavy character, accompanied by a feeling of oppression and anxiety, are sometimes experienced at different parts of the sternum.

Severe pain diffused over the whole surface of the abdomen is a not uncommon symptom of hysteria. The abdominal pain is usually associated with tympanites, and the sensitiveness to touch is so great that the patient cannot bear the contact of the bed-clothes; while deep and continuous pressure, on the other hand, may cause little or no discomfort, especially if the attention of the patient be diverted. Hysterical patients suffer greatly from cardialgia, and when it is associated, as is frequently the case, with persistent vomiting the symptoms may be mistaken for those of perforating ulcer of the stomach. Many hysterical patients suffer from a great craving for food, leading them to eat large quantities; this condition is probably caused by a hyperæsthetic condition of the mucous membrane of the stomach. Ovarian hyperæsthesia (§ 345) is sometimes observed, and, curiously, this condition is usually associated not with hyperæsthesia, but with anæsthesia of the same side. Hysterical patients often suffer, especially at the menstrual periods, from irritation and burning at the labia and at the vaginal orifice, these sensations being not unfrequently associated with increased sexual desire. The bladder and urethra are also excessively sensitive and painful in such cases.

Hysteralgia is not an unfrequent symptom of hysteria, independently of any local disease; coccygodynia without local disease is exceptional.

Hysterical patients suffer from various more or less painful affections of the back. Cutaneous hyperæsthesia is sometimes present, its favourite sites being circumscribed portions of skin over and between the scapulæ. Tenderness of the vertebræ and surrounding structures is, however, a more frequent symptom, the sensitiveness being sometimes limited to the spinous processes and at other times distributed laterally in the muscles of the vertebral column; this spinal tenderness is frequently accompanied by genuine neuralgia. The affection already described under the name of *spinal irritation* consists of spinal tenderness in association with other aggravated symptoms of hysteria. Increased sensitiveness of the muscular afferent nerves probably cause the restlessness frequently experienced by hysterical patients.

The pains and hyperæsthesia frequently occurring in and around the joints are deserving of particular attention, inasmuch as they are often mistaken for chronic articular disease. Sir Benjamin Brodie was the first to direct attention to the frequency of these affections, and he asserted that four-fifths at least of the joint diseases met with in women of the higher classes of society are purely hysterical. The hip and knee joints are most frequently affected; but the ankles, wrists, and even finger joints may be attacked. In the hysterical affection pressure upon the joint produces pain, but little or no pain is caused by forcible apposition of the articular surfaces, especially if the attention of the patient be otherwise engaged. The disease may continue for years, and in chronic cases the joint may become slightly swollen from œdema of the surrounding soft parts.

The senses of *smell* and *taste* are frequently increased in acuteness, the patients recognising tastes and odours which are inappreciable to most people. At other times there is a perversion of these senses, and the patient manifests a preference for certain tastes and odours which are disagreeable or indifferent to others. In obedience to this morbid craving hysterical patients sometimes devour chalk, cinders, or even

disgusting substances, and exhibit a liking for odours like those of assafœtida and valerian. Hallucinations also of taste and smell may be experienced in hysteria.

The sense of *hearing* often becomes extremely sensitive, but intolerance of sound is more frequently met with than true auditory hyperæsthesia. Subjective sensations, such as ringing, blowing, roaring, are sometimes heard, or there may be true auditory hallucinations.

The sense of *sight* is sometimes increased in acuteness, but intolerance of light is more frequent. Under the latter circumstances the patient shuns the light, and the power of detecting objects in the dark is increased. At times the hyperæsthesia only exists with respect to a particular colour, most commonly red. Sparks and flashes of light are sometimes complained of, while at other times there are hallucinations of sight, the objects seen being often productive of disgust and horror.

Anæsthesia.—Diminution or complete loss of sensibility is a very frequent symptom of hysteria. It may exist over a large portion of the surface of the body, and may implicate the muscles and deeper tissues as well as the nerves of special sense. Anæsthesia, in some form or another, occurs frequently after a hysterical attack, and the more severe the attack the more likely is anæsthesia to ensue; as a rule, it diminishes in the interval. Sometimes, however, an extensive and persistent anæsthesia may disappear after a fresh attack, and the sensibility of the previously affected part may become normal or exaggerated, or the anæsthesia become transferred to another portion of the body.

In the majority of cases sensibility to pain is alone altered, while the other forms of cutaneous sensibility remain normal. In some cases tactile sensibility is lost, while variations of temperature are correctly appreciated; in other cases every form of cutaneous sensibility, as well as that of the muscles, bones, and joints, is abolished.

Anæsthesia of the mucous membranes is not of uncommon occurrence either alone or in connection with cutaneous anæsthesia. It is associated with diminution or loss of reflex contractility, neither reflex spasm, lachrymation, nor reddening being provoked by the contact of foreign bodies with the affected

conjunctiva, nor sneezing by the inhalation of irritating substances when the nasal mucous membrane is implicated. Sensation may be abolished in the mucous membranes of the pharynx, larynx, and respiratory tract generally, and the occasional retention of urine and fæces in hysterical patients is probably caused by anæsthesia of the mucous membranes of the bladder and rectum, inasmuch as in such cases the bladder or rectum may sometimes be found enormously distended without having caused more than a trifling amount of discomfort. The mucous membrane of the genital organs and of the urinary passages is sometimes found insensible. The mucous membrane of the vulva and vagina may be completely anæsthetic. This condition is found in highly hysterical married women, and in them there is an entire absence of sexual desire or pleasure.

The special senses are not unfrequently affected by anæsthesia in hysteria, more especially after severe hysterical seizure. The senses of taste and smell may be lost, the loss being sometimes unilateral, at other times bilateral. Deafness of nervous origin is also occasionally observed, and it may be limited to one ear or affect both.

There may be amblyopia or complete amaurosis of one or both eyes, unilateral amblyopia being the most frequent condition. Hysterical amblyopia consists of diminution of the acuteness of vision, restriction of the field of vision and achromatopsia, while sometimes a condition simulating hemiopia may be present. An ophthalmoscopic examination does not reveal any changes in the optic discs.

The distribution of the different forms of hysterical anæsthesia is very variable. Cutaneous anæsthesia is often limited to certain circumscribed portions of the surface of the trunk and extremities; it may be observed in the region of distribution of one or more nerve trunks, be limited to one or more extremities, or be accurately confined to half the body.

In hysterical *hemianæsthesia* the loss of feeling on the anæsthetic side frequently affects the superficial parts only; but at other times the muscles, bones, and articulations are implicated.

Hemianalgesia is the most common form of the incomplete variety, the insensibility to pain being sometimes associated

with thermo-anæsthesia. In complete hemianæsthesia not only the skin, but the muscles, bones, articulations, and the special senses, and even the accessible mucous membranes on the same side of the body are implicated. Taste is abolished on the corresponding half of the tongue, the sense of smell is less acute in the corresponding nostril, and partial deafness and amblyopia exist on the same side. The anæsthesia, however, does not appear to extend to the viscera, and complete hemianæsthesia is usually associated with ovarian hyperæsthesia.

(*b*) *Motor Disturbances.—Spasms*, either tonic or clonic, may occur in hysteria in every muscle or group of muscles of the head, trunk, and extremities. Every one of the spasms already described as occurring in the area of distribution of one or several of the peripheral motor nerves may appear in hysteria in the form of a more or less persistent or of recurring contractions. It is unnecessary to describe them in detail. The facial muscles are incessantly active in many hysterical patients, so that the countenance has a restless and unsettled expression, constituting one of the main characteristics by means of which the practised physician is enabled to diagnosticate the disease.

Spasmodic closure of the glottis may produce alarming dyspnœa, and the patients are liable to attacks of convulsive laughter and weeping, which often arise apparently in the absence of any emotional disturbance. During hysterical attacks loud screams are commonly emitted, and in that form of hysteria named chorea major the patients often imitate the cries of animals by mewing, barking, or howling. Hysterical patients often suffer from a temporary acceleration and exaggeration of breathing without there being any feeling of embarrassed respiration, and at other times they suffer from temporary spasmodic pauses in the respiratory rhythm. Hiccough and yawning are frequent and sometimes very distressing symptoms.

The pharyngeal muscles are sometimes spasmodically contracted, so that swallowing becomes difficult or impossible. Spasm of the tongue is not unfrequently associated with that of the pharyngeal muscles. At every attempt to move the tongue it becomes distorted in various directions, so that articulation and swallowing become greatly impeded. The sensation of choking in the throat, named *globus hystericus*

(§ 61), is supposed by some to be caused by a spasm of the œsophagus. The sensation of a foreign body in the throat is sometimes so real that the patient, after making strenuous efforts to remove it by swallowing, puts her fingers into her throat in order to induce vomiting, by which she hopes to eject it. Actual spasm of the œsophagus may sometimes be so persistent as to resemble organic stricture.

The stomach is liable to undergo spasmodic contractions, giving rise to persistent and distressing vomiting. The patient vomits almost immediately after food is taken, so that the latter is usually ejected in an undigested condition. Some of the food is, however, probably retained, as the nutrition of the patient rarely suffers in proportion to the apparent violence and persistency of the vomiting.

Irregular peristaltic movements occur in various parts of the intestines, and these may be so energetic that they can be felt through the abdominal wall. The rolling of the intestines may convince the patient that a movable body is present in the abdomen. Spasm of certain portions of the intestines may be so persistent as to cause temporary stricture, and the bowels above the constricted portion become greatly distended with gas, giving rise to what has been called a "phantom tumour;" or a real obstruction of the bowels may sometimes be caused by accumulation of fæces behind the constricted portion. Eructations, borborygmi, and griping pains may also be caused by irregular peristaltic movements of various portions of the digestive canal.

Spasmodic retention of urine, generally combined with increased inclination to micturate, occurs in many hysterical patients; and this condition is sometimes, but not always, associated with a painful condition of the genitals.

Vaginismus, caused by spasm of the constrictor vaginæ, sometimes renders coitus difficult or impossible; it is generally associated with hyperæsthesia of the vaginal orifice, the spasm being induced by reflex action.

Paralyses.—Partial or complete loss of muscular power is a frequent symptom of hysteria. Briquet found that out of 430 cases of hysteria 120 suffered from paresis or paralysis; and Landouzy, out of 370 cases, found 40 similarly affected.

The loss of motor power may begin with mere weakness and heaviness of the limb or limbs, which gradually increase to complete paralysis. At other times the commencement is sudden, the paralysis becoming fully developed after a hysterical attack.

The distribution of the paralysis is very variable. It may assume the hemiplegic form, and in these cases the paralysis often supervenes after an attack of hysterical convulsions, attended with partial loss of consciousness, which may last for several days, so that the hemiplegia resembles the result of organic lesion of the brain. In hysterical hemiplegia there is no distortion of the face, nor deviation of the tongue on protrusion, phenomena which are almost always present at first in hemiplegia, due to cerebral lesion. In hysterical hemiplegia the paralysis is seldom complete; in the majority of cases the leg is more profoundly affected than the arm, and the loss of motor power is liable to considerable variations in intensity, especially under the influence of emotional excitement. Hysterical hemiplegia is, moreover, generally associated with the *hemianæsthesia* already described, as well as with ovarian hyperæsthesia, retention of urine, tympanites, and other symptoms of aggravated hysteria. Another feature worthy of attention is that the convulsive attack which preceded the paralysis is always produced by a profound moral shock.

One extremity only is affected, or the upper extremity on one side and the lower extremity on the other, and total paralysis of all the extremities is not unknown. The paralysis may be limited to one or more motor nerves, or to one of the branches of a nerve. Hysterical paralysis of the ocular muscles is rare, but paralysis of one or both the levator palpebræ superioris muscles is not unfrequent, and the well-known hysterical expression is probably partly due to the drooping of the upper eyelids, caused by imperfect contraction of these muscles.

The excitability of the paralysed muscles to both the faradic and galvanic currents remains unchanged even when the paralysis has existed for years, a circumstance of great importance in establishing a correct diagnosis. After long disuse the muscles may indeed undergo a certain amount of

atrophy, in which case there may be a slight diminution of electric excitability, but the "reaction of degeneration" is never established.

Anæsthesia is frequently associated with paralysis in hysterical patients, although each of these conditions may be present without the other. When both conditions are combined the anæsthesia is generally not confined to the skin, but extends to the muscles, and then "electro-muscular sensibility" is diminished or abolished. Duchenne regarded this condition as a very valuable sign of hysterical paralysis, but it must be remembered that muscular anæsthesia is sometimes absent in hysterical, and occasionally present in paralysis of apoplectic origin. Hysterical paralysis is always accompanied by other manifestations of the disease, such as spasm, hyperæsthesia, and particularly by the characteristic psychical condition.

In doubtful cases a careful observation of the course and progress of the disease will aid in clearing up the diagnosis. Hysterical paralysis is generally variable in its duration, continuing for a few hours, days, or weeks, and then completely disappearing, perhaps to return after subsequent attacks of hysteria. The mode of extension of the paralysis is sometimes characteristic. It may be pronounced at first on one-half of the body, then quickly disappear from that side and present itself on the opposite side, or it may be crossed. In some cases the paralysis continues for years unchanged in extent; in these cases hysteria may be difficult to distinguish from hemiplegia caused by circumscribed lesion of the brain, or from cerebro-spinal sclerosis and spinal paraplegia.

Contracture not unfrequently becomes developed in the paralysed extremities. In some cases the contracture appears simultaneously with the paralysis, while in other cases the paralysis continues for some time and then contracture supervenes gradually or suddenly after a fresh attack. In the upper extremities there is spasmodic flexion of the forearm, hand, and fingers; the muscles are in a state of considerable rigidity, so that it is impossible to obtain complete extension, or to increase the flexion.

The lower extremity is strongly extended upon the pelvis, and the leg upon the thigh; the foot generally assumes the position

of talipes equino-varus; and the knees are drawn inwards by contraction of the adductors of the thighs.

Contractures may, like paralysis, last for years, and then suddenly cease, generally under the effect of strong emotional disturbance. In other cases, when the contracture has continued for many years, atrophy of the affected muscles may result, accompanied by the "reaction of degeneration." Under these circumstances the contracture cannot be overcome under the deepest chloroform narcosis.

In hysterical contracture with paralysis tremor is sometimes observed, especially on attempting any movement, just as occurs in the paralysis with contracture arising from sclerosis of the lateral columns of the cord.

Charcot relates the case of a woman, the subject of hysteria, who had suffered from contracture in all the extremities for ten years. The degree of contracture had undergone many fluctuations for several years, but ultimately became permanently established, and a post-mortem examination revealed sclerosis of both lateral columns of the cord. But, as Charcot remarks, the presence of tremors in cases of hysterical contracture, which terminate abruptly in recovery, shows that this symptom cannot always be attributed to a permanent lesion of the lateral columns.

Tremor may also occur in hysteria independently of paralysis with contracture, and it then closely resembles the tremors of paralysis agitans or of cerebro-spinal multiple sclerosis. This tremor may appear in the muscles of the head, tongue, face, or hands, and is called forth and aggravated by emotional excitement. It persists during repose, if the patient be conscious of being observed.

Hysterical aphonia from paralysis of the vocal cords is a very frequent symptom of hysteria. Aphonia may appear suddenly after some mental excitement, and disappear with equal celerity. Difficulty of moving the tongue is sometimes associated with the laryngeal paralysis, and the patients no longer whisper, but resort to pantomime in order to make themselves intelligible. Laryngoscopic examination shows paralysis of the adductors of the glottis sometimes on one, sometimes on both sides.

Paralysis of the muscles of the pharynx and the œsophagus is

not an uncommon symptom of hysteria, and swallowing may consequently be rendered difficult or impossible. In such a case the œsophageal tube passes into the stomach without any obstruction. Retention of urine is common, and often paralytic in origin.

Paralysis of the muscular coat of the stomach is partly cause, partly effect of the general tympanites which is so frequently met with in hysterical patients. Tympanites may come on suddenly, in consequence of mental agitation or at the close of a hysterical attack, and sometimes reaches such a degree that the patients may be kept afloat in a bath by means of the gaseous distention. The obstinate constipation which is so frequent in hysteria is probably due to paralysis of the muscular coat of the bowels.

(*c*) *Vaso-motor and Secretory Disturbances.*—In the intervals between the attacks of hysteria the action of the heart and the pulse may be normal, unless indeed some general disease, like chlorosis, be present. Hysterical patients are, however, liable to suffer from paroxysms of palpitation. During these attacks the pulse is at first frequent, small, and hard; the skin is pale and cold; there is a feeling of fulness and oppression in the chest; and there may be a degree of mental confusion. After a time the cutaneous vessels relax and the surface is reddened and covered with perspiration; the pulse then becomes slow, full, and compressible. Hysterical patients are liable to fainting fits, caused doubtless by sudden anæmia of the brain. The cerebral anæmia may in its turn be produced either by vaso-motor contraction of the intracranial arterioles, or by sudden dilatation of the arteries of the body, especially of the abdominal arteries, permitting the blood to accumulate in the dependent parts.

Various other alterations of the vascular *tonus* may occur in hysteria, independently of the state of the cardiac action. Patients frequently complain of "rushing of the blood to the head" and flushing of the face, which may assume an intensely red colour; the hands and feet are at the same time pale and icy cold, and the mucous membranes, especially of the conjunctivæ and lips, are anæmic. Hysterical subjects are liable to become pale and to blush alternately, and the flushing of the

face is often accompanied by profuse perspiration. But the alternate contraction and dilatation of the vessels is not confined to the face. In hysterical joint affections Brodie observed that coldness and pallor of the affected extremity existed for some hours daily, to be succeeded by redness, heat, and sweating for a similar period, the latter symptoms in their turn giving place to the normal condition. The hands, which are dry and cold when at rest, often become warm and moist on the slightest attempt at manual exercise, such as writing, and even the cold hands of hysterical patients are often covered by a clammy sweat. The whole body is sometimes prone to perspire, while unilateral sweating is occasionally observed. Neuralgic affections, sometimes accompanied by herpes, are frequently associated with local hyperæmia of the skin in hysterical subjects.

Charcot has drawn attention to the fact that in the complete form of hysterical hemianæsthesia the anæsthetic side not only suffers from comparative pallor and coldness, but bleeds little or not at all on being pricked with a pin. His attention was first drawn to this peculiarity by observing, on leeches being applied to a patient affected with hysterical hemianæsthesia, that their bites yielded very little blood on the anæsthetic side; while the healthy side bled as usual. Charcot believes that hysterical ischæmia may furnish an explanation of certain reputed miraculous occurrences, as, for instance, of the statement made on good authority that in the epidemic of Saint Medard the sword blows given to the "convulsionnaires" did not cause bleeding. The amenorrhœa, so frequently associated with hysteria, is probably often caused by local ischæmia, although it sometimes results from the general anæmia which underlies both affections.

Not less remarkable than hysterical *ischæmia* is what may be termed hysterical *hyperæmia*. Hysterical hyperæmia sometimes leads to profuse and frequently repeated menstruation, although, no doubt, both the menorrhagia and hysteria sometimes result from ovarian disorder. In hysterical ischæmic amenorrhœa hæmorrhages may take place from other organs, and these are generally regarded as vicarious of menstruation. The mucous membranes of the nose, throat, stomach, and lungs are the favourite sites of these hæmorrhages; but in rare cases

they occur from any portion of the surface of the body, and in the absence of any recognisable lesion of the skin. Hæmorrhages may occur in the hysterical quite independently of any disorder of the menstrual functions, and in such cases they are due to local congestions of the affected organ.

In hysterical *hæmatemesis* it is very difficult to be certain of the absence of gastric ulcer, since the presence of hysterical symptoms does not exclude the co-existence of organic disease. In the former case, however, the general health does not suffer in proportion to the apparent gravity of the symptom, the patient may eat indigestible articles of diet with impunity, and there may be little or no general gastric disturbance. Sometimes, however, the diagnosis can be made only after prolonged and careful observation. The same remarks apply in great measure to hysterical hæmoptysis. Whenever hæmoptysis occurs in a hysterical patient, the most careful examination of the chest should be made, and the temperature taken night and morning for some days.

In the examination of hysterical patients one must be constantly on the alert against deception. Such patients simulate hæmoptysis by sucking and drawing blood from their gums, and hæmatemesis by mixing the blood of animals with vomited matters, or even by drinking the blood first and then irritating the fauces so as to induce vomiting. What is true with regard to internal hæmorrhages is still more so with respect to hæmorrhages from external surfaces. In the phenomenon known as *stigmatisation*, large bullæ form on various portions of the surface, especially on the hands and feet, chest, and forehead. These bullæ are at first filled with clear serous fluid, which soon becomes of a bloody colour, and then, after subsidence of the vesicle, a flow of blood takes place from the surface which is tolerably abundant and may persist for a long time.

There are well authenticated cases of hæmorrhage from the surface of the body. Parrot observed an escape of blood-coloured fluid from the skin of the fingers, knees, thighs, chest, and conjunctiva in a patient at different times during general convulsive attacks. Laycock mentions a case in which there was bleeding from the nipple, and quotes a similar case from Sir Astley Cooper, while subcutaneous extravasations have

been frequently observed in hysteria. When small bleeding points are observed on the surface of the body, suspicion of deception should immediately be aroused. Mention has already been made of the fact that in hysterical patients ischæmia frequently alternates with hyperæmia, and if during the former stage several pricks were made with a pin in the portion of skin affected, these would not bleed at first, but might bleed freely a short time afterwards when the hyperæmic stage supervenes.

Sudden elevation of temperature of the body is one of the most remarkable phenomena of hysteria. In the case of a lady who suffered from anomalous nervous symptoms after a fall from a horse, under the care of Mr. J. Teale, a temperature of 122° F. was recorded. More or less similar cases have been observed by Dr. Donkin. The following case, reported by Dr. Steell, is a good example :—

M. M——, 20 years of age, a nurse in the Manchester Royal Infirmary, first came under medical treatment on the 24th of October last. She had been out nursing a case of erysipelas, and seemed impressed with the idea that she had contracted the disease. A slight blush above each ankle and a transitory and slight pyrexia were, however, all the evidences of disease she then presented. A few days after admission, when the pseudo-erysipelas had entirely subsided, retention of urine was complained of, and it was learned that about a year previously she had been similarly affected, but had not come under medical supervision. The urine was drawn off twice daily, and various remedies were employed, without restoring voluntary micturition. As the case was regarded as essentially hysterical in nature, she was allowed to go on duty, care being taken that no undue distension of the bladder occurred. Her pulse and temperature at this time were always found to be normal. Abdominal pain was occasionally present, and, perhaps wrongly, was attributed to distension of the bladder. Menstruation did not deviate materially from the normal. Other remedies having failed, faradisation was employed, one electrode being attached to the stilette of a gum-elastic catheter, which was passed into the bladder, a sponge electrode being applied externally. Acute cystitis followed this application on one occasion, and during the attack ample evidence was given that the power of voluntary micturition was entirely wanting. The cystitis passed off in a few days, and did not seem to have been an unmixed evil, some return of voluntary micturition following it. For this cystitis she had been again warded on December 8th. About this time Dr. Thorburn, obstetric physician to the Infirmary, examined the patient, and found a prolapsed ovary enlarged and very tender. She stated that she had frequently pain on defecation, and abdominal pain became a frequent and distressing symptom. This was chiefly referred to

the left iliac region, where there was also tenderness on pressure, but not limited entirely to that spot. Her temperature at this time became unsteady, as shown by the following observations: December 8th, evening, 100·6°; 9th, evening, 98·6°; averaging 101° from the 11th till the 24th, when it reached 103·2° in the morning. This temperature was not maintained, and the thermometer registered 100·4° on the evening of the 25th. The temperature remained about 101° (reaching the normal, however, on the morning of the 28th) till the 30th, when the remarkable abnormity which it is the object of these lines to record began to manifest itself, temperatures of 105° and 98·6° (the former occurring during a rigor) being observed the same evening. The temperature after this continued irregular, as before, till the 3rd of January, when the thermometer in the axilla registered 106·6° at 10 p.m. during a rigor (pulse 132). On the 8th, 105·8° was noted under similar circumstances, but shortly after 99·4°. The following two days it remained normal, and on the 11th rose only to 102·6°, again to fall to normal. I regret that I am unable to furnish an unbroken curve of temperature observations, but I trust the facts I can substantiate will lose none of their value on that account. Rigors now began to occur with increasing frequency, each being accompanied by a rapid and great rise of temperature. They were irregular in occurrence, and did not conform to any definite type. Perspiration usually followed. It will be seen that the general form of the temperature curve would most closely resemble that of pyæmia, and the possible existence of an abscess in or about the ovary could not but suggest itself. The general condition of the patient was at variance with this hypothesis, the well-known features of intense illness being absent. I shall merely mention some single observations of temperature, giving the date and time. It may be taken for granted that the high temperatures were those observed during or immediately after a rigor.

