up in store in certain parts of the brain nascent or latent cells (granules) which may interpolate themselves among the depleted cells of older growth.

Education in its broadest sense includes all changes in the brain due to reactions of the organ upon afferent stimuli. It is usual to state that all the elements (cells, etc.) of the brain are preformed in it at birth. If this were so, then education would consist in the progressive modification of these cells and the perfection of a wide range of intercommunications between them. Leaving out of the account the possibility of the proliferation of new cells from a germinative epithelium, there can be no doubt that the latent cells above mentioned are called into activity by exercise, and increased brain power and a more extended range of activity are thereby secured. It would apparently follow that activity of mind would hasten the period of senility and brain decline; but as the reverse is the case, it may be assumed that proliferation or an analogous process really takes place.

C. L. Herrick.

BRAIN DISEASES: DIAGNOSIS OF LOCAL LESIONS.—History.—Although it was known in the first century that each hemisphere of the brain is in functional relation with the opposite half of the body, the facts upon which the prevailing theory of the localization of different functions in separate parts is based were not discovered until 1822. At that time Thomas Hood, in England, and Bouillaud, in France, noticed that disturbances of speech were caused by disease in the frontal lobes of the brain. M. Dax (1836) was the first to limit the area governing speech to the left frontal lobe, and Broca (1861) located it more exactly in the left third frontal convolution. The discussion of aphasia in the Academy of Medicine in Paris in 1864 awakened general interest and led to further investigation. Until that time scientific men, rejecting the unwarrantable conclusions of Gall and the phrenologists, had believed the teachings of Flourens, that the brain acts as a whole, its various parts not possessing various powers. The pathological evidence against this position collected by Broca, and strengthened during 1864–67 by facts observed by Hughlings Jackson and Meynert, received confirmation in 1870 from a new series of physiological experiments made by Fritsch and Hitzig in Berlin. These investigators found that in animals the anterior portion of the convexity of the brain is motor; that its irritation by electricity causes coordinated motions in the limbs of the opposite side, and that its destruction causes paralysis. Ferrier (1873–76), Nothnagel (1877), Munk (1881), and Luciani (1884) have confirmed these results, and have shown further that the posterior portion of the convexity is sensory, its destruction being attended by impairment of the powers of perception through the various senses. Goltz, though opposing a strict limitation of functions to definite regions, admits that the results of destruction of various parts are different, and he has noticed that extensive injury to the anterior portion changes the character of an animal from kind to vicious, while injury to the posterior portion has the opposite effect. The conclusions of physiologists differ regarding the results of experiments, but do not overthrow the theory of localization as applied to man; for a mass of pathological evidence has been collected during the past ten years which will bear but one interpretation. Charcot and his pupils in France, Nothnagel, Exner, and Wernicke in Germany, H. Jackson and Ferrier in England, and others, have gathered, classified, and analyzed a very large number of cases of brain disease of limited extent, which were accompanied by definite symptoms, and have established a causal relation between lesions of certain portions of the brain and disturbances of certain functions, both motor and sensory. It has also been discovered that deficient development of an organ is accompanied by deficient development of that part of the brain which is in functional relation with that organ, and *vice versa* (von Gudden).

Further, the researches of Flechsig (1877–84) have proven that an anatomical connection exists between certain organs and certain parts of the brain by means of tracts, which can be distinguished from one another by peculiarities in the time and process of their development. To these same tracts are limited the secondary changes which ensue when the active organ at one extremity of the tract is destroyed.

All these various kinds of evidence combine to establish the conclusion that definite parts of the brain possess distinct functions, and although there remain numerous functions whose location is unknown, and many parts of the brain whose function is undetermined, a sufficient number of facts is available to warrant in many cases of cerebral disease a localization of the lesion.

General Considerations.—Since the different parts of the brain preside over different functions, the symptoms present in any lesion will depend as much upon its situation as upon its nature. Certain general symptoms, such as headache, vertigo, convulsions, coma, or optic neuritis, occur in many forms of disease, and being indications of disturbances of nutrition, or of increased intracranial pressure, do not indicate the position of the disease. Other symptoms, however, such as disturbances of motion, of sensation, of sensory perception, of memory, or of speech, are known as local symptoms, since each is present only when a certain part of the brain is involved. It is from these that the localization of a lesion can be determined. Local symptoms must, however, be interpreted with caution, and the direct effect of the lesion must be distinguished from its indirect effect. For example, immediately after a cerebral hemorrhage, attended with headache, vertigo, or coma, and possibly general convulsions and vomiting, the local symptoms of hemiplegia, hemianæsthesia, and aphasia may be present, and may lead to the suspicion of a very extensive lesion. After a few days, however, there may remain only a partial hemiplegia, all other symptoms having subsided. In such a case the hemiplegia is the only direct local symptom; the indirect local symptoms—aphasia and hemianæsthesia—being incidental to the pressure on, or to disturbance of, circulation in parts adjacent to the actual seat of disease. It is only when a lesion is single, its effects stationary and of some duration, that a diagnosis of its position is to be made.