January	24th, 11 a.m.	107·2°	
„	24th, 12 a.m.	102·2°	
„	24th, 9 p.m.	101°	
„	25th, evening	98·4°	
„	29th, „	100·8°	
„	30th, morning	97·8°	
„	30th, evening	99·6°	
February	4th, „	105°	
„	5th, „	105·2°	
„	6th, morning	99·8°	
„	10th, „	105·4°	
„	16th, „	99·4°	
„	16th, evening	105·6°	
„	24th, morning	98°	
„	24th, 5-35 p.m.	108·6°	
„	24th, 10 p.m. (?)	98·4°	
„	26th, 6 p.m.	112°	
„	28th, 4-30 p.m.	113°	
March	1st, morning	98·2°	
„	1st, evening	100°	
March	3rd, 8-45 a.m.	111°	
„	3rd, 10-30 a.m.	108·2°	
„	3rd, 11-30 a.m.	99·2°	
„	4th, 5-15 a.m.	107·4°	
„	4th, 8-45 a.m.	108°	
„	4th, 4 p.m.	112·4°	
„	5th, 8-50 a.m.	108·6°	
„	5th, 2-30 p.m.	106°	
„	5th, 3-45 p.m.	107·4°	
„	7th, 10 a.m.	109°	
„	7th, 8-30 p.m.	102·6°	
„	8th, 6 p.m.	111·2°	
„	11th, 4-30 p.m.	111·2°	
„	12th, 10 a.m.	108°	
„	17th, evening	100·4°	
„	18th, 3-30 p.m.	116·4°	
„	19th, 4 a.m.	116°+	
„	19th, 8 p.m.	98·6°	
„	21st, 9-30 a.m.	111°+	
„	23rd, 9-30 a.m.	106°	

During the last few weeks of treatment mental disturbance has supervened. At first this accompanied the hyperpyrexia only, but latterly it has been nearly constant. She has no fixed delusion, and what delusions she has manifested have been clearly founded on recent events which occurred around her. They tended towards the melancholic type of insanity. At times she has been exceedingly violent, and always more or less suspicious. There is no family history of insanity.

I believe that the hyperpyrexial attacks were of very short duration, and where, by the above figures, a prolonged high temperature is indicated, it is only apparently so, the observations having been made during the rigors. All were made in the axilla, and different thermometers were used, several of which had their registering columns driven into the bulb at the top (indicated above by the symbol +). I should mention that the retention of urine noted at the commencement has continued, with occasional intermissions of varying duration.

These lines were written towards the end of March, and it remains for me in a few sentences to narrate the subsequent history of the case. The attacks of hyperpyrexia ceased (though transient pyrexia occurred from time to time) on the advent of a new series of phenomena, including general convulsions of extreme violence, accompanied by opisthotonos, loss of consciousness, lividity, &c., and followed by persistent trismus, simulating in a remarkable way the traumatic form of that affection. These severe symptoms gradually diminished in intensity and frequency, while general amelioration in the patient's condition set in, so that on the 12th of April she was able to be removed to the Cheadle Convalescent Hospital. There her convalescence continued with but slight interruptions, and she is now on full duty as a nurse in the institution.

Hysterical patients suffer from various anomalies of secretion and excretion. Increased flow of saliva is not an unusual symptom after a hysterical attack, and it occurs occasionally independently of the fits. At other times an abnormal dryness of the mouth, along with great thirst, is present, inducing the patients to drink large quantities of fluid.

The gastric secretion is sometimes largely increased, and may take place independently of the ingestion of food. Hysterical vomiting is sometimes caused by spasm of the muscular coat of the stomach, but at other times it appears to be determined by the irritating effect of the excessive gastric secretion upon the mucous membrane of the organ, while in many cases both conditions are probably present in co-operation. When the vomiting is due to excessive secretion, large quantities of fluid are ejected, even during fasting, and the appetite is sometimes greatly impaired, and at other times enormously increased.

Hysterical vomiting is not often accompanied by much loss of flesh, but great emaciation may take place in hysteria in the absence of vomiting or any recognisable lesion to account for it. This condition has been described by Lasègue under the name of *hysterical anorexia*, and by Sir W. Gull as *apepsia hysterica*. "These patients," says Dr. Wilks, "declare that they do not care for food, and so they take less and less until all appetite has gone, and then, indeed, a loathing may come on." In a case of this kind, that of a girl aged 18 years, which came under my observation, the emaciation was extreme, the skin being stretched over the face so as to reveal all the depressions and prominences of the jaws and malar bones. The condition of the patient reminded me forcibly of the appearance presented by those suffering from chronic starvation, due to organic stricture of the œsophagus. Some months subsequent to my seeing the patient her parents changed their residence; she almost immediately began to eat, and became quite plump in a few weeks.

Hysterical vomiting is sometimes the complement of hysterical suppression of urine. In such cases, when the suppression of urine is complete and of long continuance, the quantity vomited is large, and in a case observed by Charcot a considerable quantity of urea was detected in the vomited matters. Fernet also found urea in the vomited matters in a case of this kind. It is therefore probable that the vomiting is caused by the supplemental elimination of urea by the stomach.

The renal secretion undergoes frequent alterations in hysteria Hysterical polyuria is a very constant symptom after convulsive attacks, the urine under such circumstances being pale and of low specific gravity.

Hysterical anuria, although seldom met with, is a more interesting phenomenon than polyuria. Almost total suppression of urine may exist for a period of weeks or months without giving rise to serious symptoms besides the constant vomiting. Charcot has shown that this curious phenomenon depends, not upon a spasmodic condition of the ureter, but upon some disorder of the kidneys themselves, probably vaso-motor contraction of the renal arteries analogous to the hysterical ischæmia already described as occurring on the surface of the body.

Increase of the vaginal and uterine secretions frequently occurs in hysterical patients. Leucorrhœa, resulting from uterine disease, may sometimes be regarded as a cause of hysteria, but at other times the local discharge must be attributed to nervous influence.

(*d*) *Psychical Disturbances.* — The chief mental characteristic of hysterical patients is an excessive emotional excitability, unchecked by voluntary effort, which finds expression in various ways. Both pleasant and unpleasant emotions are excited in them with unwonted ease, so that, as Reynolds remarks, "the patient is hurried from one extreme to the other with ludicrous rapidity; and often she walks, as it were, on the narrow line where tears and laughter meet. Laughter and sobbing not only alternate but co-exist, and often without any obvious and sufficient reason for either." This excessive emotional activity necessarily induces exhaustion, and an exhausted nervous system is adapted for the retention of the painful emotions, so that, as a rule, hysterical patients are irritable, gloomy, and not only exaggerate bodily ailments which exist, but imagine those which have no existence.

Another mental peculiarity of hysterical patients—a peculiarity which lies at the root of almost all their other mental derangements—is *craving for sympathy.* Sympathy is the quality of mind which adapts man for the social state, and is the foundation of all his moral actions; the highest natures must necessarily crave for the sympathy of their fellows; and the more highly the mind is developed the more deeply rooted will the craving for sympathy probably become. Persons with well-regulated minds, however, perceive that those have no right to claim the regard, esteem, and sympathy of others, who refuse to be sympathetic in their turn and to perform the actions which are prompted by an active sympathy with the feelings and sufferings of others. They are constantly devising schemes by which they can alleviate the sufferings of other people, and thus add in some form or another to the stock of human happiness; and although they could not bear with equanimity to be regarded with dislike by their fellows, especially by those whom they esteem good and wise, yet the desire for any active demonstration of sympathy is exceedingly

small. To think of others becomes a second nature, and the true method by which to purchase the inestimable boon of human sympathy. Sympathetic natures of this class are necessarily deeply emotional, but the life of active benevolence which they lead renders it necessary for them to develop the intellect in adapting means to ends and the will by the daily exercise of self-control. Such natures are emotional, but they are also strong-willed and of vigorous intellect; in one word, their minds are well-balanced and healthy.

Contrast these individuals with the habitually hysterical. Both are emotional, and both crave for sympathy; but while the former purchase sympathy by actively bestowing it, the latter would like to be its recipients while refusing it to others. The former are unselfish and devoted to the interests of others, while the latter are selfish and regard themselves as the centre of the whole world of feeling, thought, and action.

It is this morbid desire for sympathy that prompts hysterical patients either to exaggerate a real ailment or to feign illness when they are free from it, or even to inflict bodily injury upon themselves for the purpose of arousing compassion and attention. Scarcely a disease can be mentioned which may not be simulated by the hysterical, and the methods they adopt to effect their object are truly marvellous, and would be utterly incredible unless attested upon undeniable evidence. In order to excite compassion, some injure and burn themselves, induce purulent cutaneous eruptions by the use of irritating ointments, swallow needles, or even pretend that they are about to commit suicide, although real attempts at suicide are rare.

The depraved ideas formed by hysterical patients, and the degrading actions resulting from them, defy all description. Some have drunk urine and eaten excrement in order they may vomit them; others have led their too credulous attendants to believe that urine issued from their navels, breasts, ears, or eyes; others, again, have introduced living animals, such as frogs and worms, into the anus or vagina, so that they might, by reproducing them, excite wonder, and become objects of sympathy to their friends.

But there are lower depths of human degradation which hysterical females do not fail to reach. They sometimes

intrigue against their friends, and maintain that they are persecuted and outraged, and play their part with such consummate skill as to deceive experienced physicians and learned judges. Girls about the age of puberty sometimes commit the greatest crimes which it is possible to imagine, without any recognisable motive, and apparently from the sheer love of mischief. "When you see a paragraph," says Dr. Wilks, "headed 'extraordinary occurrence,' and you read how every night loud rapping is heard in some part of the house, or how the rooms are being constantly set on fire, or how all the sheets in the house are being devoured by rats, you may be quite sure there is a young girl on the premises."

I have known a young lady at a boarding school tear her sheets and her own underclothing into shreds, and then endeavour to fasten the guilt upon a schoolmate. Another placed a carving-knife under her pillow, and when it was discovered, as was doubtless intended, she confessed to an intention of committing suicide.

These, however, are only a few of the minor vagaries which may be committed by hysterical girls. A young nurse girl has not unfrequently been known to poison the children under her charge, at other times the attempt is directed against her mistress, and it is so clumsily carried out that the lives of the whole family may be endangered. At times a piece of burning coal may be placed under the infant in the cradle, and repeated attempts be made to set the house on fire, all this being done while the girl is treated with the utmost consideration and kindness by her employers.

The well-known case of Constance Kent testifies to the frightful crimes which may be perpetrated by young girls in this strange condition. Dr. Wilks, in alluding to the case, remarks: "When a few years ago the whole country was shocked by the news of the murder of a little boy in the middle of the night whilst surrounded by members of his own family, the event was enveloped in the darkest mystery, seeing that the crime was of so extraordinary a character, and was wanting in all those objects for its commission which are usual in similar deeds. No adult, especially no man in his senses, commits a crime except to attain some end; and, therefore, the very

purposelessness of the act (except, perhaps, for revenge) convinced me that it was perpetrated by a young woman. I felt quite sure in my own mind as to the real criminal, who, even after her own confession, was considered by many incapable of such a deed."

It is not unusual for the psychical disturbance of hysteria to assume an erotic character. Girls may then assert that they have been ravished, and usually maintain that the most outrageous violence was used by the perpetrator of the crime, or that they themselves were previously drugged. The notes of the following case have been kindly supplied to me by Mr. Cullingworth, who was the medical witness called in by the police to investigate the case after her supposed dying statement had been taken :—

In December, 1876, a girl of eighteen was found one evening standing, with her clothing wet and muddy, and in an apparently stupefied condition, in the closed doorway of a restaurant in the centre of Manchester, a few yards from where she was lodging. She was taken home and to bed, and a medical man was sent for. He found her to all appearance unconscious of what was going on around her, and uttering some disjointed and incoherent complaints of having been drugged and threatened. He thought she was recovering from the effects of some narcotic, and did not at first pay much attention to her story. The following day, however, she appeared worse, and in the evening her condition was considered so critical that the police were communicated with, with a view to her statement being taken down. She was visited by two experienced detectives, who, seeing how matters stood, and having the doctor's assurance that she was in a dying state, sent at once for a magistrate, before whom she made a solemn declaration to the following effect: She believed herself to be dying. On the previous evening a solicitor, at whose office she had called on business, told her that she must go into a convent, and gave her "some sort of dark, sweet drink," which rendered her senseless. On going downstairs from the office she met a Jesuit father, whom she had seen once before. This gentleman took hold of her and pulled her along the street to a little house in a court, where there was an upper room with a bed in it and a cross on the wall. Having got her into this room, he said improper things to her, and gave her a little cake which affected her directly. The woman of the house came into the room and found her on the floor, after which she somehow got outside, the priest following. He again dragged her along in the dirt to the street corner, when he ran away.

The solicitor and the priest, both of them well-known and highly-respected, were thereupon placed under arrest in the middle of the night

on a charge of having administered certain poisonous drugs with intent to murder. The story was proved to be purely imaginary, and the magistrates dismissed the case.

It is not only the actions which are in immediate relation with the emotions that are so profoundly disturbed in hysteria, but grave disorders of the representative feelings and cognitions occur. Illusions of sight are common in the early stages of severe hysterical attacks. Charcot has drawn attention to the fact that in the grave cases of hysteria associated with ovarian hyperæsthesia and hemianæsthesia the patients frequently see rats and other odious animals on the anæsthetic side of the body, or with the eye suffering from amblyopia. At other times they have hallucinations with erroneous ideas, and some of the images that have appeared during the paroxysm are subsequently considered real. The entire hysterical paroxysm may consist of a succession of images like that occurring in vivid dreams, a condition generally described as hysterical delirium.

If the attacks be transient and occur rarely, the patient may recover completely from their effects; but if they recur frequently, persistent mental aberration is after a time established. The delirium frequently makes its appearance in a perfectly periodical manner and without any recognisable cause, being regularly preceded by a condition of ill-humour or mental irritability. In other cases a chronic and continuous form of mental disturbance is established, which is little affected by the fits.

One variety of this mental condition assumes the form of pure melancholy. The patients are anxious, wretched, and incapable of enjoying the society of others, and under such circumstances they are subject to uncontrollable impulses which urge them to commit outrageous actions.

A second form of chronic continuous hysterical mental disturbance corresponds closely to the clinical description of the so-called *folie-raisonnante.* Patients affected in this way pursue their own selfish aims with the greatest perseverance, although they are unfit for any useful employment; the sexual appetite is often strongly developed, and not rarely they are given to drunkenness. There is complete absence of the moral

sense; they are quite unable to curb their inclinations and impulses; they are liars, and cheat and steal with the greatest cunning and dexterity; and are always ready with plausible reasons to cloak the perversity of their actions, while they manifest the utmost confidence in the incontrovertible nature of their arguments. In these cases the intelligence is profoundly disturbed, for it is evident that the statement which was at one time a conscious fabrication to meet an emergency is afterwards reproduced by them with a full belief in its truth. They frequently suffer from hallucinations, which may gradually become transformed into established erroneous ideas. They are besides subject to occasional outbursts of excitement, which after a time pass into pronounced *maniacal states.*

(2) Hysterical Attacks.

Hysterical fits are exceedingly variable in the combination of their symptoms and the degree of their intensity, so that it is impossible to comprise all the various forms under one general description. The attacks occur sometimes without any recognisable cause, while at other times they are provoked by over-excitement or some slight emotional disturbance. The paroxysm always takes place when someone is present to witness it, and never during sleep, nor when the patient is alone.

(*a*) *Simple Hysterical Attack.*—The attack is preceded by the sensation of globus along with a feeling of suffocation, a painful dragging in the extremities, pain and giddiness in the head, singing in the ears, or darkening of the field of vision. It is often preceded by a fit of crying or laughing, or a combination of both; the patient suddenly screams or makes a spluttering noise, and falls down in a state of apparent unconsciousness. The head and extremities become affected with general rhythmical clonic convulsions, the breathing is accelerated and exaggerated, irregular, or temporarily arrested. The loss of consciousness is more apparent than real. The hysterical patient generally hears what is said by those around her, and she has almost always time to find a suitable place upon which to fall; she often throws herself on a couch or reclines on a sofa, and not unfrequently appears to bestow some degree of attention upon the propriety and gracefulness of her attitude.

Another peculiarity of the hysteric attack is that the facial expressions and attitudes assumed are not devoid of meaning, but are repetitions of those occurring in health under varying emotions. Sometimes the expression is that of great terror, at other times there is a frown as if of anger, and at still other times it becomes imploring or beseeching.

Hysterical attacks rarely last more than a few minutes, but they may recur in quick succession, so that they seem to form an almost continuous paroxysm, extending over a considerable period. The hysterical seizure frequently ends in a fit of crying and sobbing, there is no subsequent coma, and on recovery the patient generally passes a large quantity of clear and limpid urine of low specific gravity.

(*b*) *Cataleptic attacks* are liable to occur in hysterical patients; they are of variable duration, disappearing sometimes in the course of a few hours, and being prolonged at other times, with slight intermissions, for a period of months. Cataleptic rigidity is sometimes limited to particular limbs; but, as a rule, the whole body is implicated, and then all voluntary movements are suspended and reflex action is diminished. All the forms of general sensibility are usually lost, but one or more of the special senses may be retained. The sense of hearing is probably the one most generally retained, a fact which should be borne in mind by the attendants.

Sometimes every form of sensibility appears to be completely abolished, and in such cases the limbs retain the position in which they are placed. This condition has been called *waxy* rigidity, because the limbs can be as it were moulded in almost any position.

There are cases of complete general muscular relaxation in which the action of the heart and pulse become almost imperceptible, while respiration may be so feeble that the patient may seem to be dead. These cases have been described as "hysterical trance," and it is possible that patients may have been buried alive in this condition.

(*c*) *Hysteria in Boys.*—Boys, at the approach of puberty, not unfrequently suffer from hysterical symptoms resembling those observed in the female sex (Wilks, Roberts). Sometimes the symptoms may assume the form of globus along with attacks of

causeless weeping and sobbing; at other times there may be partial spasm of the glottis, a barking cough, attacks of dyspnœa, or some local sensory or motor disturbance. Psychical phenomena often predominate. In a case which I saw a few months ago the boy was sometimes found creeping on his hands and knees and barking like a dog; another time he jumped like a frog from the floor on to the table. The depraved form of hysteria, named *chorea major*, is often met with in boys. In this variety of the disease the patients run, dance, jump, or climb with much greater readiness and dexterity than similar actions could be performed in health, or they may sing or recite poetry, even in a foreign language.

The paroxysm of *hystero-epilepsy* will be subsequently described.

§ 940. *Course, Progress, and Terminations.*—The grouping of the symptoms in hysteria is exceedingly variable, not only in the case of different individuals, but of the same individual at different times. It is almost always a chronic disease, which exists for years, and disappears only at an advanced age. Those who are strongly predisposed to hysteria are frequently very irritable and peevish during childhood, although convulsive attacks do not generally occur until the period of puberty. When puberty is established the convulsive attacks frequently disappear, and other symptoms, more especially emotional disturbances, are then apt to become prominent.

In some hysterical patients sensory and motor disturbances are well marked and persistent, and psychical only present to a slight extent, while at other times the reverse is the case. The disease is liable to undergo many variations in its course. The symptoms may disappear for comparatively long periods, but are liable to recur on exposure to the slightest excitement.

Hysterical symptoms frequently cease after the climacteric period, but the higher degrees of mental disturbance sometimes develop at this age.

Hysterical symptoms may appear at puberty, but subsequently become latent to a certain extent, reappearing in a very pronounced manner at the climacteric period. Hysterical symptoms may be of every degree of intensity from simple

mental irritability up to the profound mental disorders which border upon insanity, and from slight sensations of globus and infra-mammary neuralgia to attacks of general convulsions, and widely spread paralysis and anæsthesia. The cases in which convulsions, paralysis, anæsthesia, and contractions are associated with severe psychical disorders constitute the most aggravated forms of the disease.

Hysteria seldom shortens life. Even cases of persistent vomiting and copious hæmorrhage are relatively innocuous in comparison with similar symptoms arising from other diseases. Cases, however, are occasionally observed in which after a severe moral shock violent hysterical symptoms become developed, and death may occur within a few days or weeks in the absence of any recognisable organic change.

In a case of hystero-epilepsy observed by Wunderlich, the patient suffered from epileptiform attacks, not attended by any increase of temperature, for more than eight weeks, when suddenly, without known cause, the patient became collapsed, and the temperature rose to 109·4° F. (43° C.) before death. In a second case, related by the same author, the patient suffered for several years from various forms of paralysis, hyperæsthesia, loss of sight and smell; ultimately difficulty of swallowing and vomiting supervened, and she died with febrile symptoms in a state of marasmus and emaciation, while post-mortem examination revealed no changes in the nervous system.

Sometimes death may occur indirectly from hysteria, as in the cases of patients who mutilate themselves, with or without the intention of committing suicide. Hysterical patients rarely attempt to commit suicide in earnest, but feigned attempts made in order to attract attention have sometimes been fatal, and occasionally suicide has actually been committed.

§ 941. *Morbid Anatomy and Physiology.*—No constant anatomical changes have been found in cases that have died from hysteria. Charcot discovered symmetrical sclerosis of the lateral columns extending nearly the whole length of the spinal cord in the case of a hysterical woman who suffered, for ten years, from paralysis with contracture of all the extremities. It is, therefore, probable that in cases of hysterical contracture

the fibres of the pyramidal tracts undergo morbid changes, at first temporary, although ultimately becoming permanent. But even if this be so, the primary change probably occurs in the motor centres of the cortex of the cerebrum. Indeed all the phenomena of hysteria may be explained most readily on the assumption that the irritability of the cortex of the brain is sometimes in excess and sometimes diminished or abolished. Increased irritability of the cells and fibres of portions or the whole of the cortex of the cerebral hemisphere supplied by the posterior cerebral artery would account for the hyperæsthesia of variable distribution and completeness on the opposite side of the body; and, conversely, diminution or loss of the irritability of those same cells and fibres would account for the various forms of anæsthesia.

Again increased irritability of the cells and fibres of portions of the cortex of one hemisphere supplied by the middle cerebral artery would account for spasms of groups of muscles on the opposite side of the body, while diminution or loss of the irritability of these cells and fibres would account for the various forms of paralysis observed. Variations in the degree of irritability of the cells and fibres of the cortex supplied by the anterior cerebral artery would account for many of the psychical disturbances. Even the numerous vaso-motor disorders observed in the course of hysteria are best explained on the supposition that they are determined by variations in the intensity of the nervous discharges from the cortex of the brain to the nerve centres in the medulla oblongata.

Hemianæsthesia from organic lesion is generally caused by disease of the sensory peduncular fibres and of Gratiolet's fibres in their ascent through the internal capsule; and hysterical hemianæsthesia might result from a loss of the irritability of these fibres without recognisable structural change, while hyperæsthesia of half the body might be caused by excessive irritability of them. Muscular spasm or paralysis, on the other hand, might be caused respectively by excess or abolition of the irritability of the fibres of the pyramidal tract. The fact that anæsthesia is sometimes associated with loss of reflex irritability shows that the irritability of the nervous tissues is modified in hysteria in more than one locality at the same time.

§ 942. *Diagnosis.*—When hysteria is fully developed, and the physician has an opportunity of inquiring into the history of the case and of watching its progress, the diagnosis presents no great difficulties. Hysteria may, however, simulate almost every possible disease, and a physician has to be constantly on his guard if he would not at some time fall a victim to the deceptions practised by the hysterical.

Individuals of a *nervous temperament* sometimes exhibit the minor characteristics of hysteria, such as great irritability of temper, exaggerated sensibility to physical impressions, and even occasionally slight motor disorders. Cases of this kind bordering upon hysteria demand the same treatment, so that it is unnecessary to enter upon the diagnosis between the affections.

Hysterical arthritic affections are particularly liable to be mistaken for organic disease of the joints. In the hysterical affection the pain varies in degree at different times and is fluctuating in character, the form of the joint is unchanged, there is no heat or redness, and the pain, like most other local hysterical pains, is limited to the surface, so that slight contact may be painful while deep pressure causes no discomfort, especially if the attention of the patient be otherwise engaged.

Pain and sensitiveness over the spinous processes of the vertebræ in hysterical subjects have led to the affection being mistaken for grave organic disease of the spinal cord, but these symptoms, indeed, are rarely present in the organic diseases. The exciting causes of hysteria simulating disease of the cord are usually emotional disturbances, which rarely induce organic spinal affections.

In hysterical *paraplegia* the lower extremities are feeble and give way under the weight of the body, while the feet drag on the ground. The paralysis is seldom complete, the patient is able to move her limbs in bed with comparative ease, and may even be able to get out of bed; but after walking two or three steps the limbs give way, the gait becomes tottering, and unless supported the patient falls. The electric excitability of the muscles is usually unaffected in hysteria, and the muscles do not undergo trophic changes.

The concomitant symptoms of hysterical paraplegia are anæsthesia of the skin and muscles of the lower extremities, tympanites, constipation, dysmenorrhœa, and retention of urine. Cerebro-spinal multiple sclerosis sometimes closely resembles hysteria, and the diagnosis between the two affections is occasionally only practicable in the later stages.

Hysterical hemiplegia differs from hemiplegia due to cerebral disease, in the following respects:—It is usually accompanied by well-marked disorders of sensibility; there is no facial or lingual paralysis; the paralysis is scarcely ever complete; in the large majority of cases it is worse in the leg than the arm; it is liable to sudden variations in intensity under the influence of emotions; the electric excitability is unchanged; and the muscles do not undergo atrophy.

Hysterical convulsions may be distinguished from epilepsy by negative characters. The loss of consciousness in the former is not complete, nor is it so sudden in its onset; there is no asphyxia; the tongue is not bitten; the attacks last longer than in epilepsy; the patient does not on the cessation of the attack fall into a profound stupor, but only appears exhausted; and there is much sobbing and crying.

It is sometimes difficult to distinguish between hysteria and hypochondriasis. Some, indeed, regard hysteria in the female, and hypochondriasis in the male, as only different manifestations of the same disease. The psychical symptoms of the two diseases are, however, different, and paralyses and convulsive attacks never occur in hypochondriasis.

Hysterical aphonia is seldom accompanied by a cough as is laryngitis, the loss of voice is sudden, while in almost all other forms of aphonia the voice becomes gradually extinct; a laryngoscopic examination shows a total absence of any structural lesion.

Hysterical neuralgia has not the intensity of the genuine disease, and there is an absence of "painful spots." The distribution of pains in hysteria is more diffused than in true neuralgia, and often not limited to any one nerve territory.

In lead paralysis the affected muscles undergo atrophy and manifest the reaction of degeneration at an early period of the disease, phenomena which never appear in hysterical paralysis.

§ 943. *Prognosis.*—So far as life is concerned, the prognosis is always favourable; it should not be forgotten, however, that a fatal issue is possible.

But although hysteria rarely causes death, it is almost equally rare for complete recovery to take place. The most hopeless cases are those in which a predisposition to the disease is inherited, and in which the symptoms commence in childhood. In such cases the disease usually becomes exaggerated at puberty, although considerable remissions often take place after this period. Many patients get well as age advances, and at times the climacteric produces a favourable change; but at other times this period induces exacerbations, and leads to the establishment of inveterate forms of mental disturbance.

The most favourable cases are those in which the disease can be traced to a distinct exciting cause that can be removed, rather than to rooted mental predisposition. When hysteria has been induced by affections of the generative organs or chlorosis, hopes may be entertained that the hysterical symptoms will vanish with the removal of these. Occasionally, however, the altered condition of the nervous system continues after the exciting cause is removed.

Remissions frequently occur in the course of the disease, and may last for years, but the symptoms generally recur from time to time in one form or another.

Sensory and motor disturbances, however severe, may suddenly disappear temporarily; but the symptoms become more obstinate to treatment every time they are repeated, and ultimately become persistent.

Transitory psychical disturbances, even when they are very violent, may not exert an unfavourable influence upon the progress of the case; and even attacks of ordinary insanity supervening in the course of hysteria are capable of complete recovery. When once the mental disturbance has assumed a chronic form the prognosis becomes extremely unfavourable, and when the signs of moral depravity, *folie-raisonnante,* or fixed erroneous ideas have become established, complete restoration seldom occurs.

§ 944. *Treatment.*—The great aim of treatment should be

to prevent the appearance of the aggravated form of hysteria. With this view the greatest attention should be paid to the early education of children who inherit a nervous temperament, or who are congenitally predisposed to hysteria. Such children should be trained to habits of unselfishness, and, under judicious management, considerable progress may be made before the child leaves the arms of the nurse. One great difficulty is often experienced in such cases. When the tendency to the disease is inherited through the mother, she herself is often irritable and explosive in disposition, at one time unnecessarily harsh to her children, again unduly indulgent to them, always capricious and never firm; while the father is too much occupied with business to be able to counteract the evil influence of the mother.

Under such circumstances the mother should be advised to place the management of her children as much as possible in the hands of some firm and judicious woman, be she nurse, relative, or governess. To train a child into habits of self-renunciation is the most difficult of tasks anyone can undertake, and can only be accomplished by the habitual exercise of self-control on the part of the teacher.

During the period of bodily development great care should be taken of the health. The diet should be plain, nourishing and abundant, the utmost care being taken that the child is not treated as an invalid, inasmuch as the observance of special rules with regard to diet is apt to foster that feeling of self-consciousness which it is so desirable to suppress. Regular hours for meals, going to bed and getting up, should be observed as part of general discipline; plenty of outdoor exercise should be taken, all the better if it can be made agreeable, or subservient to some useful purpose to engage the attention. When the young girl passes into womanhood she ought to be taught that she has a field for work and a mission in the world, and that she is surrounded by human suffering which she can alleviate; there is little doubt that the more opportunities of usefulness are multiplied for young ladies the more the tendency to hysteria will diminish.

But let us now pass to the treatment of the fully developed disease. The exciting cause can only be removed in a few cases

and even when that is possible the modification of the nervous system already induced may persist. When anæmia exists the usual treatment with iron and other tonics should be adopted. If there be indigestion with flatulence, bismuth, charcoal, alkalies, or mineral acids may be administered with or without a better infusion; constipation demands the administration of mild aperients, and atony of the alimentary canal quinine, nux vomica, or strychnine, while uterine derangements must be subjected to appropriate treatment. The treatment of disease or suspected disease of the generative organs in cases of hysteria requires the greatest tact on the part of the medical attendant. The introduction of the catheter, or a vaginal examination, if the patient be unmarried, may lead to an aggravation of all the symptoms. On the other hand, if real disease exist, local treatment may be indispensable to success. When the cause of the hysteria is a moral one it is often impossible for the physician to remove it, but even then much may be done by placing the patient under conditions as much as possible conducive to her happiness.

The drugs which have been employed in the treatment of hysteria are characterised by a pungent and often disagreeable odour. Assafœtida, galbanum, and valerian are the best known and most commonly used of this class, and many hysterical patients come to like both the taste and smell of these nauseous substances. They do not appear to produce a permanent influence upon the disease, although they may be of use in removing flatulence and other troublesome symptoms.

When general hyperæsthesia and sleeplessness exist, it may be necessary to administer narcotics, morphia injected subcutaneously being the best. The patients, however, should never be allowed to take morphia or other narcotic at their own discretion. Bromide of potassium is a valuable agent in some cases, and where there is much sleeplessness its efficacy is increased by being combined with chloral.

The electrical treatment of particular symptoms of hysteria is sometimes followed by improvement of the general condition. Beard and Rockwell have recently recommended that the entire surface of the body be treated, in successive portions, with tolerably strong induced currents, and this treatment has

been found very successful, although its efficiency depends probably on the mental impression produced rather than on the local action of the current. The influence exerted by counter irritation of various kinds appears also to be largely due to the mental impression made.

The most important part of the treatment of hysteria consists of the moral management of the patient. One of the first requisites for treatment is that the physician should possess the entire confidence of the patient and her friends. An essential condition for success is that the physician be himself convinced that hysteria is a real and not a sham disease, and that the treatment prescribed is a real and not a sham treatment. There is probably no disease which a medical man has to treat which makes so many demands upon his firmness, self-control, and ingenuity as hysteria. He must be able to sympathise with the feelings of the patient in order to command her confidence, but if his sympathy degenerate into false sentiment or into the slightest exaggeration of manner he will only be administering to the morbid craving for sympathy which is at the root of the disease, while on the other hand the slightest attempt to laugh down the patient's fancies may be fatal to success. Earnestness of purpose, determination, and fertility of resource are all called into requisition in the treatment of hysteria. The instructions given to the patient should be plain and such as can be readily carried out; they ought at the same time to task to some extent her voluntary efforts. When once instructions are issued the most implicit obedience to them should be insisted upon, for laxity in enforcing observance of rules argues want of confidence in their efficacy. The voluntary efforts of the patient should be daily exercised by making progressively increasing demands upon them. And as the power of will becomes strengthened, emotional excitement will diminish in corresponding degree.

Instead of exciting the dormant will into activity by graduated exercises, this can frequently be accomplished at once by anything which acts powerfully on the imagination. Hysterical patients, who have been bed-ridden for years, have been known to get out of bed, and walk under the influence of a strong emotion, or after having the imagination powerfully

acted upon, so that the rapid change from utter helplessness to full motor power appears almost like a miracle. Whatever may be the reason of it, there can be very little doubt that such wonderful cures are generally effected by clergymen or irregular practitioners, and rarely by the sedate, highly respectable, regular practitioner.

Painful remedies used with moderation and humanity are sometimes very useful, partly by appealing to the imagination. Of these remedies, the faradic current is probably the most generally useful, and a subcutaneous injection of morphia is for a similar reason more efficacious than the internal administration. Simple water has been successfully substituted for the narcotic injection, so great is the influence of mental impression.

It is sometimes possible to cure severe hysterical symptoms by means of threats, sudden fright, or moral shock. An unexpected shower of cold water may have the desired effect, and at other times the threat of the actual cautery is efficacious. Charcot mentions the case of a woman who had suffered for more than two years from permanent contracture of one of the lower extremities, which suddenly vanished on account of the moral shock caused by an accusation of theft brought against her; a second also in whom contracture of four years' standing suddenly disappeared in consequence of a stern admonition given her; and a third in whom a like condition vanished on account of a sudden disappointment. Great judgment must be exercised in the employment of such means, inasmuch as the disease may be aggravated instead of ameliorated by them.

Hysterical patients are treated with much greater success in the wards of an hospital than in their own homes. In aggravated cases, therefore, the patient should be removed from her home and relations and placed for a period of some months either in a public institution, or under the care of complete strangers.

During hysterical fits the patients usually take care not to injure themselves, but it is often necessary to prevent respiration being impeded by clothing. I have always found it better not to allow a hysterical woman to toss about violently

in all directions, but to hold her down gently but firmly, and to prevent her struggles. The best method of securing the patient is to place an attendant on each side of her, and to direct each to grasp one wrist with one hand and to hold the shoulder firmly down to the bed or floor with the other; if necessary the patient's legs must be held down. It is remarkable how soon a hysterical patient ceases to struggle when she finds that she is held as in a vice. So long as the patient can shake herself free from her attendants, so long is the struggle apt to be maintained, but when once she feels that she is thoroughly restrained, and that her efforts do not find outward expression, she generally ceases to struggle. The most usual remedy for arresting the attack is to dash cold water on the face and neck; although this is a useful method occasionally it is by no means the best. The plan suggested by Dr. Hare of forcibly holding the mouth and nose of the patient so as to prevent her from breathing is very effectual. In my own practice the administration of an emetic was at one time a favourite method of arresting an attack. Strong, vigorous servant-maids sometimes suffer from violent hysterical convulsions, and in them I was in the habit of giving an emetic dose of the tartrate of antimony. In most cases, however, the sulphate of zinc is safer if less efficient.

If the attack be caused by some peripheral irritation, its removal may arrest it. Severe attacks have sometimes ceased after the rectification of a misplaced uterus. In cases of hystero-epilepsy Charcot has succeeded in arresting the attacks by firm compression of the hyperæsthetic ovary, but this does not appear to be a means applicable for the convulsive attacks of ordinary hysteria. Large doses of the bromide of potassium exert a favourable influence on the fits of hystero-epilepsy, but this remedy is not of much value in the common form of convulsive attack.

Limited spasms in hysteria are best removed by general treatment and moral management, although local treatment and special remedies are occasionally found useful. In the case of patients suffering from obstinate globus, Erb recommends the repeated application of the constant current, the anode being placed at the side of the larynx.

For spasms of the stomach and bowels, sometimes warm, at other times cold applications to the abdomen are useful; opium, belladonna, quinine, valerian, or other drugs of the same class may be given internally.

Vomiting is one of the most troublesome and obstinate symptoms of hysteria. It is usual to regulate the diet, and to give only bland and easily digestible substances, such as milk cooked in various forms, but this method is not always successful. Sometimes the patients retain highly-seasoned food better than bland diet, and the vomiting may cease if they are allowed to eat raw ham or raw mince-meat well peppered. Care, however, should by taken that the nature of the case is clear. In spastic as well as in paralytic retention of urine warm sitz-baths will often bring relief; when they are unsuccessful the catheter should be employed in case the bladder becomes distended.

In hysterical paralysis electricity is the best remedy, and the application of strong faradic currents to the paralysed nerves and muscles is, as a rule, successful. Galvanisation of the spinal cord is useful in hysterical paraplegia. Aphonia may at first be treated by external faradisation, but in obstinate cases recourse must be had to the intralaryngeal application.

Passive movements and frictions of the limbs with or without embrocations are also useful as aids in treatment, and Reynolds strongly recommends the application of narrow strips of blister passing completely round the affected limbs. Compression of the larynx with the fingers sometimes temporarily restores lost speech, and a similar effect may be obtained by passing a strip of adhesive plaster across the larynx so as to partially surround the neck. Hysterical tympanites may at times be dispersed by powerful faradisation of the abdominal muscles or even by firm compression of the abdomen, but in aggravated cases the introduction of the intestinal tube may be necessary.

Hysterical anæsthesia is best treated by powerful faradic currents applied by means of the dry metallic brush. Charcot has succeeded in removing hysterical hemianæsthesia by applying various metals to the cutaneous surface. This method of treatment was introduced by Burq, but it would appear that the

anæsthetic condition is only transferred from the affected to the opposite side of the body. It is somewhat premature to pass any opinion with respect to the success of this practice. So long as certain "passes" over the surface of the body can induce a condition of general anæsthesia, such as that met with in the mesmeric state, there is nothing absurd in supposing that similar passes and the application of coins to the surface may have a curative effect in conditions of partial anæsthesia.

Hysterical neuralgia and hyperæsthesia demand the employment of narcotics and anæsthetics. Caffein, guarana, and chloride of ammonium are useful in hysterical cephalalgia. Chloral hydrate given in scruple and half-drachm doses to procure sleep is occasionally useful, and inhalation of chloroform may be necessary in order to overcome contractures of muscles.

CHAPTER V.

CATALEPSY, TRANCE, ECSTASY, AND OTHER ALLIED CONDITIONS.

(I.) CATALEPSY.

CATALEPSY is characterised by attacks of partial or complete loss of consciousness, accompanied by stiffening of the voluntary muscles, having the peculiarity that the limbs retain for a relatively long period the positions in which they may be placed by passive motion.

§ 945. *Etiology.*—Catalepsy often occurs as one of the many manifestations of hysteria, while at other times it is caused by chronic cerebral disease, such as softening, tubercular meningitis, and tumours. In some cases the cataleptic condition appears to be premonitory of true epileptic attacks. Some cases, however, cannot be traced to either of these causes, and then catalepsy may be called *idiopathic* or *essential;* cases of the latter form are observed in families who inherit a decided neurotic disposition. The disease is most frequently observed about the age of puberty, but it has been met with as early as five years of age, and occasionally in advanced age. The two sexes appear to be equally liable to the idiopathic variety, but the hysterical is almost exclusively observed in the female. The exciting causes of idiopathic catalepsy are gastric and intestinal irritation, and great emotional disturbance. Malarial infection is said to have caused typical attacks of catalepsy.

§ 946. *Symptoms.*—The cataleptic attack is sometimes preceded by premonitory symptoms, such as headache, vertigo,

trembling of individual muscles, and an undefined sense of discomfort. As a rule, the attack begins abruptly; the movements of the patient are suddenly arrested, it may be while he is speaking or performing some action; the face becomes deadly pale; the respirations are slow and tranquil; the pulse is soft; and, although consciousness is lost, the attitude of the patient at the time of the seizure is retained. The muscles in action at the beginning of the attack appear to be the first to become rigid, but the spasm rapidly extends, as a rule, to all the voluntary muscles, although occasionally it is partial or unilateral. The affected muscles feel firm, and offer resistance to passive movements of the limbs; when once this resistance is overcome, the limbs, head, and neck, or features may be placed in constrained positions, which they retain for a comparatively long period. After the first resistance of the muscles has been overcome the limbs possess a flexibility and pliability, which has been compared to that of soft wax. It has consequently been named *flexibilitas cerea.* This condition of the muscles enables the limbs to be moulded in any position compatible with the rigidity of bones and inextensibility of ligaments, and the constrained attitudes in which the limbs may be placed are maintained without change during the whole course of the attack. "I was shown," says Dr. Wilks, "a man in Morningside whom they could mould in any position. Whilst in bed on his back they could arrange his arms and legs in any posture and there his limbs would remain. Dr. Savage has a case in Bethlem of a young man who will keep his arm stretched out for two hours, and stand on one leg for a great length of time. If made to follow another patient, he will continue to do so until he is stopped." But even during the cataleptic condition the muscular stiffness does not persist in its full intensity for a lengthened period. After some minutes the stiffness diminishes somewhat, so that the arm, for instance, when raised horizontally falls lower by its own weight, and the limb undergoes a slight trembling, indicating the approaching exhaustion of the muscles.

Consciousness is usually abolished, but not in all cases. A certain amount of consciousness may be retained in the early stage of the attack or be present throughout, so that strong

peripheral irritation may cause pain which will be remembered by the patient. Reflex irritability is sometimes lost; at other times certain reflex actions, such as closure of the eyelids on touching the conjunctiva, are retained. The electric contractility of the muscles remains; and, according to the observations of Benedikt, the galvanic irritability of the nerves is increased during the attack, but becomes rapidly diminished during the intervals. In one case observed by Rosenthal the electrical reaction of the nerves to both currents was perfectly normal; in another it was increased.

The organic functions are not usually seriously interfered with. The respiration may be normal, but is generally slow and shallow. The pulse is slow, soft, and compressible. The temperature is generally lowered, and at times the surface of the body becomes icy cold. When the surface of the body is cold, and the pulse at the wrist and respiration are almost imperceptible, the condition may be mistaken for real death.

The attack of catalepsy is sometimes very brief, lasting only a few minutes, at other times several hours or days. Attacks described as being very protracted are in reality made up of a succession of these, separated from one another by intervals in which the patients recover either wholly or partially. The seizures sometimes disappear quite suddenly, and the patients at once recover full consciousness and immediately resume the actions which had been interrupted. As a rule, however, recovery is gradual, patients at first being stupefied as if awaking from a profound sleep, a certain amount of muscular stiffness remaining for some time, which renders motion difficult and slow.

In simple catalepsy no mental disorder is observed in the intervals between the attacks, but when it is merely a symptom of profound nervous disease the intervals may be characterised by the occurrence of hysterical convulsions, delirium, maniacal attacks, and hallucinations, or the catalepsy may be associated with ecstasy and somnambulism.

§ 947. *Course.*—The course of catalepsy is usually chronic, extending over many years. Some individuals suffer only from a small number of attacks separated by intervals of many years.

Others, again, have frequent periodical attacks. In hysterical catalepsy the slightest external influence may suffice to provoke a paroxysm. Catalepsy of malarial origin follows the regular course of other malarial neuroses; they are sometimes accompanied by fever and sweating, and generally yield to the usual antiperiodic remedies. Cases caused by sudden fright or injury may also run an acute course, the disease terminating after a single attack or after a series of them. Cataleptic attacks are hardly ever fatal of themselves.

(II.) TRANCE.

§ 948. In this condition the patient lies for days together in an apparently insensible condition without eating or drinking. The state of complete insensibility is not, however, continuous, inasmuch as there occur periods during which the patient notices those around her, and may partake of small quantities of food. The condition of the patient is not, indeed, unlike that of a hibernating animal. In the state of trance the patient usually lies in a warm room, well covered with clothing, so that little heat is lost by radiation; the mental functions are in abeyance, indicating that the molecular changes which are the correlatives of mental actions have ceased; and all muscular movements are suspended with the exception of the cardiac contractions, and slight respiratory movements. Under such circumstances the amount of waste must be small. If Dr. Tanner (and there are no grounds for believing that any deception was practised by him), with all the waste implied by the possession of active mental faculties, outbursts of temper, walking and driving in the open air, could live forty days without food, it may be inferred that persons in the state of trance might live fasting for a much longer period. The physician must, of course, be on his guard against deception in cases of trance.

(III.) ECSTASY.

§ 949. This condition is closely allied to trance, the patient being insensible to outward impressions in both. In ecstasy the mind is absorbed with some fixed idea, generally of a

religious character, and the patient becomes oblivious of surrounding events and objects. The limbs are motionless, and often fixed in maintaining a particular attitude; the breathing is slow and feeble; the pulse is almost imperceptible; the eyes are often bright and animated; and the countenance has an expression of rapture (Maudsley).

(IV.) SOMNAMBULISM AND HYPNOTISM.

§ 950. In *somnambulism* the patients appear to be wholly unconscious, yet they walk, climb, and avoid obstacles, and may manifest greater strength, agility, and precision of muscular adjustments than during waking hours.

Hypnotism or *mesmerism* is, as Maudsley remarks, a kind of artificially induced somnambulism. The subject, who is probably always of a neurotic temperament, is induced to look steadily at the operator, the latter attracting his attention by making a few gentle "passes" with his hand. Mr. Braid, of Manchester, directed the person to look upon a disc or some bright object held in front of and a little above the level of the eyes. After a short time there is a slight tremor of the eyeballs of the subject, his pupils dilate, and he falls into the mesmeric condition. In this state the mental functions are abolished, and all the actions of the subject are afterwards determined by the suggestions of the operator. Under the influence of these suggestions the subject may sing, recite poetry, and perform the most absurd and outrageous actions. He may be made to eat a raw cabbage amidst all the outward signs of enjoyment to appease a suggested hunger; he may spit out pure water given him to drink with all the signs of disgust, on the suggestion that it is bitter and nauseous, or drink infusion of wormwood with apparent relish on being told that it is an agreeable beverage; or he may be made to sneeze violently on being asked to take a pinch of snuff from an empty box. Hysterical patients may be thrown into a condition of trance or of catalepsy, or one half of the body may be thrown into trance and the other half into catalepsy, by being made to look upon a bright light (Charcot). A condition much resembling the hypnotic state is sometimes induced by disease. A curious case of

this kind is transcribed by Dr. Wilks from *Galignani,* and as the case is a remarkable one in many ways I quote it at length.

A Living Automaton.—A curious patient is just now an inmate of Dr. Mesnet's ward at the Hôpital St. Antoine. His profession was that of a singer at the Cafés Chantants. During the war of 1870-71 he was hit over the left ear by a musket bullet, which carried off about 2½ inches of the parietal bone, and laid bare the brain on the left side. This led to a temporary paralysis of the members on the opposite side, as is always the case; but he was eventually cured of this, while the tremendous wound on the skull began to heal, so that after a time he could resume his professional duties at the cafés to the satisfaction of the public. Suddenly, however, he was seized with nervous symptoms, lasting from 24 to 48 hours, and of such an extraordinary nature that it was considered safe to take him to the hospital. His malady is easier to illustrate by examples than to define. When he is in his fit he has no sensitiveness of his own, and will bear physical pain without being aware of it; but his will may be influenced by contact with exterior objects. Set him on his feet, and, as soon as they touch the ground, they awaken in him the desire of walking; he then marches straight on quite steadily, with fixed eyes, without saying a word, or knowing what is going on about him. If he meets with an obstacle on his way he will touch it, and try to make out by feeling what it is, and then attempt to get out of its way. If several persons join hands and form a ring around him, he will try to find an opening by repeatedly crossing over from one side to the other, and this without betraying the slightest consciousness or impatience. Put a pen into his hand; this will instantly awaken in him the desire of writing; he will fumble about for ink and paper, and, if these be placed before him, he will write a very sensible business letter; but, when the fit is over, he will recollect nothing at all about it. Give him some cigarette paper, and he will instantly take out his tobacco bag, roll a cigarette very cleverly, and light it with a match from his own box. Put them out one after another, he will try from first to last to get a light, and put up in the end with his ill-success. But ignite a match yourself, and give it him, he will not use it, and let it burn between his fingers. Fill his tobacco bag with anything, no matter what—shavings, cotton, lint, hay, &c.—he will roll his cigarette just the same, light and smoke it without perceiving the hoax. But, better still, put a pair of gloves into his hand, and he will put them on at once; this, reminding him of his profession, will make him look for his music. A roll of paper is then given to him, upon which he assumes the attitude of a singer before the public, and warbles some piece of his repertory. If you place yourself before him he will feel about on your person, and, meeting with your watch, he will transfer it from your pocket to his own; but, on the other hand, he will allow you, without any resistance or impatience whatever, to take it back again.

§ 951. *Morbid Anatomy and Physiology.*—Post-mortem examinations have only revealed changes in those cases in which catalepsy and its allies are mere symptoms of grave organic disease. Schwartz reports the case of a boy who, after an injury, suffered at first from an affection resembling chorea, and later cataleptoid attacks, and who, after two years, died from anæmia and marasmus. The autopsy revealed serous effusion in the arachnoid, softening of the corpus striatum and optic thalamus, especially of the left side; and a brownish-red, jelly-like mass, covering the spinal dura mater along the posterior surface of the cord from the cervical to the lumbar enlargement. Meissner examined a man, 47 years of age, who suffered from catalepsy for six years, and in the three last years of his life from maniacal and epileptic symptoms, with paralysis of the right side; he found an epithelioma growing from the dura mater in the anterior fossa over the ethmoid bone; the anterior third of the right cerebral hemisphere, as well as the right corpus striatum, was much softened. Lasègue found no change in the brains of two men affected with catalepsy examined by him.

The information obtained from post-mortem examination is as yet far too scanty to throw much light on the pathology of the disease. One noteworthy fact in connection with the slighter forms of cataleptic attacks is their similarity to some cases of the *petit mal* of epilepsy. It is a somewhat significant fact that in Meissner's case of catalepsy the disease was situated in the præfrontal region of the cerebral hemispheres. A functional or organic lesion of the cortex of the cerebrum might no doubt account for the sudden loss of consciousness, but the most characteristic features of catalepsy—the muscular rigidity and flexibilitas cerea—are still unexplained. Most authors believe that the cataleptic rigidity is only an increase of the normal tonus of the voluntary muscles, and some think that the diminution or loss of voluntary innervation which occurs in catalepsy causes an increase in the reflex tonus of the muscles, just as reflex excitability is increased by the removal of the cerebral hemispheres in frogs. But no amount of increase of the reflex tonus would account for the condition known as flexibilitas cerea, and what is a still more fatal objection to this

theory is that general reflex irritability, instead of being increased in catalepsy, is on the contrary often much diminished or abolished.

We have already seen that complete hemianæsthesia is probably caused by a temporary or permanent arrest of the functions of the centripetal fibres in their ascent through the internal capsule, or of that part of the cortex of the brain which is supplied by the posterior cerebral artery. Suppose now that a complete bilateral hemianæsthesia exists, what would be the condition of the patient? There would be complete loss of every form of cutaneous and muscular sensibility as well as of sensation in the bones and joints; there would be loss of taste on both sides of the tongue, and of smell in both nostrils; and instead of there being amblyopia and partial deafness on one side, as in hemianæsthesia, there would be complete blindness and deafness on both sides, inasmuch as, according to the hypothesis, the sensory centres in both hemispheres either have ceased to act or the impressions made on the peripheral sense organs fail to be conducted to them. But impressions made on the periphery would, however, reach the cortex of the brain through the optic thalamus, and the subject of bilateral hemianæsthesia, although effectually cut off from the external world so far as the anatomical substratum of consciousness is concerned, would perform various complicated actions in response to peripheral impressions, but without being attended by consciousness. The condition would, indeed, be very similar to that observed in somnambulism, the mesmeric state, and various post-epileptic and allied conditions.

§ 952. *Diagnosis.*—During the presence of muscular stiffening and flexibilitas cerea, the diagnosis can present no difficulties. Only very cleverly executed simulation could give rise to any doubt; and in these cases careful testing of the sensibility, reflex irritability, and electrical reactions, along with comprehensive observation of the concomitant symptoms, ought to be sufficient to afford a safeguard against deception.

§ 953. *Prognosis.*—The prognosis of uncomplicated cases of catalepsy is always favourable as regards life; but with respect

to complete recovery it is unfavourable. Cases arising from malarial infection afford a better prognosis, and the same may be said with respect to acute attacks occurring in comparatively healthy persons as the result of injuries and mental shocks.

§ 954. *Treatment.*—The aims of treatment in catalepsy are to arrest the attacks and to prevent their return. The latter indication can be best effected when the cause can be removed; in catalepsy due to malaria, quinine alone, or combined with morphia, may effect a complete cure. Favourable results have been obtained by the use of tonics, iron, ergot, morphia, the cold douche, and the faradic current. The galvanic current has hitherto proved useless.

Attempts to put an end to the attack itself have not been very successful. A slight peripheral irritation may arouse patients in hysterical catalepsy, but in the idiopathic form very strong cutaneous irritation has often no influence. In protracted cases artificial feeding by the stomach pump and nutritious enemata must be had recourse to.

Trance must be treated on the same general principles as hysteria, while those who are liable to attacks of somnambulism ought to have their bedroom windows and doors well fastened at night.

CHAPTER VI.

EPILEPSY AND ECLAMPSIA.

(I.) EPILEPSY.

EPILEPSY is a chronic functional disease of the nervous system, characterised by recurring paroxysms of impairment or loss of consciousness, accompanied generally by partial or general convulsions.

§ 955. *Etiology.*—Hereditary predisposition plays an important part in the production of epilepsy. The transmission may be direct, as when the progeny of an epileptic parent are affected; or indirect, when one or more generations escape, and the disease reappears in the descendants. But in other cases the hereditary tendency is still more indirect. The family of the patient may have a proclivity to nervous disease, declaring itself as insanity in one member, as hysteria in a second, indulgence in alcoholic excess in a third, neuralgia in a fourth, and epilepsy in a fifth, while other members may only exhibit the slighter forms of instability termed nervousness. A hereditary taint may be traced in rather more than one-third of all cases (Reynolds, Gowers). The children of consanguineous marriages appear to suffer from epilepsy in greater proportion than other children. All family peculiarities, whether good or bad, are intensified in the children by intermarriage; and when both parents inherit an unstable nervous system the probability of some nervous disorder appearing in the progeny is greatly increased. Under these circumstances one or more of the children may suffer from epileptic convulsions when the parents manifest only nervousness, neuralgia, or hysteria. The

disease is transmitted more frequently through the mother than the father, and the females of a family are more likely to suffer than the males (Gowers).

The female is rather more frequently affected with epilepsy than the male sex, in the proportion of 114 to 100 according to Dr. Gowers. Females are attacked with hystero-epilepsy in the proportion of 2 to 1 of males.

Age is an important predisposing cause of epilepsy. Out of 1,450 cases analysed by Dr. Gowers the disease began under ten years of age in 29 per cent, between ten and twenty in 46 per cent, between twenty and thirty in 15·7 per cent, between thirty and forty in 6 per cent, between forty and fifty in 2 per cent, between fifty and sixty in 1 per cent, and above sixty years of age in ⅓rd per cent. The proportion in which the two sexes suffer varies at different periods of life. Speaking broadly, of the number attacked under thirty years of age the males exceed the females, while beyond the proportion is reversed. It has been asserted that when a hereditary tendency exists the disease begins before twenty years of age, but Dr. Gowers maintains that the influence continues until a late period of life.

Defective nutrition of the body generally, including the nervous system, such as is met with in anæmia, chlorosis, scrofula, and rickets, appears to beget a certain instability of the nervous system which predisposes to the production of epilepsy. Chronic alcoholism is also often associated with epilepsy, but it is difficult to determine whether the former is the cause of the latter, or both result from a hereditary taint.

Of the *exciting* causes of epilepsy, profound emotional disturbance, arising from fright, grief, and anxiety, is the most frequent and important. The first attack of epilepsy often appears after a sudden fright. This cause is most frequently in operation under twenty years of age, and the female sex is especially liable to be affected by it. Epilepsy is sometimes excited in apparently healthy individuals by witnessing another in convulsion. Eccentric irritation is another frequent exciting cause of epilepsy. Of the cases which date their origin from infancy, the first fits occur in a large proportion during dentition. It must be remembered that children who have suffered from eclamptic

attacks during dentition frequently develop true epilepsy at puberty, but it is probable that there exists in such cases a strong predisposition to the disease.

The influence of sexual excess in the production of epilepsy is probably over-estimated. Women, the subjects of epilepsy, not unfrequently suffer from a fit during the menstrual period, and the disease may be caused by uterine and ovarian derangements and by pregnancy. Among other reputed causes of epilepsy may be mentioned diarrhœa, dysentery, overloading the stomach, irritation of the intestinal canal from the presence of worms, the passage of gall stones, over-exertion, and exposure to cold.

Epilepsy is also liable to become established in the course of or during convalescence from acute febrile diseases, a considerable proportion of such cases following scarlet fever. In these a connection between the eclampsia of scarlatinal nephritis and the subsequent recurrence of convulsions can only occasionally be traced (Gowers). Chronic lead-poisoning is sometimes attended by recurring convulsions like those of epilepsy.

Injuries to the head and sunstroke are frequently followed by epilepsy. Cases in which the skull is fractured or coarse structural changes are set up in the brain are at present excluded from consideration. Injuries to nerve trunks are liable to be followed by epilepsy, and the first seizure usually occurs weeks, months, or even years after the injury. The convulsion is generally preceded by some peculiar sensation proceeding from the region of the affected nerve.

§ 956. *Symptoms.*—The symptoms of epilepsy may be divided into (1) those which precede the paroxysm, (2) those occurring during the paroxysm, and (3) those observed in the intervals between the attacks.

(1) *Premonitory Symptoms.*—The premonitory symptoms of the epileptic attack may be subdivided into remote and immediate warnings, the latter forming the *auræ epilepticæ*. The remote warnings may extend over hours or days before the occurrence of the attack; they usually consist of such symptoms as headache, dizziness, confusion of thought, or some mental change, the patient becoming depressed and morose, or

excited, lively, and irritable. In a case recently under my care the patient stated that for hours before an attack he became sometimes very much depressed in spirits and suspicious that his friends were speaking and plotting against him, while at other times he was unaccountably joyous. He then volunteered the statement: "I am expecting an attack to-day, for instance, I feel so happy and joyous, and there is nothing in my circumstances to make me so, as I have just lost my situation through these fits." During examination his right cheek was suffused with a bright red blush; while the left one was, as his face had hitherto always been when I saw him, remarkably pale. If the cortex of the hemispheres of the brain were as freely supplied with blood as the right cheek, this might account for his joyous feelings.

Aurœ Epilepticœ.—Our information with regard to the immediate warning of the epileptic paroxysm consists of the subsequent account which the patient is able to give of his feelings before loss of consciousness is complete. In more theoretical language, the aura is the mental correlative of the commencing molecular change in the brain, which is the physical cause of the epileptic attack. Consciousness is abolished so soon that there is no aura described in about half the cases.

The aura may be (*a*) motor, (*b*) sensory, (*c*) vaso-motor and secretory, or (*d*) psychical.

(*a*) *Motor Aurœ.*—It is not always easy to distinguish between a motor and a sensory aura. In epileptic seizures, where consciousness is retained throughout the attack, or lost only at a comparatively late period, the patient is often able to describe the convulsions of the limbs in objective language. He may be able to tell that he first felt the thumb dragged across the palm and the fingers flexed on the thumb, and that he had then to hold the convulsed hand with the other one in order to arrest its movements.

A patient of my own was in the habit of acting before the students with great fidelity the phenomena of the attack. He first produced twitching of the muscles of the angle of the mouth, then rotated his head and eyes, and finally flexed his fingers, and shook his arm to show how the convulsions had

invaded the upper extremity. The attack in this case was due to coarse lesion of the cortex syphilitic in origin, and consciousness was only partially suspended towards the termination of the attack. In idiopathic epilepsy, on the other hand, consciousness becomes confused at such an early period that the patient is unable to describe his feelings in objective language, although he may still do so in subjective language. When, for instance, the convulsion begins in the hand, the patient, instead of describing the thumb as being drawn into the palm, says that he feels a dragging sensation in the thumb or a feeling of creeping, or numbness in the hand, which gradually passes up the arm. The sensation begins sometimes in the muscles of the shoulder and passes down the arm; but these cases are probably always associated with a coarse lesion in the brain. When the convulsion begins in the lower extremity, the aura generally begins as a creeping sensation in the big toe, which passes up the leg and may extend to the arm before unconsciousness supervenes.

The motor aura begins not unfrequently in the side of the face, and is generally described as a feeling of "the face being drawn," or it may begin in the side of the tongue, and be described as a feeling of something crawling. The tongue is associated in its actions with different sets of muscles, according as it is engaged in articulation, mastication, or deglutition, and these associated movements appear to be sometimes dimly represented by the epileptic aura. In a case mentioned by Dr. Gowers tingling in the tongue was associated with twitching of the lips, in another with a sensation of lateral movements of the jaw, and in a third it was followed by a feeling of sickness, succeeded by a sensation of something rising in the throat and then by palpitation of the heart.

The aura consists sometimes of sudden inability to speak—a temporary aphasia,—or there may be motor auræ referred to the eyeballs, which are generally described in subjective language. The patient, for instance, never says that the attack began by a squint, but states that he suffered from double vision. On rare occasions the patient may describe that his eyes were turned in a particular direction; but, as a rule, the rotation of the eyeballs is felt by the patient as a displacement

of external objects in the opposite direction, and he consequently complains of things whirling round him, of swimming in the head, or of vertigo and sickness. At times patients see objects recede from them and become smaller or approach them and become larger, sensations probably depending upon variations in the tension of the muscles of accommodation. One of the most remarkable features presented by motor auræ is that, as a rule, all of them begin in small muscles, such as those of the eyeballs, tongue, face, and hands. These muscles are engaged in the most special actions, and consequently the motor auræ may be said to begin in the more special and to pass gradually to the more general actions. Sometimes the aura begins in muscles like those of the shoulder engaged in general actions, while the muscles engaged in special actions are secondarily involved. Such cases are probably always due to organic disease of the brain, the lesion being situated near the longitudinal fissure and away from the special centres of the operculum. Sometimes the aura may occur in both hands or both legs simultaneously, or there may be a sensation of trembling in the muscles of the back, while at other times general tremor or jerking of the muscles is complained of.

(*b*) *Sensory Auræ.*—The sensory auræ may be referred to any of the centripetal nerves of the body, namely, those of the skin, muscles, and bones, the nerves of special sense, and the nerves of the viscera. As already remarked, it is not always easy to determine whether the sensations referred by the patients to the extremities are due in any particular case to a discharge from a sensory or a motor centre in connection with the part. Dr. Gowers remarks that there are two modes in which the aura extends from the arm to the leg, the one by continuity, the sensation passing up the arm and down the trunk to the leg; and the other by separate commencement in the muscles of the lower extremity. The same is also true with regard to the method of invasion from the lower to the upper extremity. In the continuous method of invasion, Dr. Gowers believes that the discharge from the sensory centre takes the lead, while in the discontinuous mode of invasion the discharge of the motor centre is primary. The sensory aura sometimes consists of a feeling of general heat or cold, and at other times

is distinctly localised. Besides various sensations, as tingling, numbness, or pain in the extremities, it may consist of headache, frontal, vertical, or unilateral, or a feeling of pressure in the head (Gowers).

Aurœ of the Special Senses.—The auræ of the special senses may consist of undeveloped sensations, which Hughlings-Jackson, following Spencer, has named *crude* sensations, or of more elaborate sensory representations. During the former the discharge is limited to a sensory centre, so that a simple sensation only is revived; during the latter the molecular disturbance extends to neighbouring sensory centres, so that complicated perceptions result.

Visual Aurœ.—The aura sometimes consists of sparks, flashes of light, and coloured vision. In some cases the fit begins by a coloured vision, followed by spectral faces, the former being a crude sensation, and the latter revived perceptions. Illusions of sight are not uncommonly observed. In a case of my own the attack is ushered in by the patient finding himself surrounded by lions and tigers, ready to spring upon him. In some cases the fit is preceded by a vision of beautiful places, but disagreeable objects and scenes are more common. In many cases the attack is ushered in by sudden blindness of one or both eyes.

Auditory aurœ may consist at times of hissing, ringing, or explosive noises; at other times voices are heard, or the patient hears distinct words and sentences. The patient may hear a noise in the ear at first, and this be followed by a distinct vocal utterance, while in other cases music is heard. At times the attack is ushered in by a sensation of unusual stillness.

Gustatory aurœ consist of metallic or other abnormal sensations of taste. The alteration in the sense of taste may sometimes be manifested by semi-voluntary or automatic movements. In a case mentioned by Dr. Hughlings-Jackson the patient "put different substances into his mouth; at the seaside, sand; at school he drank ink; in the country he bit pieces of butterflies; he smacked his lips in the attacks, and his brother said he had a look of disgust."

Olfactory aurœ consist, as a rule, of unpleasant smells, generally described in subjective language, and only occasionally

in objective language. Dr. Hughlings-Jackson mentions the case of a patient who held his nose when the attack began; probably in this case there was an objective sensation of smell.

In glancing over the different sensory auræ, the most remarkable characteristic presented is that a general sensation, as a red colour, often precedes the appearance of the more special sensations, such as are afforded by definite images of persons and things. With the motor aura we saw that, as a rule, the special precedes the general; but it would appear that in the sensory auræ the general precedes the special.

These facts find their interpretation in inferences already made from embryological considerations; namely, that the special movements are represented in the motor area of the cortex of the brain by comparatively small and young cells situated in the neighbourhood of the vessels; while the special sensations are represented in the sensory area of the cortex by comparatively large and old cells thrust away from the vessels by the growth of young cells, the latter of which represent the general sensations.

Visceral Aurœ.—The most common of all the immediate warnings of epilepsy are sensations referred to different portions of the area of distribution of the pneumogastric nerve. Of these a sensation referred to the pit of the stomach, named the *epigastric aura,* is by far the most frequent. It is sometimes described as pain, at other times as a feeling of "coldness," "burning," or as a vague, indescribable sensation. The painful epigastric feeling is generally fixed; while the vague sensation ascends to the throat and causes a feeling of choking, or to the head, when immediate loss of consciousness ensues (Gowers). In some cases a feeling of suffocation constitutes the warning; in others there may be nausea and retching, a feeling of intense hunger, or palpitation with or without angina.

(*c*) *Vaso-motor and Secretory Aurœ.*—The attacks may be ushered in by a sensation of coldness, or numbness in the fingers and toes, and the affected part becomes pale and cold to the touch. At other times spots in various parts of the body assume a red colour, and become the seats of feelings of transient heat. Faintness, which constitutes a frequent aura, may probably sometimes be associated with sudden dilatation of the abdominal vessels.

(*d*) *Psychical Aurœ.*—The immediate warning often consists of intense horror or alarm, and the patient may look startled, guilty, or frightened. In other cases it consists of an intellectual perversion. The patient feels as if he were suddenly in a strange country, or the idea suddenly seizes him that an object, at which he may be looking and which he has never seen before, was known to him previously. In many cases the aura is what Dr. Hughlings-Jackson has named "a dreamy state" or a "voluminous" mental condition. This condition is similar to that said to be experienced by persons in the act of drowning, during which all the past events of life seem to crowd upon the memory.

Order of Succession of the Phenomena of the Aura.—This subject is so wide that it can only be briefly indicated here. We have already seen that when the aura is motor, and the spasm begins in the muscles of the head, those of the arm are next invaded and those of the leg last; when the spasm begins in the muscles of the hand those of the face are next invaded and those of the leg last, and when the spasm begins in the muscles of the leg those of the arm are next invaded and those of the face last. The order in which the convulsions spread in these three forms of onset may be readily explained by reference to the topographical distribution of the motor centres in the cortex.

An undeveloped or crude sensation of sight, such as a red light, is sometimes followed by a developed perception, such as the image of a man; a confused noise may be succeeded by strains of music; a subjective sensation of smell may be followed by efforts to prevent the effluvia gaining admission to the olfactory chambers, showing that the sensation had become objective. It would appear that in all these cases the discharge begins in the sensory centre, and as it spreads it involves a portion of the cortex, in which connections are formed with other sensory centres, so that the discharge is now accompanied by a perception. This process is, however, soon arrested by the rapid impairment of consciousness, during which both sensations and perceptions become fainter and soon disappear. A visual is not unfrequently associated with an auditory aura, sometimes the one and sometimes the other taking the pre-

cedence. The association of these sensations is readily explained by the proximity of the visual and auditory centres as determined by Ferrier. In some cases the epigastric aura is followed by an emotion of fear or of anguish, and the patient may have a facial expression corresponding to it. It has been suggested by Hughlings-Jackson that there may often be a determinate relation between sensory and visceral auræ, and between the "dreamy" state of psychical auræ, and post-epileptic actions; but little or no progress has hitherto been made in collecting materials to decide the question.

(2) *The Epileptic Paroxysm.* — The symptoms of the paroxysm are very variable, but for purposes of description they may be divided into (*a*) *epilepsia mitior*, or *le petit mal*, in which there is impairment or abolition of consciousness, but no manifest spasms; (*b*) *epilepsia gravior*, or *le haut mal*, in which there is loss of consciousness along with general tonic and clonic convulsions; (*c*) *epileptiform* seizures, in which pronounced spasms are present in half the body, but with only slight suspension of consciousness.

(*a*) *Epilepsia mitior, or le petit mal.*—The description of the minor attacks of epilepsy need not detain us long. The attack consists of momentary confusion of thought, or transitory unconsciousness. The patient, for instance, may be attacked while speaking; he becomes suddenly unconscious, there is a pause probably in the middle of a sentence, but in a few seconds speech is resumed at the point where it was interrupted and the sentence is finished. At times the attack may consist of a feeling of fainting, along with confusion of mind. In other cases there is momentary vertigo, slight pallor of the face, and transitory unconsciousness. Indeed, any one of the numerous auræ just described may, along with slight confusion of mind, constitute a minor attack of epilepsy. Many of the sensations described as immediate warnings may be experienced in the absence of epilepsy; but if the sensation recur at periodical intervals, and be attended by some confusion of thought, the occurrence of genuine epilepsy may be suspected, although a visual sensation, along with some confusion of thought, often precedes an attack of migraine. If there be

involuntary discharge of urine or fæces during the attack the affection is undoubtedly epileptic.

In many of these slighter attacks loss of consciousness is accompanied by minor degrees of muscular spasm. At the onset of the attacks the countenance becomes ghastly pale, the pupils contracted, and the eyes fixed and staring, or there may be slight strabismus or drawing of the mouth, while in other cases there may be partial rotation of the head and eyes, chewing movements, or rolling about of the tongue. There may be again momentary rigidity or slight tremor of all the muscles of the body, while transitory arrest of respiration is not uncommon. Sometimes the patient utters a shriek, reels, or walks hurriedly round the room, and then recovers. At times the unconsciousness may last for a considerable period, but the patient may go on with the work in which he was engaged as if he were conscious. In a case mentioned by Trousseau the patient continued to play the violin with accuracy during short periods of unconsciousnesss. It must be remembered that the slighter attacks of epilepsy are liable to be followed by the condition named "epileptic mania," to be subsequently described.

(*b*) *Epilepsia gravior, or le haut mal.*—The epileptic paroxysm may, for the purposes of description, be divided into three stages. The *first* is characterised by loss of consciousness with *tonic* spasm; the *second,* by loss of consciousness with *clonic* spasm; and the *third,* by cessation of the spasm and gradual restoration to consciousness. A *fourth* or *after* stage may be added.

(i.) *The First Stage.*—The true epileptic attack is ushered in by three prominent symptoms occurring simultaneously. These are loss of consciousness, sudden falling, and great pallor of the face, while a fourth symptom is often present in the form of a loud and piercing cry. The loss of consciousness is sudden and complete, every form of sensibility and mental operation being completely abolished, although certain reflex actions are retained (Romberg). The patient often falls, as if struck by lightning, either forwards on his face, backwards on his occiput, or latterly, and so instantaneously that he has no time to select a place or attitude, and may consequently fall into fire, water, or from a height. At other times the patient

has sufficient warning of the impending attack to enable him to sit or lie down. Pallor of the face is probably always present at the beginning of the attack, although the symptom is sometimes so transient that it may pass unobserved.

The epileptic cry which the patient often utters immediately before or during the fall is loud and piercing, and alarms, according to Romberg, both man and animals. When the patient falls to the ground he remains for a period of from two to forty seconds in a rigid condition, caused by a tonic, although unequal contraction, of all the muscles of the body. Various distortions are thus produced; there is conjugate deviation of the eyes, with rotation of the head and neck; the pupils are dilated and insensible to light; the countenance is variously altered; the jaws are firmly closed, and the tongue may be severely bitten; there is opisthotonos; and the different segments of the lower extremities are extended upon one another and upon the trunk, the foot being rotated inwards and the toes widely separated; the segments of the upper extremities again are flexed upon one another, the thumb being bent into the palm, the fingers closed, and the hand pronated; and the forearm is flexed or sometimes extended upon the arm. The respiratory muscles are in a state of tonic spasm, and the breathing is arrested. The hideous cry uttered as the patient falls is probably produced by spasm of the expiratory muscles with closed glottis. The pallor of the face is soon replaced by a dull red or dusky hue, and the veins of the head and neck become greatly distended, the carotids throb violently, and the action of the heart is forcible, although the pulse is small or imperceptible at the wrist.

(ii.) *The Second Period.*—After a variable period of from two to forty seconds, the tonic gives place to clonic spasms, which are usually more pronounced on one side of the body. The muscles of the face, tongue, pharynx, and larynx are usually first affected by clonic spasm, and those of the trunk and extremities are afterwards invaded.

The patient now presents a hideous appearance, the head is alternately drawn laterally, or forwards and backwards; the eyeballs are convulsively rotated in various directions, but rotation in an upward and outward direction predominates, so

that the pupils are hidden, and only the whites of the eyes are visible under the blinking half-closed lids; the face is variously distorted, and the convulsive closure of the jaws is often so violent that teeth are broken and the tongue severely bitten, while the blood from the wound, mixing with the saliva, oozes through the clenched teeth as a sanguineous froth. The trunk and limbs are variously thrown about, and the contents of the bladder, rectum, or vesiculæ seminales may be evacuated.

The venous hyperæmia reaches its maximum just as the clonic spasms are beginning to abate in severity; and the skin is bathed in sweat, which in some cases has a fœtid odour. The heart beats tumultuously; the carotids throb; and the pulse, if it can be felt, is fuller and more laboured than during the period of tetanic contractions. The pupils are alternately contracted and dilated, and are said to be slightly sensible to light. This stage may last from a few seconds to five or ten minutes, the average duration being from two to three minutes.

(iii.) *The Third Stage.*—During the third stage there is a gradual return to consciousness and voluntary power. The convulsions either cease suddenly or wear off gradually, the period of transition being marked by partial jerkings of some muscular groups, or by a diffused tremor of the body.

General muscular relaxation is now established, but coma persists for a short time longer. The patient soon attempts to change his position; he opens his eyes and looks around him with a bewildered expression, and perhaps attempts to speak.

The respiratory movements have become more natural in rhythm, although they are still somewhat irregular; the pupils are contracted; the pulse is variable, but generally full and quieter than during the previous stage; the conjunctivæ are injected; petechiæ are often observed on the eyelids, forehead, and temples; and the patient is exhausted and disposed to sleep.

The attack is often followed by vomiting, and a large quantity of pale urine is often passed. The temperature of the body appears to be normal after single attacks of epilepsy.

(iv.) *The Fourth or After-Stage.*—The after-symptoms of epilepsy differ greatly in duration, severity, and nature. The

patient recovers occasionally in about a quarter of an hour after the attack, and resumes his previous occupation; but, as a rule, recovery is delayed for a much longer period. He suffers from lassitude and stupor, from which he is aroused with difficulty, and, if awakened, he is peevish and irritable, while the general muscular relaxation is occasionally interrupted by momentary clonic spasms or fibrillary contractions. The average duration of the stupor is about an hour when the attack occurs during the day; but when it occurs in the evening it passes insensibly into the ordinary nocturnal sleep.

Complications.—Various mental disturbances are by far the most important of the complications of epilepsy. The patient sometimes exhibits marked mental derangement immediately before as well as after the paroxysm, and a maniacal condition constitutes sometimes the principal feature of the attack. A person the subject of epileptic vertigo may continue for some seconds, minutes, or even hours in a dull, half stupid condition. He may mutter a few incoherent words, or some lewd expression, no matter how foreign to his habit; he may unbutton his clothes and expose his person, urinate in a public assembly, exhibit himself naked to his domestics, or even walk in public naked unless prevented, and on recovery he has only the vaguest recollection of what has occurred. These, however, are only a few of the minor actions which may be done by individuals subject to epileptic vertigo immediately after the attack. The most motiveless and atrocious crimes are sometimes committed in this condition, so that some medical jurists are of opinion that no epileptic is responsible for his actions. Epileptics sometimes have a warning of the approaching maniacal state, so that they can warn their friends to protect themselves; but generally the seizure is more or less sudden. There is every variety of intermediate form between the milder and severer cases. Reynolds says that *epileptic mania* occurs in about one-tenth of all cases of epilepsy, including the minor attacks of epileptic vertigo; having occurred in the case of any one individual, it is apt to appear again, especially when several fits have followed in rapid succession.

Epileptic delirium is not always furious and dangerous. It may appear in the form of preternatural gaiety or illusions

of the senses before the attack, or during the intervals. Meningitis has been known to follow epileptic paroxysms; but it is generally the result of an injury inflicted by the fall, and both apoplexy and permanent paralysis are rare complications of idiopathic epilepsy. Idiocy and epilepsy are not unfrequently associated, in which case the former disease is generally congenital.

Attacks of Hystero-epilepsy.—Hystero-epileptic attacks are not often seen in this country in the classical form described by French authors. The patient in the intervals suffers from various hysterical symptoms, the most usual of which are complete or incomplete hysterical hemianæsthesia, and ovarian hyperæsthesia. The paroxysm is always preceded by an aura, consisting of a sensation proceeding from the region of the hyperæsthetic ovary towards the epigastrium, and ascending to the throat and finally to the head, when the patient utters a loud shriek and falls insensible to the ground. All the muscles of the body now become the subjects of tonic spasm; the head is retracted, and the body and limbs are arched backwards and rigid; the respirations are stertorous and infrequent; and foam, sometimes blood-stained on account of the tongue having being bitten, generally issues from the mouth. The tonic stage is followed by a few clonic convulsions, but these soon cease, and a state characterised by general muscular relaxation, stertorous respiration, and coma terminates the portion of the attack resembling the epileptic paroxysm.

The *second* stage, or what the French call the "*phase des grands mouvements*," now makes its appearance. It is characterised by violent contortions of the body, and gesticulations having a purposive character. There is opisthotonos, or the body is bent forwards or laterally; while at other times it is maintained in a rigid position, with the lower extremities extended and the upper stretched out.

The *third* stage, or stage of emotional attitudes *(phase des attitudes passionelles)*, now appears, and during its continuance the patient assumes in rapid succession attitudes and gestures expressive of various emotions. The first attitude assumed by the patient is usually a threatening one; she raises herself in

a half sitting posture, the brows frown, the fists are clenched, and the face presents an angry and menacing expression; this attitude, however, soon gives place to an expression of abject fear, which in its turn makes room for a look of intense happiness and beatitude. But the expression of beatitude is also fleeting in character, and is succeeded by one of intense voluptuousness, followed by gestures which lead to this stage being called the *phase of lubricity*. Terror now seizes the patient, she sees rats and other odious animals that evoke from her passionate exclamations of dread and disgust, and this is followed by a stage in which she appears to be labouring under the idea that she has committed a great offence, and sues for mercy. The patient now hears strains of music, she looks pleased, and may join in humming the tune; but her singing is soon followed by weeping, broken by reproaches addressed to her parents as the cause of her misery. This last phase constitutes the stage of recovery, but hallucinations may persist for some time. The patient can always describe subsequently the hallucinations to which she was subject, and each of the attitudes is found to have corresponded to a hallucination.

An attack of hystero-epilepsy may be provoked at any time by various manipulations, such as "suddenly 'gripping' the skin of the breast on both sides on a level with the fifth rib, and midway between the anterior and posterior boundaries of the axillæ."* An attack is also readily induced by pinching a fold of the skin of the sub-inguinal region, or by slight pressure on the region of the hyperæsthetic ovary. Sudden and firm pressure exerted on the affected ovary, however, instantly arrests the paroxysm at any stage. When such compression of the ovary is made, the patient's mouth opens widely, the tongue is spasmodically protruded, and the convulsions cease.

The phenomena presented by attacks of hystero-epilepsy in this country are too variable to be comprised under one general description. In some cases there is a condition of emprosthotonos, instead of opisthotonos, during the second stage of the attack, this form being very liable to occur in boys. In other cases the legs are alternately flexed and extended, and the

* An Account of the Phenomena of Hystero-Epilepsy, by Arthur Gamgee, M.D., F.R.S., "British Medical Journal," vol. ii., 1878.

patient, who lies on her back, may be propelled from one end of the room to the other. In still other cases the attack consists of great difficulty of breathing caused by intense respiratory spasm (Gowers).

(*c*) *Epileptiform Seizures.*—This form of epilepsy was first described by Bravais, but its pathology was fully investigated by Hughlings-Jackson, and it is consequently named "Jacksonian Epilepsy." In it the convulsions are partial, being limited to one half of the body, and consciousness is either retained throughout the attack, or lost only at a comparatively late period. The fits are accompanied or followed by paresis of the convulsed limbs. These seizures are caused by coarse disease situated near the cortex of the brain, the most common lesion being a gumma. The convulsions which supervene upon the spastic hemiplegia of childhood (unilateral atrophy of the brain) are at first partial, but after a time become general. An interesting case of epilepsy in a girl aged 6½ years is recorded by Dr. Sturge, in which the convulsions, which began when the patient was 6 months old, were at first limited to the left half of the body, and always began in the left hand after they had become general. The left half of the body was observed to be weak for some time after each partial convulsion. The patient was born with an extensive "mother's mark" on the right side of the head and face; it extended to the mucous membrane of the tongue, uvula, and pharynx on the same side, the sclerotic coat, retina, and choroid of the right eye being also implicated. Dr. Sturge makes the very probable conjecture that the attacks were due to a "port wine mark" on the surface of the right hemisphere of the brain similar to that on the face.

(3) *The Interparoxysmal Condition.*—The condition which exists during the interval between the epileptic attacks was first fully examined by Dr. Russell Reynolds, and deserves careful attention. The most important phenomena observed are those which belong to the mental condition of the patients. In a considerable number of cases no mental disturbance or weakness whatever can be discovered in the condition of the patient between the attacks, and occasionally individuals possessed of high intellectual powers are the subjects of

epilepsy. The classical cases of Julius Cæsar and Napoleon, both of whom suffered from the disease, may be cited as examples.

Slight impairment of memory, especially with regard to recent events, while the remembrance of remote occurrences is intact, is the most common and generally the first mental change. The next grade of mental impairment declares itself in a certain amount of mental dulness and want of apprehension; while a still lower degradation is manifested by confusion of ideas, general want of comprehension, and deficiency in intellectual activity, associated with a stupid and vacant expression. The moral nature is almost invariably perverted, the patients being gloomy, irritable, and distrustful.

One or other of these minor mental defects may continue to exist without any appreciable change during a series of years; but at other times the mental change, beginning with minor degrees of impairment, passes through lower and lower depths of deterioration until the patient arrives at a condition of complete imbecility.

The conditions which determine mental failure in epileptics have been carefully examined by Dr. Russell Reynolds; the following are the most important conclusions at which he arrived. Hereditary taint is without influence, and the same may be said of the duration of the disease, the state of the general health, the number of attacks, the nature of the exciting cause, and the severity of the paroxysm when judged by the duration of the subsequent coma. Frequency of recurrence of the seizures exerts a certain amount of influence in the production of mental impairment, yet there is no constant connection between the two phenomena. Mental impairment is, according to Reynolds, more frequently associated with minor than with major attacks of epilepsy, and appears to be not in *direct* but rather in *inverse* proportion to the degree of muscular manifestation.

Motor Manifestations.—Some motor disturbances occur in the intervals between the attacks in the majority of instances. These appear in the form of simple muscular trembling, or there may be clonic or tonic spasms in single groups of muscles. Clonic spasm is the most frequent, and may occur in either of

two forms. There may be more or less constant choreiform movement, which continues during sleep, but is exaggerated during waking hours, as in the spastic hemiplegia of childhood; or there may be violent spasmodic shakings of the limbs or of the trunk, occurring at irregular intervals, and with special frequency just as the patient is falling asleep; the muscular jerking is sometimes so sudden and violent that the patient is thrown out of bed; or, if standing, he may be thrown down. Cramp or tonic contraction is comparatively rare.

Sensory Manifestations.—The sensory disturbances are by no means so constant and important during the intervals as the motor. Headache and vertigo are the two symptoms most frequently complained of, and the latter must sometimes be regarded in the light of an abortive attack.

State of the General Health.—There is no constant relation between epilepsy and any particular state of health. It is no doubt sometimes associated with a feeble state of the general health, but it is much more frequent to find epilepsy in those who are in other respects quite healthy, and some epileptics are remarkable for their Herculean frame and strength. The most frequent change which Dr. Reynolds observed in epileptics was subnormal temperature, and the least frequent impaired nutrition.

Relations between the Symptoms of Epilepsy.

(*a*) *Forms of Attack.*—Hereditary taint appears to exert an influence in predisposing to the severer form of epilepsy, although milder attacks may be associated with the severer in the cases where there is a frequent recurrence of the latter. When epilepsy is developed at an early age, there is a somewhat increased proclivity to the attack in its milder form. When epilepsy becomes developed in consequence of a peripheral source of irritation, an interval of weeks or even months elapses between the time the injury is inflicted and the first seizure. During this period painful sensations may be felt, or there may be clonic or tonic spasms in the area of distribution of the affected nerve, or the two conditions may be combined. After a time spasmodic seizures appear with loss of consciousness; they are ushered in either by a sensory, motor, or vaso-motor

aura, which is always of the same kind in the same individual, and begins in the region of the injured nerve. When a cicatrix is found in the course of the affected nerve, an attack may occasionally be produced by pressing upon it. Dr. Ogle reports a case in which a seizure could be produced by touching the upper extremity, but the epileptogenous zone is not usually distinctly marked.

(b) Frequency of Attacks.—The paroxysms as a rule recur very irregularly, but Reynolds thinks that frequently "the recurrence of attacks has some relation to time, as marked by its natural division into days, and periods of seven days, and multiples of seven days. Thus a large number of epileptics have their seizures every day, every two weeks, three weeks, and four weeks, while only a much smaller number suffer at such irregular intervals as cannot be thus expressed."

Sometimes the mode of recurrence is what has been termed "serial." The patient suffers from two or more attacks in a day, usually within twelve hours, and then there is a free interval of from one to several weeks. This mode of recurrence is more frequent in the female than in the male sex.

The frequency of the paroxysms varies within wide limits, some patients only having one seizure a year, others having thousands; but Reynolds states that half the cases are found to have a rate of recurrence ranging from one attack in fourteen to one in thirty days. A high rate of frequency is generally observed in those who are in more or less robust health; and a low rate of frequency in those whose general condition has undergone marked deterioration. Early commencement of epilepsy is commonly, but not always, associated with a high rate of frequency of recurrence.

The groups of attacks may be composed of from four to a hundred or more single seizures in twenty-four hours. This condition may extend over a much longer period, and Delasiauve saw in a boy of fifteen, within one month, a "collective seizure" which was composed of 2,500 "fragmentary seizures." The French have designated this condition *état de mal épileptique (status epilepticus)*, and Bourneville has recently drawn attention to the great increase of temperature which characterises the condition. The patients lie in profound coma, and the

temperature may rise to 107·6° F., and still higher in fatal cases. In favourable cases the temperature gradually falls, but in other cases a subsequent rise takes place, coma becomes profound, and the patient dies, often with symptoms of collapse associated with the formation of acute "bed-sores."

It is well to remember that the attacks frequently occur at night, and especially is epilepsy apt to commence in this way. Our attention should be directed especially to this fact if a previously healthy patient complain sometimes on waking in the morning of such symptoms as depression, stupidity, and headache; the surmise of epilepsy will be rendered certain if in addition there have been involuntary discharge of urine or fæces, if the tongue be bitten, or if small hæmorrhages into the skin be found.

§ 957. *Course, Duration, and Terminations.*—Epilepsy is essentially chronic and may last for years, and, although death occasionally occurs during a paroxysm, this is exceedingly rare. The course of the disease does not appear to be much influenced by surrounding circumstances, and not nearly so much as might be anticipated by the health of the patient in other respects. The abuse of alcoholic liquors aggravates the attacks, and even drinking a moderate allowance of beer or wine appears to act unfavourably on the course of the disease. Excess in eating and drinking tea and coffee also appears to aggravate the disease. The influence which a moderate indulgence of venery exerts upon the course of epilepsy is not well determined. Many instances are known where a paroxysm came on during coitus, and venereal excess doubtless aggravates the disease.

During the course of acute diseases the epileptic paroxysms usually cease, but generally return during convalescence; a similar effect is sometimes produced by external injuries. Chronic diseases act in different ways, sometimes mitigating, at other times aggravating the paroxysms, and at still other times not appearing to exert any influence on the course of the disease.

Mental affections of one kind or another are frequently associated with epilepsy, but Reynolds has shown that their concomitance is not nearly so frequent as was at one time

supposed. It appears that some mental affection is found associated with epilepsy in about one-third of all cases, and especially in those cases where the paroxysms follow each other in unusually rapid succession. In some of those cases there is every reason to conclude that the mental affection and epileptic attacks are joint effects of one common lesion.

§ 958. *Morbid Anatomy.*—Post-mortem examination has revealed the most various anatomical changes after death from epilepsy in almost every organ of the body, but none of these are constant and some are quite exceptional. Various irregularities have been found in the structure of the skull, especially if the disease be hereditary or have existed from an early age. The bones of the skull may be thickened, and there may be exostoses on the internal surface of the skull, or contraction of the carotid foramen or of the foramen magnum.

The meninges of the brain are sometimes opaque, thickened, or distorted, especially if osteosclerosis exist at the same time. Various statements have been made with respect to the weight of the brain, Echeverria considering that the brain is increased in weight, while Meynert found a decrease in weight in epileptic insanity. Asymmetry of the cerebral hemispheres has been observed pretty frequently. Meynert also found a difference between the two hippocami majores, caused by progressive atrophy, associated with cartilaginous hardness of one of them, but he regarded this condition not so much a cause as a result of epilepsy. An abnormal distribution of the grey substance of the cerebrum and cerebellum has been met with; but this condition has also been found apart from epilepsy. Disease of the pituitary body is sometimes associated with epilepsy. In chronic cases microscopical changes have frequently been found by Schroeder van der Kolk, Echeverria, and L. Meyer in the medulla oblongata, and also in other parts of the brain and uppermost part of the cervical region of the cord. The changes observed consisted of a granular albuminous exudation, granule cells, dilatation of capillaries, with softening of their walls, and pigmentation of the ganglion cells, especially in the nuclei of origin of the hypoglossus and vagus. Analogous changes were found in various parts of the cerebrum, cerebellum, and basal

ganglia. The cortex of the brain is affected, either directly or indirectly in the large majority of cases of epilepsy. The primary morbid change may then be found in the skull, dura mater, pituitary body, the cortex itself, or even the white substance beneath it; but it is probable that implication of the cortex by the lesion is the essential condition in all of them. In epileptiform seizures, from coarse disease of the brain, the lesion is always situated in or near the cortex of the brain.

Experimental Researches.—The connection subsisting between general convulsions and anæmia of the brain has already been considered. Kussmaul and Tenner found that when the brain in animals is rapidly deprived of arterial blood, either by bleeding or ligature of the four great arteries going to the head, general convulsions and loss of consciousness were invariably produced. They also endeavoured to produce an epileptic attack by faradisation of the sympathetic, but only succeeded in one case after both vertebrals and one carotid were ligatured. The experiments of Brown-Séquard on guinea-pigs, with the view of determining the nature of epilepsy, are very important. He found that injury to various parts of the nervous system developed in these animals an epileptic condition. Section of some of the larger nerve trunks, such as the sciatic, internal popliteal, and posterior roots of the nerves of the leg, partial or complete section of the spinal cord, and wounding of the medulla, crura cerebri, or corpora quadrigemina are the lesions most surely followed by epilepsy. After the wounds heal a state of increased excitability persists; spasmodic twitches appear first in certain groups of muscles, and after a time the animals are seized with complete epileptic attacks. He also found that irritation of the skin over a certain limited area in the antero-lateral region of the neck—generally the area of distribution of the trigeminus and occipitalis—determines an epileptic attack, and that a considerable proportion of the subsequent progeny of those animals are epileptics.

At the meeting of the British Association at Liverpool, where I was present, Dr. Brown-Séquard exhibited a descendant of a guinea-pig that had become epileptic from section of the sciatic nerve, and on gently ruffling the hair at the back of one ear an epileptic attack was induced. The portion of skin, irritation of which causes the epileptic attack, Brown-Séquard has called the *epileptogenous zone*, and it is important to notice that it is slight and superficial irritation of it which provokes the attack; severe irritation, like pinching, may even arrest a commencing attack, and if the skin is burned or cut it loses its epileptogenous character. The skin of the area is, indeed, to a certain degree *anæsthetic*, and in the animals which after a long time recover, as some of them do, the anæsthesia of the epileptogenous zone gradually diminishes, and disappears along with the epileptic tendency.

Another noteworthy circumstance with respect to the epileptogenous

zone is that it is always found on the same side as the injury of the nerve or spinal cord, but on the opposite side to the injury when the crus cerebri has been wounded.

Westphal found that a light tap on the head of a guinea-pig is followed by an attack of general convulsions. The animal soon recovers and may appear well for some weeks, but after a time an epileptogenous zone is developed, irritation of which provokes epileptic attacks, and the attacks also occur spontaneously.

Hitzig has induced artificial epilepsy by injury to the cortical centre of the anterior extremity ; and, according to Ferrier, an epileptic attack may be induced by passing an induction current of moderate strength through the cortex of one of the hemispheres. In the case of a woman whose brain was more or less exposed, in consequence of cancerous ulceration of the skull, Dr. Bartholow applied an induction current directly to the brain in the region of the postero-parietal lobe—a proceeding which was followed by convulsions of the opposite extremities.

Various experiments have been undertaken with the view of determining the starting point of the general convulsions in epilepsy. The most successful of this kind are those of Nothnagel, who proved that general convulsions may be induced by irritation of a circumscribed spot on the floor of the fourth ventricle, a spot which he calls the " convulsion centre."

§ 959. *Morbid Physiology.*—In idiopathic epilepsy no constant anatomical lesion has been discovered, and it may therefore be inferred that the lesion is a molecular one. The question we have now to determine is whether the lesion is a diffused one, affecting the whole nervous system, or a local one, affecting only a definite region. In anæmia, for instance, there is an excess of irritability of the whole nervous system, rendering the patients subject to mental irritability, and less able to withstand any external causes of irritation. In such cases there is excess of the irritability of the nervous system generally, but something more than this is necessary to constitute epilepsy. It is not sufficient that there should be a diffused excess of irritability; the nature of the epileptic paroxysm and its periodic recurrence point to the existence of a rupture of the natural balance between the degree of irritability of different parts of the nervous system, or, in other words, to a state in which some part or parts of the nervous system develop increased irritability, while the remaining portions retain the normal amount. It now becomes necessary to determine more minutely the part of the nervous system where the molecular lesion is situated. Different regions of the nervous system may be affected in different cases.

In some cases there is an epileptogenous zone, in others it is absent; and in some again the sciatic nerve has been injured, and probably remains in a permanently irritable condition. Injury to the spinal cord, the tissues of which may be presumed to remain in an abnormal state of irritability, may also be a source of epilepsy. But the more or less uniform character of the epileptic paroxysm shows that, no matter what other regions may be affected, a molecular disturbance must take place in some one definite region of the nervous system in every attack. Is it possible to localise this region? The most important problems in the pathology of epilepsy cluster around the answer to this question.

The epileptic paroxysm consists of loss of consciousness and convulsions, and our problem now is to determine what are the localities disturbance of which will produce these functional disturbances. Nothnagel has shown that there is a certain limited spot in the floor of the fourth ventricle by irritation of which it is possible to throw the whole of the voluntary muscles into tonic and clonic spasms, and he has consequently named it the "convulsion centre." He believes that irritation of this centre is a necessary concomitant of every epileptic paroxysm. But although irritation of this centre might account for the convulsions it would not account for the loss of consciousness. But the vaso-motor centre which is situated between the upper part of the medulla oblongata and the pons, close to the convulsion centre, is also supposed to be implicated in the molecular disturbance during the paroxysm. Irritation of the vaso-motor centre causes contraction of all the arteries of the body, including the arteries of the brain, and it is to the anæmia caused by the contraction of the vessels of the brain that the loss of consciousness is, according to this theory, to be attributed. Combined excitation of the vaso-motor and convulsion centres is then, according to Nothnagel's theory, the necessary pathological condition of the epileptic paroxysm; excitation of the former centre induces contraction of the vessels of the brain and the consequent anæmia causes loss of consciousness; while excitation of the latter centre induces the muscular contractions. The muscles of the face and neck are first attacked by convulsions, and by their contractions the

large veins of the neck are pressed upon and the return of blood from the brain is thus prevented, so that the *anæmia* of the first moments is succeeded by an intense venous hyperæmia which augments the irritability of the convulsion centre and prolongs the convulsion and coma. Kussmaul and Tenner have shown that when the brain is rapidly deprived of arterial blood, either by bleeding, ligature, or compression of the four great arteries going to the head, coma and general epileptic twitchings are invariably produced. It must be remembered, however, that they did not succeed in a single instance in producing loss of consciousness merely by faradisation of the cervical sympathetics. And if the local contraction of the main arteries of the brain caused by faradisation of the cervical sympathetics will not cause loss of consciousness or convulsions, it is not probable that the contraction of the arteries of the body generally caused by excitation of the vaso-motor centre will cause these states, inasmuch as the general arterial tension will be raised and the more powerful contractions of the heart which will ensue will maintain the cerebral circulation. This hypothesis also overlooks the fact that loss of consciousness is the predominant feature of epilepsy, and not the convulsions. Some cases of true epilepsy are characterised by slight temporary loss of consciousness without any appreciable motor disturbance, a state which differs widely from syncope caused by anæmia of the brain, and is unlikely to be caused by vaso-motor action.

Dr. Todd was the first to attribute epilepsy to an explosion of nerve force; but this theory has assumed definite shape in the hands of Dr. Hughlings-Jackson, who suggested that convulsions of all kinds associated with loss of consciousness were caused by discharging lesions of the cortex of the brain. This hypothesis has received a considerable amount of experimental verification. Hitzig was able to determine epileptic paroxysms by various injuries to the cortex of the brain, and Ferrier obtained a similar result by passing a faradic current of moderate intensity through the cortex, the electrodes being widely separated, so that a large portion of the cortex was included in the circuit.

A survey of the anatomical changes found in epilepsy has

already shown us that, of all the manifold lesions observed, disease of the cortex of the brain predominates. Other facts tend to the same conclusion, such as that where epilepsy is associated with imbecility, structural changes are often found on the surface of the brain, and that general paralysis of the insane, a disease in which structural changes are generally found in the cortex of the præfrontal region, is very often accompanied by epilepsy.

According to Todd's theory, as elaborated by Hughlings-Jackson, the convulsions of epilepsy are due to a large discharge of nervous energy from the cortex of the brain along the centrifugal nerve paths, and the loss of consciousness is caused by the temporary exhaustion which succeeds to excessive nervous discharge. The temporary paralysis of the convulsed limbs observed after epileptiform seizures is also, according to this theory, due to temporary exhaustion of nerve force following the excessive discharge. But, if this be so, it may be asked, why are general convulsions not followed by temporary paralysis? The reply is that they are so followed. After an epileptic attack there is complete muscular relaxation, but as the patient is at the same time unconscious the degree of paralysis present cannot be estimated. Even after consciousness is regained, general muscular feebleness often remains for a time, which, although not called paralysis, is really paralytic in nature (Hughlings-Jackson). The unseemly and apparently immoral actions performed, and the atrocious crimes often committed by patients after minor attacks of epilepsy, may be explained on the supposition that the inhibitory influence of the highest centres is temporarily suspended, thus permitting the centres which preside over automatic actions and animal instincts to spring into greater activity (Anstie). Irritation of the peripheral nerves or of the floor of the fourth ventricle may determine the nervous discharge from the cortex which constitutes epilepsy, as well as direct irritation of the cortex itself.

Epileptiform seizures are always caused by a coarse lesion situated in or near the area of distribution of the Sylvian artery to the cortex, and it may, therefore, be inferred that when attacks of idiopathic epilepsy are ushered in by a motor aura, the molecular disturbance begins in some part of the motor

area of the cortex. When, on the other hand, the aura consists of sensory disturbances, the discharge probably begins in the area of the cortex supplied by the posterior cerebral artery. When the aura consists of emotional states of fear and anger, it is probable that the discharge also begins in the area of the posterior cerebral artery, inasmuch as these emotions are probably often preceded by hallucinations of the senses, or at least by some disturbance of the sensory apparatus. When the aura consists of "dreamy" states, the discharge probably begins in the region of the cortex supplied by the anterior cerebral artery, and the cases in which unconsciousness supervenes suddenly without being preceded by an aura probably also take origin in a molecular disturbance of this area.

§ 960. *Diagnosis.*—Epilepsy is often simulated by impostors, and sometimes so successfully that it is very difficult to detect the fraud. The physician must take into consideration all the circumstances of the case, but the symptoms which can hardly be simulated are pallor of the face, and dilatation and insensibility to light of the pupil at the beginning of the seizure. The slighter attacks of epilepsy are frequently described by the patient as slight "fainting fits," and it is somewhat difficult to distinguish the two affections. If the attacks recur at regular intervals in the absence of any disturbance of the circulation to account for them, they must be regarded as epilepsy. The diagnosis is rendered clearer if, in addition, an epigastric or other sensory aura be described, or if the attack be attended by convulsion, twitching, or impaired consciousness.

Hysteria in its ordinary form may be readily distinguished from epilepsy by the history of the case before the attack, and by the absence of the distortion of the features, dilatation and insensibility of the pupils. Hysterical attacks are unaccompanied by complete loss of consciousness, the tongue is not bitten, there is no marked asphyxia, and the patient, although exhausted, does not pass into stupor before recovery. The diagnosis between hysteria and hystero-epilepsy is readily made when the patient is seen during an attack. Attacks of the latter are always preceded by an aura, and there is com-

plete, although it may be transitory, loss of consciousness. The diagnosis between epilepsy and eclampsia will be considered when the latter disease is under consideration.

When *organic diseases of the nervous system* are attended with convulsions, they present other symptoms over and above the fits by means of which they may be distinguished from epilepsy. The most usual intracranial diseases associated with convulsions are tumour, chronic softening, and chronic meningitis; but in all these diseases some characteristic symptoms, such as optic neuritis, paralysis, or persistent psychical disturbances, are present, which render the diagnosis between them and genuine epilepsy comparatively easy.

§ 961. *Prognosis.*—The prognosis in genuine epilepsy is unfavourable as regards complete and permanent recovery, especially if the disease has been established for some time. It ought, however, to be remembered that a few cases are completely curable, and that, even when the disease has been of long standing, a considerable improvement may take place. In a considerable number of cases, probably the majority, no treatment has hitherto produced any beneficial effect.

The following circumstances influence the prognosis. Hereditary taint gives an unfavourable indication; but an early commencement of the disease is favourable. Herpin thinks that when epilepsy begins after the fiftieth year the prospects are still better. The longer the disease has lasted, the greater the improbability of recovery. Reynolds thinks that those cases in which the intervals between the attacks are much prolonged are less amenable to treatment than are those which exhibit a more rapid recurrence, while Herpin thinks that the prognosis becomes more unfavourable in proportion to the number of seizures suffered in a given time. When the epileptic attacks are caused by peripheral irritation the prognosis is favourable, unless the disease has already been long established, while of course on the other hand central disease renders the prognosis unfavourable. Mental failure is of evil omen, since it indicates in all probability that a profound and permanent molecular change has taken place in the grey matter of the cortex. The danger to life is remote, since it is rare for an epileptic to die in one of the attacks.

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§ 962. *Treatment.*—The treatment of epilepsy must be directed to the removal of the conditions upon which the attacks depend, and to the mitigation or avoidance of the seizures themselves.

When the attacks are maintained by a peripheral source of irritation, this must, of course, be removed if possible. Epilepsy has sometimes been known to disappear after the extirpation of a cicatrix, the removal of a tumour pressing on a nerve, or the opening of an abscess; and the same result has been obtained by the removal of sources of irritation in the alimentary canal, such as worms. When an aura constantly recurred in the course of the same nerve, it was formerly the practice to perform neurotomy, or to amputate a finger or even the arm. No good results attended this practice, and it is now abandoned. The practice of trephining was also extensively employed in former times in every case which resisted medical treatment, and this treatment may possibly be successfully used in a limited number of cases of epilepsy from organic disease. It is scarcely necessary to add that in epilepsy, as in all other chronic diseases, the general health must be carefully attended to and the diet regulated. Alcohol, tea, and coffee should be sparingly used as articles of diet.

Excessive mental effort, emotional excitement, and worry must be avoided; but a moderate degree of intellectual work may be useful, and a certain amount of bodily exercise, short of fatigue, should be enjoined.

Some epileptics have been much improved by the treatment adopted in hydropathic establishments. The application of ice bags along the spine was strongly recommended by Chapman; but Reynolds, who has given the treatment a fair trial, says that he has found the results absolutely negative. Electricity in its various forms has been employed in the treatment of epilepsy, but with little success.

Counter-irritation was at one time extensively used, but the prevailing opinion at present is that the practice is useless. If the presence of an epileptogenous zone be ascertained, a blister over the sensitive area may be attended with benefit.

Bromide of potassium has been found more generally useful in the treatment of epilepsy than any other drug; to do good

it must be given in doses ranging from ten to forty grains three times daily. The results of this treatment are that a few cases have been completely cured; in other cases the attacks have been arrested for varying periods of months or years, but have recurred on the drug being omitted, and ceased again on its being readministered. In still other cases the attacks have been diminished in severity, although not removed; while in a few cases the drug does not appear to exert any influence on the disease. When large doses of bromide of potassium are administered for some time, it is apt to produce an eruption of acne, which soon subsides on the drug being discontinued; or the eruption may, according to Dr. Wilks, be prevented by combining arsenic with the bromide. Chloral is sometimes a useful adjunct to the bromide of potassium. Next to the bromide of potassium the salts of zinc, especially the oxide, have proved the most generally useful in the treatment of the disease. This remedy appears to be more efficient with patients under twenty years of age than in those of maturer age. The oxide may be given in doses ranging from two to five grains three times daily. The sulphate has also been employed in large doses with frequent success. The sulphate may be given at first in doses of three grains, and progressively increased to scruple doses three times a day. The bromide of zinc has been administered in gradually increasing doses up to a scruple three times a day.

The oxide of zinc may be combined with the extract of belladonna or hyoscyamus, or with the powdered root of valerian.

The *ammonio-sulphate of copper* was at one time much used in the treatment of epilepsy, but has lately fallen into disuse.

The nitrate of silver was at one time much relied upon in the treatment of epilepsy, but confidence in its curative power is much shaken in the present day. The records of the older authors, however, amply prove that beneficial results followed its employment, and it may be worth while to give it a trial when other methods of treatment have failed.

Belladonna has been long used as a remedy for epilepsy, and recently its alkaloid, atropine, has been substituted for it. The judgment formed by Reynolds is now pretty generally endorsed by authors, namely, that by means of belladonna an amelioration

is often obtained for various troublesome concomitant symptoms, such as disturbed sleep, trembling, and nervous uneasiness.

Digitalis, either alone or in combination with bromide of potassium, is useful in some cases, especially if there be signs of cardiac failure.

Indian hemp has been found useful as an accessory in the treatment of epilepsy, and by its means headache and restlessness have been relieved, but it does not appear even to have appreciably mitigated the disease.

If there be a suspicion of syphilis being the cause of epilepsy, iodide of potassium should of course be given.

The treatment of the attack should be directed to its prevention, and this is only possible when it is preceded by a distinct warning. When an "aura" is present, the attack may sometimes be arrested by cauterising or blistering the surface from which the aura commences, or by applying pressure, as by a tight ligature, between the starting point of the aura and the trunk. Some patients are able to arrest the paroxysm by a strong mental effort to perform a definite action.

When the aura consists of contraction of a definite group of muscles, the attack may be arrested by forcible extension of them. There are some grounds for believing that the paroxysm may be sometimes arrested by a sudden impression on the surface of the body. Inhalation of chloroform or of ammonia, or a draught of some diffusible stimulant administered at the moment of onset, may arrest an attack.

Dr. Crichton Browne was able to ward off several attacks by causing the patients to inhale nitrite of amyl when a fit threatened. The well-known action of this agent, in paralysing the vaso-motor nerves, supplies the rationale of the treatment.

When the attack is once established, it passes through its regular phases without being influenced by treatment. Measures must be adopted for preventing the patient injuring himself. All tight bands about the throat must be loosened, and a piece of indiarubber or wood should be passed between the teeth, to prevent the tongue being bitten.

When the paroxysm is over, the patient ought to be placed with the head and shoulders raised, and allowed to sleep without interference.

If the paroxysm be long continued, so that there is danger of death supervening from congestion of the lungs, blood-letting may relieve the circulation so much as to arrest the attack. When the fits are violent, a careful trial may be made of the inhalation of chloroform, which is so useful in the treatment of eclampsia.

(II.) ECLAMPSIA.

§ 963. *Definition.*—Eclampsia is an acute affection arising without structural lesion of the nervous system, and characterised by partial or general convulsions, accompanied by a more or less complete loss of consciousness.

§ 964. *Etiology.*—Age is a most important predisposing cause of eclampsia. Convulsions are frequent during the first two years of life, but become rare after the fifth and exceptional after the seventh year of life.

The influence of hereditary predisposition in the production of convulsions is shown by the fact that successive infants of one family are liable to be attacked with convulsions in the absence of any definite cause. Bouchut mentions an instance of a family of ten persons, all of whom had convulsions in infancy. One of these married and had ten children, and nine of them suffered from convulsions. The children of parents who manifest evidences of a neuropathic constitution, as hysteria, neuralgia, or epilepsy, are more liable to be attacked by convulsions than the children of the healthy.

All debilitating causes, as insufficient food, profuse diarrhœa, copious hæmorrhages, malarial cachexia, and various diseases, greatly increase the tendency to convulsions. Amongst the debilitating diseases which predispose to convulsions rickets holds a prominent place. Out of 65 infants attacked with convulsions, Dr. Gee found that no less than 56 of the number were rachitic. Convulsions occur more frequently in children during hot than cold weather, and some authors assert that they are more frequent in female than in male infants, but the influence of sex is not well ascertained.

Eclampsia has been divided into several varieties according to the exciting cause of the convulsions. These are : (1), Idio-

pathic convulsions; (2), Reflex convulsions; (3), Convulsions of fever; (4), Convulsions of asphyxia; (5), Uræmic convulsions; (6), Puerperal convulsions; (7), Toxic convulsions.

(1) In *idiopathic* convulsions the exciting is quite subordinate to the predisposing cause. Some children are so predisposed to convulsions that the slightest exciting cause, such as fear, anger, or a slight colic, may induce a convulsion, while at other times an attack supervenes in the entire absence of any appreciable cause.

(2) *Reflex* convulsions are occasioned by irritation of the extremities of the peripheral nerves. The nature and situation of the external irritation varies indefinitely. Amongst the most usual causes of irritation pricking by pins, wounds and burns of the surface of the body, retention of urine, the presence of a calculus in the kidney, foreign bodies in the external auditory meatus, and irritation of the digestive canal from the presence of worms or undigested food and of the gums during painful dentition may be mentioned.

(3) *Febrile* convulsions manifest themselves at the outset of acute diseases, more particularly in lobar pneumonia, the eruptive fevers, and intermittent fever.

This form of convulsion appears to correspond to the rigor which ushers in most acute febrile diseases in the adult; and if not caused, it is at least accompanied by rapid elevation of temperature. It must not be confounded with the convulsions which supervene in the course of febrile diseases, since the latter are usually symptomatic of cerebral hyperæmia, or of some form of meningitis.

(4) Convulsions due to *asphyxia* occur in the course of diseases of the respiratory organs; they are frequently observed during severe attacks of whooping cough, and may appear as terminal phenomena in most of the diseases of infancy.

(5) *Uræmic* convulsions in children generally result from scarlatinal nephritis, but they have been occasionally observed immediately after birth. These convulsions are also observed in other forms of both acute and chronic nephritis.

(6) *Puerperal* eclampsia is, as a rule, merely a form of uræmic convulsions, although some cases are probably caused by reflex irritation through the uterine nerves or the sacral plexus.

(7) *Toxic* convulsions might be held to include uræmic convulsions, inasmuch as the latter, according to some pathologists, result from the accumulation of urea in the blood, and its conversion into carbonate of ammonia. This hypothesis is, however, doubtful, and it is therefore better to place uræmic convulsions in a separate category.

Certain metallic and organic poisons and irrespirable gases give rise to attacks of convulsions. Amongst these agents the most usual are prussic acid, nicotine, picrotoxine, œnanthe crocata, carbonic oxide, and carburetted hydrogen.

§ 965. *Symptoms.*—An attack of eclampsia cannot be distinguished from a true epileptic seizure, and it is therefore unnecessary to give a minute description of it. Infantile convulsions have been divided into *internal* and *external*, the muscles of the glottis and the respiratory muscles being chiefly affected by spasm in the former and the muscles of external relation in the latter.

The symptoms caused by spasm of the glottis have already been described (§ 279), and we shall consequently limit our further remarks to the external convulsions of children. An attack of eclampsia may occur either with or without premonitory symptoms; the invasion without prodromata being, according to Rilliet and Barthez, the more common.

The premonitory symptoms, when present, usually consist of sleeplessness, and restlessness or drowsiness for a day or two before the attack; while immediately before it the pulse is often hard and wiry, the countenance assumes a frightened expression, or the child starts up frightened from a fitful and uneasy sleep. The convulsion usually begins by conjugate deviation of the eyes, and slight jerking contractions of the muscles of the angles of the mouth. The natural look of the infant is now exchanged for a fixed stare, followed soon afterwards by an upward rotation of the eyeballs, the latter being in its turn followed by a fixed stare and that again by an upward rotation of the globes. The eyeballs are often rotated to the right or left as well as upwards, and the two are generally moved unequally, so that a considerable degree of strabismus may occur. The pupils are sometimes dilated, sometimes con-

tracted, and when they are completely concealed by the superior lids, the whites of the eyes being alone visible, the countenance assumes a frightful and characteristic aspect.

Clonic spasms of the facial muscles produce a series of grimaces and contortions, in which the labial commissures are drawn outwards, and at each successive jerk a peculiar sucking noise is made by the passage of air through the mouth, the lips being covered by a frothy, and often slightly sanguinolent mucus. The superior lip is sometimes drawn upwards, so as to expose the teeth, and the countenance then assumes an almost savage expression. The inferior jaw is sometimes agitated by clonic spasms, while at other times there is trismus, interrupted from time to time by grinding of the teeth. The head is usually strongly retracted, and sometimes rotated to one side. The thumb is flexed into the palm, and the fingers are flexed over the thumb; the forearm is bent upon the arm and is constantly agitated by slight movements of semiflexion and semiextension; the hand is alternately pronated and supinated; and the segments of the superior extremities are contorted into every imaginable shape. The inferior extremities are affected in a similar manner, although to a less degree than the superior. The muscles of the trunk occasionally participate in the clonic convulsions, but as a rule the trunk is maintained rigid by tonic contraction of its muscles. The contraction of the muscles of one-half the body may predominate over those of the opposite side, and then the child is arched laterally in such a way that he may be projected out of bed by the convulsion. The spasmodic contraction of the diaphragm and of the muscles of the larynx produce a peculiar and characteristic noise when air is drawn into the chest during inspiration. Involuntary evacuations may occasionally take place during convulsions. Deglutition is rarely impossible, although attempts to get the infant to swallow during the convulsion are attended with danger.

There is complete loss of consciousness during the attack, but reflex excitability is partially retained. When the convulsion is prolonged the face becomes of a violet colour and bathed in perspiration; the head is hot and the extremities cold; the skin is moist; the pulse is frequent and difficult to count, owing to

jerking of the tendons; and the respirations are accelerated, but stertorous only in aggravated cases.

The ocular muscles and those of facial expression are usually the first to be affected with clonic spasm, and then the muscles of the fingers and forearm. In the more severe convulsions the muscles of the shoulders are affected, but the spasms do not implicate the muscles of the back and lower extremities except in very aggravated cases. The great tonic contractions which form the first stage of the epileptic attack frequently fail altogether in eclampsia.

§ 966. *Course, Duration, and Terminations.*—The duration of an attack of eclampsia varies considerably according to circumstances. The convulsion may sometimes cease in a few minutes, while at other times they recur for hours or days, with only short intervals of calm.

The *terminal convulsions* of asphyxia are generally partial, incomplete, and alternate with coma. The *initial* convulsions of fever are intense and generalised, but are usually limited to a single attack. *Uræmic* convulsions are characterised by their violence, the frequent repetition of the paroxysm, and the profound coma which alternates with or succeeds the latter. After violent and prolonged convulsions ecchymoses of the skin, especially over the face and eyelids, and acute pains of the affected limbs, are frequently observed. Fractures of long bones, dislocations, and ruptures of tendons have been rarely recorded.

An attack of convulsions is frequently followed by complete and rapid re-establishment of health, but in other cases recovery takes place slowly. When the convulsion is due to a meningeal hæmorrhage or some other organic lesion of the brain, it generally assumes a unilateral character, and is followed by paralysis with contractures, choreiform movements, aphasia, or idiocy. Essential convulsions sometimes end in death, which may result after a single violent seizure, or after a series of them occurring in rapid succession. Death is usually produced by asphyxia, either occurring suddenly from spasm of the glottis, or more slowly from coma.

967. *Diagnosis.*—In any particular case of eclampsia it is difficult to decide whether or not the case is one of essential convulsions, epilepsy, or convulsion symptomatic of organic lesion of the brain. The chief points which ought to be attended to in order to arrive at a probable diagnosis are the age of the patient, the state of the temperature and urine, the character of the convulsions, and the previous health during the intervals between the attacks.

Eclampsia is most frequently observed during the first years of life, and is rare beyond that age, except as the result of definite causes; such as albuminuria, and the invasion of an eruptive fever or other acute disease. When the attacks are repeated beyond two years of age, at irregular intervals of months or years, epilepsy may be inferred, unless symptoms pointing to a focal lesion of the brain are present. The state of the temperature is the best guide in deciding between essential convulsions and the initial convulsions of acute diseases, the thermometer being nearly normal in the former and rising to between 103° F. and 104° F. in the latter. The urine should always be examined for albumen in cases of convulsions, in order to determine whether or not the attack depends upon the presence of Bright's disease.

If the convulsions are unilateral in character, or consist of local spasms without loss of consciousness, they are likely to be due to organic disease of the brain or its membranes. Such cases are generally followed by some degree of paralysis with subsequent contracture. If the convulsion be preceded by a well-marked aura, if its onset be marked by a sudden pallor and a piercing cry, and if the first stage be attended by well-marked tonic contractions, the mouth covered by froth, and the tongue bitten, the attack is one of true epilepsy. In epilepsy the return to health after the attack is rapid and perfect, and in the intervals between the severer paroxysms the patient may suffer from attacks of *petit mal.*

§ 968. *Prognosis.*—The prognosis of eclampsia depends upon the character of the attacks, and the causes by which they are produced.

Frequent repetition of the convulsions, the presence of

stertor, cyanosis, or spasm of the glottis, and a small uncountable pulse afford a grave prognosis, whatever may be the cause of the attack.

Essential convulsions are only grave as indicating a neurotic disposition, and, when the attacks recur frequently, there is danger lest they develop into confirmed epilepsy.

Convulsions occurring in cachectic infants and in those exhausted by profuse diarrhœa are almost always the precursors of death.

The initial convulsions of fever derive all their significance from the disease with which they are associated. The convulsions which occur in the course of fevers always justify a grave prognosis.

The convulsions of asphyxia are almost always fatal. Uræmic convulsions terminate more frequently in recovery than in death. If the infant survive the first 24 or 36 hours he may be regarded as safe.

§ 969. *Treatment.*—The most obvious indication of treatment is to remove the cause of the attack, and in reflex convulsion the removal of the cause is often successful. If the gum be tightly stretched over a tooth it may be scarified, but the tooth should be near the surface and the gums hot and inflamed before this practice is adopted. If the bowels be constipated, and especially if they be tympanitic, an injection of warm water is useful. I have often seen the convulsions cease immediately on the bowels being opened after an enema of warm water. If the convulsions are the result of a smart attack of diarrhœa, and especially if the fontanelles are depressed, a small starch enema, with half a teaspoonful of brandy and from 2 to 5 minims of tincture of opium, may be administered. If there be grounds for believing that the convulsions are caused by the presence of worms, an anthelmintic, and if from the presence of undigested food, a smart purgative should be administered. Predisposing causes, such as anæmia, insufficient nourishment, and rickets, must be removed by appropriate treatment.

During the convulsion plenty of fresh air should be admitted into the room, and all articles of clothing should be removed from the neck and chest of the infant. A warm bath is often

useful, but when there is hyperpyrexia, the cold bath should be used in preference. Compression of the carotids and bleeding have been recommended as means of arresting the attack, but the former is ineffectual and the latter only rarely applicable. The inhalation of chloroform is by far the best method of treating attacks of eclampsia. This treatment is applicable to all cases of essential and uræmic convulsions, and is only contra-indicated in cases where there is a considerable elevation of temperature, and in which there is a cyanotic tint of countenance and stertorous breathing. No harm results from keeping the child many hours, if necessary, under the full influence of chloroform. When once the infant is able to swallow, bromide of potassium or chloral hydrate may be given either separately or combined. A grain of the bromide of potassium for each year of age may be given every four hours, and its efficacy appears to be increased by adding an equal quantity of chloral to each dose. If chloral be given alone, a full dose—three grains to a child one year of age—must be administered.

CHAPTER VII.

TOXIC, AND FEBRILE AND POST-FEBRILE NERVOUS DISORDERS.

(I.) ALCOHOLIC NERVOUS DISEASES.

THE subject of acute poisoning by alcohol belongs rather to the province of the toxicologist than to that of the neurologist, and will be passed over here. Besides a large number of nervous disorders produced by the ingestion of alcohol need not be discussed, inasmuch as alcohol is only an indirect cause of them. Amongst these may be mentioned apoplectic attacks caused by degeneration of the vascular system, and chronic dementia caused by hæmatoma of the dura mater.

§ 970. *Delirium Tremens.*—The symptoms known under the name of *delirium tremens* occur in persons after prolonged indulgence in alcoholic excess, or as the result of a single debauch. The characteristic features of the affection are preceded by restlessness, tremor more marked in the hands and in the morning, and wakefulness at night, while a severe attack of vomiting is frequent in the morning. The patient is irritable, suspicious, quarrelsome, and agitated, and the little sleep obtained is broken by disagreeable dreams. The appetite fails completely, so that little or no food is taken for several days; the breath has a characteristic foul odour; the tongue is tremulous on protrusion, red and glazed, or more commonly covered with a thick, creamy fur; the skin is bathed in profuse perspiration; the face is generally flushed, but occasionally pale; the pupils are usually dilated, and the conjunctivæ injected;

the temperature is slightly elevated; and the pulse is usually large, soft, and dicrotous.

Tremors are always present, and are only an aggravation of a slighter degree of the same which had existed for months previously (Anstie). Hallucinations of special sense now make their appearance, those of sight being most common. The patient sees, especially at night when about to go to sleep, sparks of fire and floating bodies; but soon distinct objects, especially those productive of disgust or terror, are seen in broad daylight. The patient sees himself surrounded by insects, snakes, rats, and monsters of variable shape, and armed men pursuing him with threatening gestures. He talks incessantly in an incoherent and rambling manner, and looks suspiciously under the bed, and in every corner of the room, to satisfy himself that none of the imaginary beings by which he fancies himself surrounded are lodging there. His actions, indeed, appear to be largely determined by the nature of his hallucinations. At times he will busy himself in endeavouring to catch the insects which crawl over his bed, or he will get up and search everywhere for something which has disappeared in a corner of the room; while at other times he will dodge about in order to avert a threatened blow, or endeavour to run or hide in abject terror behind an article of furniture, in order to escape from some pursuing foe. The prevailing mental character during the attack is one of terror and cowardice, although the patient may occasionally turn upon his attendant in the belief that the latter is plotting against him, or is about to inflict upon him some bodily injury.

As a rule, the patient is very tractable to his medical attendant, and gives ready obedience to his commands during his visit; but he is not unfrequently violent towards his friends, and especially to his wife.

At the end of three or four days, or at most a week from the commencement, the patient, when the attack is about to terminate favourably, falls into a quiet sleep and awakes refreshed and calm. Some cases, especially if the patient have previously suffered from repeated attacks, terminate fatally by coma or asthenia. In fatal cases the temperature rises to 103° F. or 104° F.; the pulse is extremely rapid and feeble; the tremors

become general, and associated with subsultus tendinum; epileptic convulsions, followed by coma, may supervene and prove fatal, or bed-sores appear and the patient dies exhausted. Death is not unfrequently caused by an intercurrent attack of pneumonia, or some other acute disease.

§ 971. *Alcoholic Paraplegia.*—Dr. Wilks has drawn attention to a condition of partial paraplegia associated with anæsthesia, or pains in the limbs, which is liable to occur in persons, especially women, who have indulged in alcoholic excess. Chronic pains in the limbs may be complained of long before the symptoms of paralysis appear, or there may be a certain degree of motor inco-ordination. The immoderate use of chloral hydrate may occasion chronic pains in the limbs (Anstie), and a distressing case came under my own observation in which the same symptoms were caused by the prolonged and intemperate use of chlorodyne. The pains in the limbs disappeared rapidly when the drug was discontinued.

§ 972. *Treatment.*—The patient should be placed in a dark room, and the utmost quiet enjoined. He should be constantly watched by one or two trustworthy attendants, and the use of mechanical means to restrain his movements should if possible be avoided. Nutriment should be frequently administered in the form of beef-tea, soups, milk, and eggs. A full dose of chloral, either alone or combined with bromide of potassium, may be given at once, and smaller doses repeated at stated intervals. If sleep be not procured on the second night, a full dose of opium or morphia may be administered on the third and subsequent evenings at the usual bed time. According to my experience, opium acts better when given after chloral has been used than at the outbreak of the symptoms. If symptoms of asthenia be present, it may be necessary to give a certain amount of the alcoholic stimulus to which the patient has been accustomed; but, as a rule, alcohol should be wholly forbidden.

(II.) SATURNINE NERVOUS DISEASES.

§ 973. It is impossible to enter into a full discussion of all the deleterious effects produced by the prolonged introduction

of small quantities of lead into the system; it must suffice to mention a few of the leading nervous affections caused by this poison.

Chronic lead-poisoning at one time frequently resulted from the use of drinking-water stored in leaden cisterns or conveyed through leaden pipes, but this seldom happens now. Lead-poisoning is most frequently met with amongst painters, and workmen pursuing various trades in which lead is used. The poison may enter the system by being swallowed along with the saliva, through the lungs by fine particles of the carbonate being diffused in the air, or through the mucous membrane of the nose by the adulteration of snuff with red lead (Winter). Some individuals are much more susceptible to the action of lead than others; and, as was first suggested by Dr. Garrod, those who inherit a predisposition to gout appear to be particularly liable to become poisoned by lead.

§ 974. *Symptoms.*—One of the most valuable indications of the presence of lead in the system is afforded by the formation of a blue line along the edges of the gums immediately adjoining the teeth. The blue line, although situated in the substance of the gums, appears to be produced by the formation of a sulphide, the latter being formed by sulphuretted hydrogen emitted from decomposing matters on the teeth.

Sensory Disturbance.—Phenomena of sensory irritation may be manifested in the form of hyperæsthesia of the superficial and neuralgia of the deeper parts. Rosenthal states that cutaneous hyperæsthesia often accompanies paroxysms of pain, and that it may alternate with anæsthesia. *Arthralgia*, probably of a neuralgic character, is a prominent symptom of chronic lead-poisoning. The pains in the joints occur in paroxysms, and may appear in the upper or lower extremities, or in the jaws. *Lead colic*, probably also of neuralgic origin, is one of the most frequent and important symptoms. The pains are chiefly referred to the umbilical region; they are liable to paroxysmal exacerbations of great severity, although a considerable degree of uneasiness or pain remains during the intervals (§ 334).

Cutaneous *anæsthesia* is, however, much more commonly

observed than hyperæsthesia, and is, according to Bean, one of the most characteristic symptoms of chronic lead-poisoning. It is variable in its distribution, and may be complete or incomplete. Tactile anæsthesia is often associated with motor paralysis, colic, or arthralgia. It is most frequently situated on the skin of the backs of the hands and forearms, the external surface of the calves, and the abdomen and chest, the skin over the epigastrium, however, remaining always free (Bean). Loss of feeling sometimes extends to the veil of the palate and uvula. In other cases analgesia, thermo-anæsthesia, and loss of the sensibility to tickling may be present, while tactile sensibility remains unimpaired. The electric sensibility is often lost (Raymond). The anæsthesia is often transitory, and, according to Renaut, is sometimes caused by cutaneous anæmia, and may be made to disappear by rubefaciants and profuse diaphoresis.

Deafness, according to Tanquerel, frequently follows an attack of arthralgia, and diminution of taste on half the tongue, and of smell in one nostril, has been observed. But the affections of sight are more frequent and important than those of the other special senses. These consist of transitory amblyopia without ophthalmoscopic changes; persistent amblyopia passing on to amaurosis of both eyes and rarely of one only, attended by atrophy of the optic nerve; amblyopia with double optic neuritis; and amblyopia with albuminuric retinitis, in association with granular kidneys.

Motor Disturbances.—Motor are more commonly observed than sensory disorders in lead-poisoning. Almost all the muscles of the body may be affected, although certain groups are attacked by preference. In partial paralysis the extensor muscles of the forearm are more frequently affected than any other group; and consequently when the arms are held out horizontally, with the hand in a state of pronation, the hand is flexed at the wrist and cannot be extended, this condition being technically called *wrist-drop*. The common extensors of the fingers are first attacked, then the extensors of the index and little fingers, and lastly, in succession, the extensor secundi internodii pollicis, the extensors of the wrist, the extensor primi internodii pollicis, and the extensor ossis metacarpi pollicis. The supinator longus is spared until a comparatively late period of

the disease, and is never affected, according to Remak, unless the paralysis extend to the muscles of the upper arm. Sometimes the paralysis begins in the muscles of the upper arm, and then the deltoid, biceps, coraco-brachialis, and supinator longus are affected (the upper arm type of Remak) (§ 396). When the muscles of the inferior extremities are paralysed, the anterior muscles of the leg are generally the first to be affected; but the tibialis anticus is often spared under such circumstances. In some cases all the muscles of both upper and lower extremities are paralysed, and on rare occasions the muscles of the thorax and back, those of phonation and speech, the intercostal muscles and even the diaphragm have been affected. The sphincters remain always unaffected in the paraplegic form. Paralysis of the muscles of the glottis has been observed by Trousseau in horses employed in red-lead factories. The paralysed muscles undergo rapid atrophy, and lose their faradic contractility before voluntary power is completely abolished, while the reaction of degeneration appears in them at an early period.

The duration of lead paralysis is very variable; it may last for a period of weeks, months, or years, and both it and the colic are liable to recur on renewed exposure to the cause.

Tremor is sometimes observed in lead-poisoning. It is often limited to the upper extremities, and in aggravated cases may extend so as to become general. Ataxic symptoms have occasionally been observed, and are generally associated with anæsthesia (Raymond). The patients in lead-poisoning suffer from chronic dyspepsia and occasional attacks of jaundice; there is pronounced anæmia; the arteries undergo various degenerations; and chronic Bright's disease, with its associated effects on the mechanism of the circulation, is frequently observed. It is scarcely necessary to add that the hemiplegia, which is apt to occur from rupture of a vessel in the brain under these circumstances, must be distinguished from true lead paralysis. Women poisoned by lead suffer from menstrual disorders and profound anæmia, and, if bearing children, they frequently abort or have stillborn children; idiocy, imbecility, and epilepsy appear to be frequently observed amongst the children of workers in lead. Impotency is said to occur in advanced cases.

Psychical Disturbances.—Before the outbreak of pronounced cerebral symptoms the patient often suffers from headache, vertigo, and drowsiness during the day and sleeplessness at night; or there may be a state of agitation or complete apathy. Cerebral disturbance sometimes assumes the form of quiet delirium, accompanied by hallucinations of sight and hearing, or on the other hand the delirium may be furious. But the most common cerebral disturbance is convulsions. Sometimes the loss of consciousness is not complete and the convulsions may be partial, and limited to the muscles of the face and of one or more limbs; or they may be general and represented by general trembling of the body. At other times the attack assumes the form of eclampsia. These convulsions are generally followed by a prolonged stage of unconsciousness, with stertorous breathing. The patient may be comatose after a first attack, or after a succession of attacks quickly following each other, and separated in some cases by intervals, during which there is furious delirium. The patient generally recovers from the first attack, but is liable to die in subsequent attacks. Apoplectiform attacks may occur in the later stage of lead paralysis, and are accompanied by paralysis of variable distribution; inasmuch as the patient often recovers motor power quickly, these attacks cannot always be due to hæmorrhage.

§ 975. *Morbid Anatomy.*—The morbid anatomy of lead-poisoning has been studied by Lanceraux, Gombault and Charcot, Westphal, Vulpian and Raymond, Erb, and many others; a careful paper on the subject, by Dr. S. Moritz, of Manchester, has recently appeared in the "Journal of Anatomy and Physiology." The microscopical changes observed in the muscles are more or less similar to those already described as occurring in progressive muscular atrophy (§ 414). The most important changes observed have been in the intra-muscular nerve fibres. The connective tissue is thickened, the sheath of the primitive fibres is also thickened, the nuclei are largely developed between them; the axis-cylinders are sometimes distinctly visible, and at other times apparently disappear (Moritz). The nerves, especially the musculo-spiral, have been found altered in various degrees. Kussmaul and Meyer observed

sclerosis of the cœliac and upper cervical ganglia, with proliferation of the connective tissue and deformity of the cells. Vulpian observed vitreous degeneration and atrophy of the ganglion cells of the anterior horns of the spinal cord, and a similar observation has recently been made by Monakow. Other observers have failed to detect any changes in the spinal cord.

§ 976. *Morbid Physiology.*—There can be little doubt that, as first suggested by Duchenne, the muscular disease in lead paralysis is secondary to nervous changes. Some observers believe that the disease begins in the intra-muscular nerve fibres; while others believe that the primary disease is situated in the ganglion cells of the spinal cord. It is at least certain that the muscles are affected in groups according as they are associated in their actions, and not according to the distribution of a particular nerve, such as the musculo-spiral. This mode of invasion corresponds to what occurs in infantile paralysis and progressive muscular atrophy, both of them spinal diseases, and differs from that of paralysis of peripheral origin. These considerations tend to show that the paralysis is probably of spinal origin (Remak).

§ 977. *Diagnosis and Prognosis.*—Saturnine neuroses are generally easily recognised by the knowledge of the occupation of the patient and the presence of a blue line on the gums. The prognosis is at first favourable, but if colic have frequently recurred, or paralysis have existed for a long time, and there be much muscular wasting and cachexia, it becomes gloomy, especially when the patient remains exposed to the poison.

§ 978. *Treatment.*—Patients whose occupations expose them to poisoning by lead should if possible seek other employment. If this be not possible, the patient ought to be instructed to observe great personal cleanliness, to wash the teeth daily, and to rinse the mouth frequently with cold water. The use of lemonade made with sulphuric acid is said to prevent lead-poisoning by converting the carbonate of lead in the stomach into an insoluble sulphate. The most important method used

for procuring elimination of the poison appears to be the internal administration of iodide of potassium, which is said to convert the insoluble salts of lead deposited in the tissues into a soluble double salt, capable of being removed. The warm bath may be used as an adjunct in treatment; no benefit appears to result from the addition of a soluble sulphide to the water.

Lead colic must be treated on the same general principles as other forms of colic with constipation (§ 336). The paralysed muscles must be subjected to electrical treatment.

(III.) MERCURIALISM.

§ 979. Chronic mercurial poisoning may be due to the absorption of mercurial preparations through the skin or mucous membrane, or to the inhalation of the vapour of mercury. The workmen engaged in quicksilver mines, and in trades in which mercury is employed, such as that of gilders and looking-glass makers, are liable to be affected by it.

§ 980. *Symptoms.*—The symptoms of chronic poisoning by mercury often begin by slight numbness in the hands or feet, and occasional neuralgic pains in certain joints, especially those of the thumbs, elbows, feet, and knees. These sensory disturbances are accompanied or soon followed by slight tremor, which may for some time remain limited to the hands and arms. The tremor, like that of disseminated sclerosis, only reveals itself when the patient makes a voluntary effort; but at an advanced period of the disease it persists during repose, and may even continue during sleep. The tremor gradually becomes more pronounced, and extends to all parts of the muscular system. The lower extremities tremble, especially at the knees, when the patient stands or walks, and the patient is incapable of performing any delicate manipulations, while in aggravated cases he may be unable to carry a glass of water to his mouth, as in disseminated sclerosis. The head and neck are maintained in a state of constant oscillatory movement when the patient is in the erect posture; the lips are tremulous; the utterance becomes broken and indistinct; mastication is

rendered difficult; and even respiration becomes irregular and laboured. The muscles of the eyeballs are said never to be affected in mercurial poisoning, a fact of great importance in distinguishing it from cerebro-spinal multiple sclerosis. Muscular weakness is associated with the tremor, but distinct paralysis does not occur, and there is no loss of sensation. When the tremors attain great intensity, they persist during repose, and render the patient restless and sleepless at night; the appetite fails; the pulse, strong and slow at first, becomes small, feeble, and frequent; while the patient becomes emaciated, and assumes a cachectic appearance. In the advanced stage of the disease serious cerebral symptoms supervene, such as constant headache, sleeplessness, loss of memory, epilepsy, and coma.

§ 981. *Treatment.*—The patient must first of all be removed from the influence of mercury, whatever be the way in which he may be exposed to it. Iodide of potassium may be administered with the view of converting the mercurial compound already in the system into a soluble double salt. The affected muscles are to be subjected to local treatment by galvanism.

(IV.) SYPHILIS OF THE NERVOUS SYSTEM.

It has been abundantly shown in the course of this work that syphilis may be a cause of almost all the organic diseases to which the peripheral nerves, spinal cord, and brain are liable. Syphilis, indeed, as Mr. Jonathan Hutchinson remarks, mimics nearly all the organic diseases of the nervous system, as well as those of other organs. Instead, therefore, of attempting to write a detailed description of the numerous manifestations of syphilis of the nervous system, it will suffice here if we recapitulate briefly the anatomical alterations produced by the action of the poison, bring into prominence the chief points which must be attended to in recognising its presence, and make a few remarks on treatment.

§ 982. *Morbid Anatomy.*—Syphilis of the nervous system usually belongs to the later secondary or to the tertiary mani-

festations of the disease, although in rare cases it may appear in the first few months, or the year following infection. Syphilitic growths are sometimes developed in the nervous system as many as twenty or even thirty years after the primary infection. Syphilis of the nervous system occurs with greatest frequency in middle age. Affections of the nervous system not unfrequently occur during the first few years of life as the result of the congenital disease.

Syphilitic lesions may be subdivided into: (1) Primary, or those which are directly due to the action of syphilis; and (2) Secondary lesions, or those which are indirect and remote consequences of it.

(1) Primary Syphilitic Lesions.

(*a*) *Disease of the Bones and Periosteum.*—Syphilitic exostoses, periostitis, osteitis, and caries of bones in the neighbourhood of nervous structures may implicate the latter in disease. In this manner the peripheral nerves, as they pass through bony channels, may be compressed or otherwise injured, the vertebral canal may be narrowed and the spinal cord pressed upon, and disease may be set up in the brain and its membranes by syphilitic affections of the cranial bones.

(*b*) *Formation of Gummata.*—These have already been described sufficiently for our purpose (§ 732). Gummata may grow in the dura mater or pia mater. When a gumma grows in the dura mater it develops between its two layers and becomes encapsulated. When it is developed in the subarachnoid space all the surrounding tissues, including the membranes, the blood-vessels and nerves which traverse the space, and the substance of the brain itself, are involved in the lesion. The majority of cerebral gummata originate from the subarachnoid space and pia mater, and grow towards the substance of the brain. If the growth be situated on the convexity and lateral surfaces of the hemispheres, the dura mater becomes so closely adherent to the cortex that the former cannot be separated without producing laceration of the latter. If it be situated at the base of the brain the dura mater is less frequently implicated, and the new growth then usually fills the spaces around the chiasma and infundibulum, the interpeduncular

space, and the spaces at the anterior and posterior borders of the pons.

Gummata may also grow between the layers of the dura mater or in the subarachnoid space in the vertebral canal. The membranes become adherent to one another, and the spinal cord is compressed and gradually destroyed at the level of the growth.

The peripheral nerves may also be affected by a gumma situated in their neighbourhood, or by the extension of the infiltration into the substance of the nerve. The cranial nerves are most commonly implicated at their points of origin and before they become covered by a prolongation of the dura mater.

(*c*) *Syphilitic Infiltration.* — The gummatous formation sometimes forms a diffused infiltration in the substance of the nervous tissues instead of forming circumscribed tumours. A layer of gummatous tissue may in this manner be formed in the pia mater on the surface of the convolutions of the brain.

(*d*) *Syphilitic Sclerosis.*—It is very probable that syphilitic sclerosis is always preceded by an infiltration of the nervous tissues by young cells similar to those observed in the gummatous infiltrations. These cells become infiltrated around the vessels and in the connective tissue septa and neuroglia, and subsequently undergo partial organisation and cicatricial contraction. This process leads to the gradual destruction of the nerve elements, just as occurs in ordinary chronic interstitial inflammation of the brain, spinal cord, and peripheral nerves.

(*e*) *Syphilitic Adhesion and Opacities.*—When the cellular infiltration occurs in the membranes and subsequently undergoes organisation and retraction, a fibroid tissue is formed which renders the portion affected dense, opaque, and inelastic; adhesions form between the dura mater and pia mater, and between the latter and the cortex of the brain or the surface of the spinal cord. When the membranes over the base of the skull and brain are affected, the cranial nerves may be surrounded by cicatricial tissue. When the pia mater becomes indurated, the calibre of the vessels supplied to the cortex of the brain is liable to be diminished, and the nervous tissues are thus imperfectly supplied with nourishment.

(*f*) *Syphilitic Periarterites and Endarterites.*—It is probable that the adventitia of the smaller arteries are implicated to a greater or lesser extent in all the syphilitic processes which have been described. Medium sized vessels may sometimes be surrounded by concentric layers of gummatous tissue, which ultimately compress them so as to cause their partial obliteration. A gumma may, like any other tumour, during its growth compress and obliterate both arteries and veins in its neighbourhood. But the walls of the arteries are liable to be affected in syphilis in a much more direct manner than by any of the processes just described. The substance of the walls may be infiltrated with cells, and these may undergo partial organisation and cicatricial retraction, or form gummatous masses. The infiltration may take place chiefly into the adventitia of the vessel (periarteritis) or between the intima and endothelium (endarteritis), but it is probable that in most cases all the coats are more or less infiltrated. When the cellular infiltration is diffused throughout all the coats of the vessel and undergoes partial organisation and cicatricial retraction, the walls of the affected artery become inelastic and brittle, while its calibre is uniformly reduced in size. When, on the other hand, the infiltration is more limited, hard circumscribed spots may be found, which project from the external or internal surfaces of the vessel, distorting it in various ways. It would appear that distinct gummata may form in the walls of arteries, and either project from its external surface or into its lumen, and in the latter case may either obstruct the vessel completely or be washed off to be arrested as an embolus in one of the smaller branches.

(2) Secondary Syphilitic Lesions.

The processes which result indirectly from syphilis are—(*a*) *Inflammation;* (*b*) *Partial ischæmia, with necrotic softening.*

(*a*) *Inflammation.*—Syphilitic diseases of the bones of the cranium may set up suppurative arachnitis (Wilks and Moxon), but the purulent affection is of itself not a syphilitic lesion. Syphilitic gummata act like foreign bodies on the surrounding tissues, and consequently the membranes in its neighbourhood

are usually thickened and adherent, while the cerebral tissue surrounding it is maintained in a state of irritation. It is probable that the thickened layer by which a gumma is sometimes encrusted is formed by partial organisation of inflammatory products in the tissues immediately adjoining the syphilitic tissue. Some cases reported appear to show that the presence of a gumma on the surface of the brain may set up an acute attack of meningo-encephalitis (Gamel). Acute ascending spinal paralysis is liable to occur in syphilitic subjects, but in such cases it is difficult to determine whether or not the lesion is a primary or secondary result of the syphilitic poison.

When once a sclerosis of nervous tissues is set up by a syphilitic lesion, it is probable that the process may assume a progressive character independently of the syphilitic poison.

(*b*) *Partial Ischæmia and Necrotic Softening.*—When a portion of the pia mater undergoes fibroid thickening in syphilis, the calibre of the vessels which pass through it to nourish the subjacent nervous tissues is reduced in size, and these tissues suffer from anæmia. As the fibroid thickening is probably always local in syphilis, the resulting anæmia is also local. Much more important, however, is the anæmia caused by obliteration of vessels. Obliteration of the vessels may occur in several ways; but occlusion of an artery by the formation of a thrombus at a point where its inner surface has been rendered uneven and its calibre diminished is by far the most common and important of these. Obliteration of an artery, in whatever way it may be brought about, is followed by partial ischæmia, and local softening in those portions of the brain where the terminal arteries do not anastomose with one another.

Situation and Mode of Distribution of Syphilitic Lesions. From what has already been said it will be seen that gummata as a rule form in the membranes of the brain and spinal cord. It follows that the cortex of the brain and the white columns of the spinal cord are especially liable to be affected by gummata. The favourite situations of gummata in the brain are the base and cortex of the convexity in the region of distribution of the middle and anterior cerebral arteries.

Syphilitic thrombosis, like every other form of obstruction

of arteries, assumes greater importance when it occurs in the arteries of the brain than in those of other parts of the nervous system. The middle cerebral artery and its branches are particularly liable to become occluded in syphilis, hence the frequency with which hemiplegia with or without aphasia occurs in syphilitic subjects.

Gummatous growth, whether it form a circumscribed tumour or be infiltrated, is usually more or less localised, and consequently gives rise to the symptoms characteristic of focal diseases of the brain.

Syphilitic lesions are very liable to be multiple, or, in other words, to appear at different parts of the nervous system at the same time, so that the symptoms produced are such as those resulting from more than one focus of disease. When the lesions are bilateral they are seldom symmetrically placed, they often appear at different times, and frequently differ in kind. A syphilitic lesion compressing a cranial nerve on one side may be associated with a gumma of the cortex of the opposite hemisphere, but rarely with a gumma compressing the corresponding nerve on the opposite side. A syphilitic lesion of one of the cranial nerves is often associated with syphilitic thrombosis of cerebral vessels, but these lesions usually appear at different times, so that there is a history of two separate attacks.

§ 983. *Diagnosis.*—In some cases constitutional symptoms are so apparent that the presence of syphilis cannot be overlooked. If characteristic cutaneous eruptions and ulcerations, osseous defects in the nose and palate, be present, the nature of the case can hardly remain in doubt; although it must not be forgotten that persons who have previously suffered from syphilis are also liable to nervous diseases of non-syphilitic origin. Nervous affections, as a rule, belong to the later manifestations of the disease, and make their appearance long after the more prominent symptoms of the constitutional disease have ceased to exist. Search must then be made for cicatrices on the genitals or on the groins, round pigmented spots on the skin; depressed and irregular cicatrices over the forehead and front of the legs with the integument adhering to the subjacent bones; radiated cicatrices on the mucous membranes,

especially of the mouth; circular depressions on the arches of the palate or tonsils, which look as if a piece of tissue had been punched out; irregular protuberances on the surfaces of the bones; a moderate degree of, but hard, swelling of the occipital, cervical, or cubital lymphatic glands; and enlargement and knobby induration or atrophy of one testicle. An inquiry into the history of a case may throw great light upon its nature. If the patient be a man, it may be asked whether he has ever suffered from syphilitic infection. In the case of a married woman, valuable information may be obtained by ascertaining whether or not she has had miscarriages, if some of her children were still-born or died soon after birth, or whether they manifest any of the characteristic symptoms of congenital syphilis.

(1) *Syphilitic Lesions of the Peripheral Nerves.*—Syphilitic lesions of peripheral nerves, like all other similar lesions, are manifested by symptoms of irritation, as hyperæsthesia, neuralgia, and spasm; followed by symptoms of depression, as simple anæsthesia, anæsthesia dolorosa, or paralysis in the region of distribution of the affected nerve, the symptoms of depression being much more important and frequent than those of irritation. Syphilitic disease of the peripheral spinal nerves may occur; but the cranial nerves are much more frequently affected. In syphilitic disease of the motor nerves the paralysis is sometimes limited to a single muscle; while the other muscles supplied by the same nerve remain unaffected, or manifest only a slight degree of weakness. The oculo-motorius appears to be the most frequently affected of the cerebral nerves, and *ptosis* generally precedes paralysis of the recti muscles. When, therefore, ptosis is suddenly developed without any apparent cause, syphilis should be suspected. It must be remembered that paralysis of one or more of the ocular muscles is liable to appear in the early stage of locomotor ataxy, and cases presenting these symptoms should, therefore, be carefully examined to see whether or not lancinating pains, absence of the deep reflexes, or ataxia be present.

If double optic neuritis, paroxysmal vomiting, and headache be present along with the paralysis of the third nerve, the latter is caused by the pressure of a tumour on the nerve at the base of the brain, but even then the tumour may be of syphilitic

origin. Aneurism of one of the arteries at the base of the brain may give rise to similar symptoms, but it is frequently associated with vegetations on the cardiac valves (Ogle, Church). Basilar meningitis may also paralyse the third nerve; the acute form of this disease bears no resemblance to syphilis, but the chronic form of meningitis may be indistinguishable from it, except by the fact that treatment is not followed by favourable results. The sixth nerve is also frequently affected in syphilis, either separately or along with the fifth or seventh nerve on the same side. Syphilitic disease of the fifth is not unfrequent, and the nerve may be affected at its origin, at the Gasserian ganglion, or in its separate divisions. Disease of this nerve first declares itself by neuralgic pains in the region of its distribution, which are liable to nocturnal exacerbations, and occasionally anæsthesia may be associated with the pain. The motor root of the nerve is often affected, and then there is masticatory paralysis with atrophy and the reaction of degeneration in the affected muscles. The paralysis is sometimes preceded by spasmodic movements of the affected muscles. When the Gasserian ganglion is implicated there is lachrymation and neuroparalytic ophthalmia. Syphilitic affections of the fifth nerve are probably never bilateral, and if both be paralysed by the pressure of a tumour at the base of the brain, the growth is likely to be cancer (Hutchinson).

The seventh nerve is, with the exception of the motor nerves of the eyeball, more frequently affected in syphilis than any other cranial nerve. The paralysis may affect all the branches of the nerve, or, contrary to what occurs in central paralysis of the facial, the muscles about the eye may be the first to become paralysed. The hypoglossal nerve is probably never subject to isolated paralysis in syphilis. The optic nerves, chiasma, or tracts may be the first to suffer, and unilateral amaurosis with descending neuro-retinitis, or different forms of hemiopia may occur according to the situation of the lesion. Some cases of amblyopia or amaurosis have been recorded in which no lesion could be detected by ophthalmoscopic examination, but which were cured by antisyphilitic treatment. Various forms of neuralgia are held to be of syphilitic origin in the absence of anatomical proof, because they occur in syphilitic

subjects and yield to antisyphilitic treatment. The most frequent of these are sciatica, occipital neuralgia, and neuralgias of the testicle, scrotum, and various viscera.

(2) *Syphilitic Lesions of the Spinal Cord and its Membranes.*—These form late manifestations of the disease, and, as a rule, marked cachexia is present before their appearance. The nervous symptoms are generally preceded by general languor and a feeling of debility; after a time symptoms of sensory irritation set in, which may last for months without paralysis. Pains, increased by pressure, are sometimes situated at a fixed spot, over the vertebral column, in the cervical, lumbar, or sacral region. At other times they are situated in the extremities; at first limited to an arm or leg, but later involving the other limbs. The pains are more rheumatic than neuralgic in character, and are subject to great variations in duration and intensity (Heubner). The patient often complains of paræsthesiæ, such as formication, tingling, and numbness in the affected extremity.

After a time motor disturbances appear in the form of rigidity and temporary spasms of groups of muscles or an extremity. The symptoms are liable to great fluctuations, and may entirely disappear for a time, the free intervals being sometimes of several months' duration. Sooner or later, however, the symptoms of irritation give place to those of paralysis. The patient complains of increasing weakness in one leg or in both the leg and arm of the same side if the lesion be situated in the cervical region, and in a short time complete paralysis is developed. Before long the opposite side of the body is affected, and the paraplegia becomes complete. The accompanying disturbances of sensibility do not increase in corresponding ratio as they do in myelitis or other tumours of the cord. The extent of the paralysis will depend upon the seat of the lesion. When the lumbar region is affected both lower extremities will be paralysed, but one usually to a greater extent than the other, and the sphincters will also be involved in the paralysis.

After a time the symptoms remain stationary for a considerable period, and the patient is confined to bed for weeks or even months. If energetic treatment be adopted, the case may slowly improve and terminate in comparative recovery, the

most favourable cases being those in which the morbid process is limited to the lowest part of the cord.

Improvement begins in the less affected extremity, which after a time completely regains its motor power; but, although the other extremity improves, a certain degree of motor weakness persists. When the sphincters are affected, bed-sores and cystitis with their usual deleterious consequences are apt to develop.

When the cervical region is implicated, and especially the upper portion, the prognosis is very grave, a condition of general paralysis being rapidly developed.

But even aggravated cases may improve under energetic antisyphilitic treatment, although the spinal cord remains to a greater or lesser extent permanently diseased. If the syphilitic lesion have extended from the pia mater to the lateral columns a spastic paralysis, resembling more or less that of primary lateral sclerosis, is developed; while locomotor ataxia is simulated if the lesion be limited to the posterior columns. In the above cases the syphilitic lesion consists of the formation of a gummatous tissue, either in the form of a more or less circumscribed tumour or diffused infiltration into the spinal cord; at other times the lesion appears to assume the form of a chronic degeneration or sclerosis from the commencement. It is probable that about half of the cases of locomotor ataxia are of syphilitic origin (Buzzard, Gowers, Erb), and in most of these the lesion is probably from the first a chronic degeneration. The grey matter appears to be primarily affected at other times. Progressive muscular atrophy is probably sometimes of syphilitic origin, while labio-glosso-laryngeal paralysis is frequently and exophthalmoplegia externa is said to be always of syphilitic origin. A case came under my own observation in which the symptoms of acute spinal paralysis of adults occurred in a man at the age of 23, while he was suffering from secondary symptoms. He was 47 years of age when I saw him, and the symptoms present were slight ptosis, paralysis of the superior rectus, and comparative dilatation and sluggish movement of the pupil of the right eye, a slight degree of atrophy of the right half of the orbicularis oris, paralysis with decided atrophy of the muscles of the right half of the tongue, and

complete paralysis and atrophy with loss of the electric contractility of all the muscles which produce dorsal flexion of the foot. All sensory disturbances were absent. The presence of multiple lesions in this case points to its syphilitic origin. It must also be remembered that acute ascending paralysis is apt to become developed in syphilitic subjects; but no lesion which can be regarded as characteristic of syphilis has been discovered in the spinal cords of such cases.

(3) *Syphilitic Lesions of the Brain and its Membranes.*—The outbreak of cerebral syphilis is generally preceded by premonitory symptoms. *Headache* is the most constant and important of these, and it may precede more pronounced cerebral symptoms by days, months, or even years. It occurs in paroxysms which are sometimes so intense as to be almost insupportable. The pain is seldom diffused over the whole head, but generally occupies the lateral, anterior, or posterior half, or is limited to a very circumscribed region which is tender to pressure. The headache is liable to nocturnal exacerbations of great severity, while there is a remission or complete intermission in the morning; it may entirely disappear for weeks or months, without treatment, and afterwards recur with great severity. *Sleeplessness* is another important symptom of this early stage; it is sometimes but not always the result of the headache, and may continue during the remissions of the latter. Other premonitory symptoms of less constancy and importance are, attacks of dizziness, feeling of fainting, numbness in the head, shooting pains in the extremities, general discomfort, slight loss of memory, mental confusion, great excitability of manner, and irritability of temper. Some of these symptoms probably occur in every case, but they may sometimes be so slight and transient that the patient does not complain of them unless questioned.

The purely nervous symptoms depend upon the nature and situation of the lesion, and may be divided into the following varieties: (*a*) Symptoms caused by the presence of a gumma within the cranium, (*b*) those caused by occlusion of one of the arteries of the brain, and (*c*) those caused by chronic degenerative changes.

(*a*) *Gumma.*—If the gummatous tissue form a distinctly

circumscribed growth, it gives rise to double optic neuritis, and the other symptoms which characterise intracranial tumours. If the syphilitic tissue be infiltrated, the symptoms of a focal disease are probably present, but without double optic neuritis. We have already seen that syphilitic growths are situated near the surface of the brain. When the lesion is situated at the base of the brain, the most prominent phenomena will be those of pressure upon the cranial nerves, which have already been considered. When, again, the growth is situated on the convexity, the most prominent symptoms are due to implication of the cortex of the brain. The primary lesion of the cortex is generally irritative; but, as the growth enlarges, part of the cortex is injured, so that a destroying lesion is superadded to the irritative or discharging one. If the lesion be situated in the area of distribution of the Sylvian artery, the symptoms begin by a unilateral epileptiform attack, usually followed by some degree of paralysis of the muscles first implicated in the convulsions. These epileptiform convulsions (Jacksonian Epilepsy) have been already fully considered, and it is unnecessary to describe them further. If the syphilitic lesion be situated in the region of the anterior cerebral artery, then psychical disturbances predominate, consisting of a drowsy delirium followed by a somnolent condition, and more or less coma.

(*b*) *Neurotic Softening.*—Occlusion of a cerebral vessel, as the result of syphilis, produces all the usual symptoms of that accident from any other cause. When a vessel is occluded, unless collateral circulation be soon established, a focus of softening results, which produces the usual phenomena of focal disease. If the vessels of the lenticular nucleus are occluded, hemiplegia may be produced, but the patient recovers; occlusion of Broca's artery causes ataxic aphasia, and of the posterior branch of the Sylvian artery, amnesic aphasia; while softening in the area of the anterior cerebral artery gives rise to loss of memory, confusion of ideas, and other psychical disturbances. What distinguishes syphilitic thrombosis from other forms of occlusion of vessels is that it often occurs at a comparatively early period of life, when atheroma of the arteries is not usually present, and in the absence of all the conditions which give

rise to embolism or hæmorrhage. Syphilitic thrombosis is besides often associated with peripheral paralysis of one of the cranial nerves. It must be remembered that unilateral amaurosis may occur in syphilis from thrombosis of the central artery of the retina.

(*c*) *Chronic Degenerative Changes.*—In cases of this kind the course of the disease is more or less like that of general paralysis of the insane. The symptoms begin insidiously, with feelings of general uneasiness and discomfort, the health suffers, there is an unusual degree of mental irritability, or great mental activity interrupted by attacks of confusion of ideas. The patient commonly has ideas of grandeur, and may indulge in extravagance much beyond his means. In cases of the kind the syphilitic affection is liable to manifest itself by a fresh attack of constitutional symptoms in the throat, nose, or hands. After a time new symptoms make their appearance; the patient is easily fatigued, he is no longer capable of undergoing sustained exertion, the gait is staggering, and his movements are uncertain and hesitating. The patient complains of numbness, formication, and shooting pains in one of his limbs, the speech is hesitating, and stammering, the tongue trembles, and memory and intelligence decrease gradually and steadily. Various forms of paralysis now make their appearance; the gait is ataxic; the writing is irregular and ultimately becomes illegible; and after a variable period of years the patient dies with cystitis, bedsores and their consequences, unless carried off by some intercurrent affection.

§ 984. *Treatment.*—The treatment of syphilitic nervous affections must be prompt and energetic, inasmuch as delay may lead to irreparable injury being done to the part affected. If the presence of a gumma be suspected, the iodide of potassium should be at once administered in doses of a scruple to half a drachm three times daily. The iodide may from the first be combined with mercurial treatment, or the administration of the latter may be deferred until the former has had time to dissipate the gumma. The iodide, however, should never be trusted alone, as the lesion is likely to recur in some other situation within a period of a few months, unless mercury be

administered. In aggravated cases from three to four scruples of mercurial ointment should be rubbed daily over the lower and upper extremities, the abdomen, and back during the first fourteen days. If improvement be manifested at the end of this time, the same quantity should be rubbed in every other day for several weeks, and then half the quantity at the same intervals for several additional weeks. The mouth and teeth should be frequently washed during this treatment, in order to prevent salivation. In milder cases the internal administration of mercury is more convenient than the inunction, and equally efficacious, and no preparation can answer the purpose better than the bichloride.

In syphilitic epilepsy the bromide of potassium may be combined with the iodide, and to allay neuralgic and other pains morphia must be had recourse to. In the treatment of painful affections of the peripheral nerves chloral may, according to Dowse, be added to the solution of the iodide of potassium.

(V.) FEBRILE AND POST-FEBRILE NEUROSES.

§ 985. Numerous nervous disturbances are liable to occur in the course of or during convalescence from febrile diseases. The diseases which are most commonly accompanied or followed by nervous disorders are typhoid fever, the acute exanthemata, acute dysentery, pneumonia and pleurisy, nephritis and cystitis, acute rheumatism, intermittent fever, and, above all, diphtheria. The nervous disorders of febrile disease are generally the same as those which arise from other causes, and may affect the peripheral nerves, the spinal cord, or the brain.

(1) *Neurosis of Typhoid Fever.*—During the first stage of typhoid the patient may complain of cutaneous hyperæsthesia, which may involve a considerable portion of the limbs and trunk. Hyperæsthesia and neuralgiform pains are often present in the muscles of the limbs, neck, thorax, and abdomen. These pains are frequently associated with cutaneous hyperæsthesia; they cause severe suffering to the patient, and all movements tending to stretch the affected muscles are avoided. Anæsthesia, either complete or partial, and of variable distribution, may appear in the course of the fever, and may be present

as *anæsthesia dolorosa.* It may occur during convalescence, and prove rebellious to treatment. Neuralgic pains often occur during the first week of the fever in the region of the occipital, supra-orbital, or other nerve; during convalescence similar pains are apt to appear in the loins and along the vertebral column, or in the region of distribution of one of the branches of the brachial, lumbar, or sacral plexuses.

The muscles supplied by certain nerves may be paralysed in the course of typhoid fever. A case is reported by Eisenlohr in which the muscles of the left lower extremities supplied by the tibial and peroneal nerves were paralysed during a relapse of typhoid fever. Electrical examination of the affected muscles revealed the reaction of degeneration in them. Paralysis of the abductors of the glottis has been known to occur during convalescence and to render the operation of tracheotomy necessary. Paralysis sometimes assumes the paraplegic form. Cormack records a case in which paraplegia gradually supervened about the seventh week from the beginning of an attack of typhoid fever. The patient also had paralysis of the external rectus muscle of the left eye, and retention of urine, but ultimately recovered.

The central forms of myelitis are liable to supervene in the course of typhoid fever. Leudet observed a case in which acute ascending paralysis developed during convalescence from a mild attack, and proved fatal in six days. Another case is reported by Vulpian in which symptoms of acute myelitis supervened during convalescence from typhoid fever, and although the patient ultimately recovered, paraplegia and atrophy of the muscles persisted for a long time. Progressive muscular atrophy, ataxia, multiple sclerosis, and other chronic affections of the spinal cord may become established during convalescence from typhoid fever.

Cerebral disturbances not unfrequently occur during or subsequently to an attack of typhoid fever. A case of temporary aphasia is mentioned by Trousseau as occurring during convalescence from typhoid. Temporary hemiplegia may supervene after an attack of typhoid. In other cases there may be aphasia, delirium of various forms, or acute insanity.

(2) *Nervous Disorders of the Acute Exanthemata.*—The

rachialgia which ushers in an attack of smallpox is no doubt of nervous origin; it is often accompanied by considerable weakness or even complete paralysis of the lower extremities, the bladder being sometimes involved in the paralysis. The paralytic phenomena generally disappear with the rachialgia, but loss of motor power of a more durable character may supervene during convalescence. The paralysis is limited to certain groups of muscles or to one of the extremities, or may assume the form of paraplegia. Loss of motor power is often associated with sensory disturbances, and the affected muscles may undergo atrophy or contracture. Hemiplegia with aphasia or other severe cerebral symptoms may complicate variola (Rosenthal). Rubeola, scarlet fever, and erysipelas may be accompanied or followed by local or general paralysis, or hemiplegia with persistent aphasia. Infantile paralysis is very liable to occur in the course of the acute exanthemata.

Diphtheritic Nervous Disorders. — Paralysis occurs after diphtheria probably more frequently than after all the other acute diseases together. An interval of several days or weeks may elapse between the termination of the general disease and the appearance of the paralytic phenomena. Diphtheritic paralysis almost invariably begins in the muscles of the soft palate and pharynx; it is associated with anæsthesia of the affected parts and loss of faradic contractility in the muscles implicated. Motor paralysis is sometimes preceded by violent vomiting and retardation of the pulse, while death appears to be sometimes caused by paralysis of respiration (Landouzy). These phenomena seem to be the result of a primary irritation followed by paralysis of the vagus, and it is unnecessary to describe them in detail (§§ 270 to 282). I have known glycosuria to occur during convalescence from diphtheria, but I am unable to state whether the symptom was transitory or permanent. Paralysis may invade various other muscles, such as those of the vocal cords, tongue, and eyeballs, the muscles of accommodation being specially liable to be affected. The paralysis extends to the muscles of the trunk and extremities; it is rarely complete, but the bladder and rectum may be involved; Trousseau observed impotence in young men, lasting several weeks or months. Death has been known to result from

paralysis of the diaphragm. The sensory disturbances consist of hyperæsthesia at first, followed by numbness and anæsthesia. The sense of taste, smell, or hearing may occasionally be affected, and disturbances of vision are frequent. The disorders of sight may be caused by paralysis of some of the external or internal muscles of the eye; there may be complete amaurosis, which may continue during several weeks, no appreciable changes being present in the discs.

(3) *Paralysis of Acute Febrile Diseases.*—Acute rheumatism is sometimes followed by a local paralysis in the region of distribution of one of the peripheral nerves. Pneumonia and pleurisy are sometimes complicated by paraplegia or hemiplegia, but it is doubtful whether there is anything more than an accidental connection between the local disease and the nervous disorders. The forms of paralysis termed reflex have already been considered.

(4) *Nervous Disorders of Intermittent Fever.*—Paralysis sometimes appears suddenly during the febrile paroxysm, and ceases suddenly with it; at other times it constitutes the only evidence of the presence of malarial poisoning (pernicious paralytic fever), and in other cases it assumes a chronic form, and is associated with great cachexia. Of the forms of paralysis which occur suddenly, hemiplegia with aphasia is by far the most usual. In the pernicious paralytic variety paraplegia, with partial anæsthesia and disturbances of sight and hearing, is sometimes found associated with aphasia (Vincent).

§ 986. *Morbid Anatomy and Physiology.*—The changes which the muscles undergo in acute diseases have been carefully studied by Hayem, but this subject is much too wide to be discussed here. The local paralyses associated with anæsthesia occurring after acute diseases are doubtless of peripheral origin, and are probably caused by neuritis. Even in paraplegia the disease in the spinal cord is supposed by some authors to be secondary to that of the peripheral nerves, and the result of an ascending neuritis. In diphtheritic paralysis Charcot and Vulpian observed degeneration of the motor nerves of the velum palati. Buhl states that there is a nuclear exudation into the

sheaths of the paralysed nerves similar to the diphtheritic exudation occurring into the connective and mucous tissues. Pierret observed spots of false membrane on the spinal cord and medulla oblongata, associated with perineuritis of the roots of the corresponding nerves. Vulpian observed slight changes in the ganglion cells of the anterior horns in two cases, but found nothing abnormal in a third. Dêjerine found neuritis of the anterior roots and of the intra-muscular nerves, along with slight alterations in the grey substance of the cord; the posterior roots and white substances were normal. Westphal found spots of softening disseminated throughout the spinal cord in a case of paraplegia occurring in the course of smallpox. It is manifest that various and manifold lesions may be found in febrile and post-febrile nervous disorders affecting peripheral nerves, spinal cord, or brain. The nature of these lesions is probably very various. Sometimes the lesion consists of hyperæmia, at other times of anæmia, either general, or partial from occlusion of vessels, or it may be inflammatory or degenerative from the first.

§ 987. *Diagnosis and Prognosis.*—That a nervous disorder has occurred during the course or shortly after an acute disease is rendered evident by the history of the case. The chief problem of diagnosis then is to determine whether or not the lesion be functional or organic, or localised in the peripheral nerves, spinal cord, brain, or sympathetic system. In order to determine the latter question, it is necessary to possess an accurate knowledge of all the diseases, especially all the forms of paralysis, to which the nervous system is liable. No number of special diagnostic rules will supply the place of this knowledge, and such rules are superfluous to those possessing it. It is scarcely necessary to remind the reader of the value of electrical examination of the paralysed nerves and muscles in determining whether the paralysis be of peripheral, spinal, or cerebral origin.

The *prognosis* is, as a rule, favourable in the nervous disorders occurring in connection with acute diseases. In diphtheritic paralysis recovery usually takes place in some weeks, but in severe cases it may be delayed for six months and upwards. The

severity of the primary diphtheritic attack bears no proportion to the intensity and duration of the subsequent paralysis.

§ 988. *Treatment.*—The treatment must vary according to the nature of the lesion and other circumstances. If there be evidence of hyperæmia of the spinal cord or brain, cold application should be employed. As a rule, however, tonic and stimulating treatment is required. If there be no organic disease strychnia is indicated. The best results are obtained from electrical treatment, hydrotherapeutics, and change of air and scene.

INDEX.

ERRATA.

Volume I.

Page 41. Line 8th from the top,
,, Line 12th from the top,
,, Line 14th from the top, } for "centre" read "cortex."
,, 48. Line 16th from the top, for "cenereum" read "cinereum."
,, 48. Line 5th from the bottom, for "P" read "p."
,, 76. Line 11th from the bottom, for "hœmorrhidal" read "hæmorrhoidal."
,, 77. Line 2nd from the top, for "adventitæ" read "adventitiæ."
,, 104. Line 12th from the top, for "*æthesicæ*" read "*æsthesicæ.*"
,, 225. Line 11th from the bottom, for "cullus" read "callus."
,, 258. Line 11th from the bottom, for "becomes" read "become."
,, 449. Line 5th from the bottom, for "*Sternatatio*" read "*Sternutatio.*"
,, 496. Line 19th from the bottom, for "injection" read "injection of morphia."
,, 559. Line 21st from the bottom, for "nitrate" read "nitrite."

Volume II.

Page 62. Line 11th from the bottom, for "ducussating" read "decussating."
,, 168. Line 10th from the top, for "Cruvielhier" read "Cruveilhier."
,, 201. Line 1st at the top, for "Greisinger" read "Griesenger."
,, 214. Line 1st at the top, for "thirty" read "twenty."
,, 411. Line 11th from the top, for "variolli" read "Varolii."
,, 411. Line 16th from the top, for "Variolii" read "Varolii."
,, 427. Line 19th from the bottom, for "*of the Skull*" read "*to the Skull.*"
,, 434. Line 10th from the bottom, for "*corpora striatum*" read "*corpus striatum.*"
,, 558. Line 19th from the bottom, for "grammes" read "grains."
,, 641. Line 15th from the top, for "began the" read "began."

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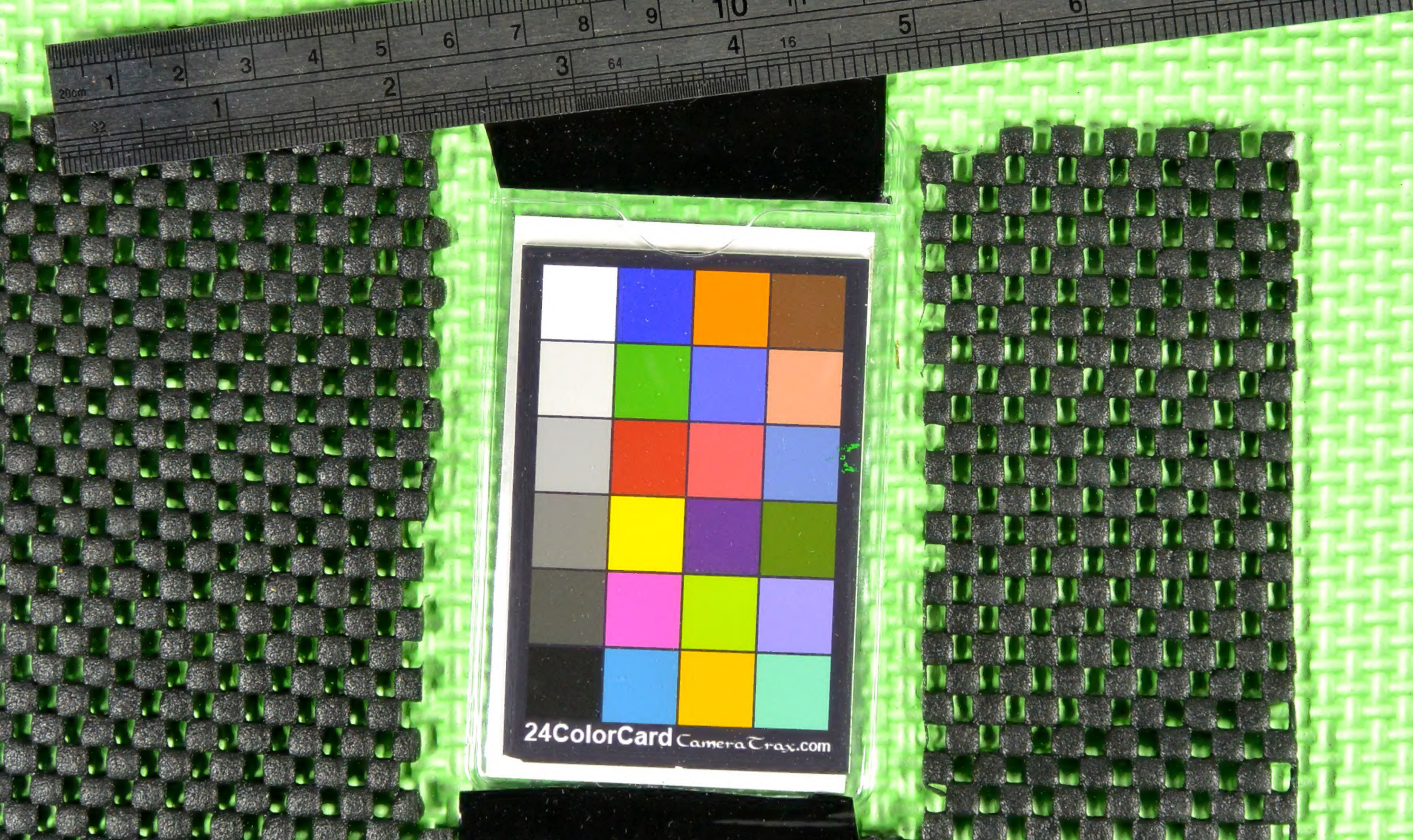
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