In diagnosticating the position of a lesion it is necessary to distinguish disease in the cortex from disease within the hemisphere. The functions of these parts are different. The gray cortex receives and initiates impulses. The white matter within the hemisphere transmits the impulses. The impulses sent along white tracts to the cortex become conscious perceptions only when they reach their destination in the gray matter. The impulses passing along the white tracts from the cortex have been started in the gray matter as conscious volitions by effort. Thus sensation or motion may be suspended either by disease in the cortex or by disease in the tracts within the hemisphere. The cortex has another function. A sensation once perceived, or a motion once acquired, leaves behind it a trace, whose nature is unknown, which shows itself in a disposition in the cells of the cortex to react more promptly to a similar impulse than to a dissimilar one. This is the physical basis of memory. Since similar impulses always enter by the same sensory organ, and since each organ is connected with its own region of the cortex, it follows that the various memories are distributed in various regions. But these memories are often associated in consciousness, and this association is secured by means of white fibres which pass between and connect the various regions. It becomes evident, therefore, that diseases of memory may afford an important clue to the location of a lesion; and that the distinction between a disease of the gray cortex involving a loss of a certain kind of memory, and one of the white tracts within the hemisphere interfering with the proper association of ideas must not be overlooked. No part of the gray matter can act vicariously for another part. Each tract conveys its own impulses.

Diagnosis.—1. Cortex Cerebri.—1. Lesions involving the frontal lobes upon the base may destroy the olfactory bulb or tract and produce anosmia on the side of the lesion. Lesions in the other convolutions of the frontal lobes, excepting those in the posterior part of the third convolution of the left side, present no distinctive local symptoms. Some disturbance of mental action, manifested by an inability to concentrate the attention, to think connectedly, and to control the emotions, or even by a condition of imbecility, may be caused by disease in this region.

These convolutions are often defective in idiots, and their comparative development in animals determines the mental power of the individual. But disease in this region in man does not cause a loss of any particular mental faculty, and for the higher powers of mind no location can be determined. Normal mental action implies the integrity of the entire brain. When general symptoms of cerebral disease are present, but no local symptoms can be found, the possibility of disease in the frontal convolutions is to be considered, and the occurrence of the mental disturbance mentioned affords a presumption in favor of this location.

Lesions in the posterior part of the third frontal convolution on the left side in right-handed, and on the right side in left-handed persons give rise to ataxic or motor

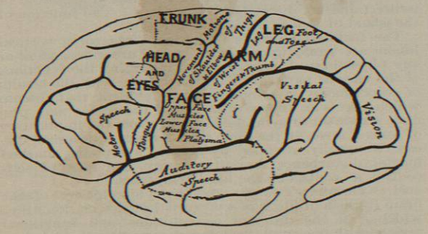

Fig. 870.—Diagram of the Fissures and Convolutions of the Convexity of the Left Hemisphere of the Brain, with the areas presiding over various functions. The speech areas are shown on this hemisphere. The motor area is more extensive on the left than on the right hemisphere.

aphasia (Fig. 870). In this area are located the memories of the combination of motor acts necessary to the pronunciation of words, memories which have been acquired by practice. If these memories are blotted out, the ability to initiate the impulse required to produce a given sound is lost, and speechlessness results. When this convolution alone is affected the patient can understand what is said to him, and may be able to write, but cannot talk (see *Aphasia*).

2. Lesions of the anterior and posterior central convolutions and of the paracentral lobule produce disturbances of motion (Fig. 870). The motor tracts which connect these convolutions of each hemisphere with the body decussate partially in the medulla, and the degree of the decussation differs in different individuals. In the large majority of persons the tracts which cross to the opposite side are so much larger than those which go to the same side that the symptoms of cerebral disease are noticed only on the side of the body opposite to the side of the lesion. In all cases, however, except in those in which the decussation is complete (one in sixty), the side which is apparently normal is slightly affected. The disturbances of motion may be in the form of spasms and convulsions, or in the form of paralysis. Lesions irritating the motor region give rise to the former; those which destroy the cortex to the latter. The lower third of the anterior central convolution is in functional relation with the muscles of the face and tongue (Fig. 870). The middle third of both central convolutions governs the arm (Fig. 870), the motions of the shoulder, elbow, and hand lying from before backward and from above downward in the order named. The upper portion of both convolutions and the paracentral lobules contain the motor centres for the body and leg, the motions of the hip, knee, and foot lying from before backward and from above downward in the order named. The area related to the movement of the eyes is located by Landouzy and Exner in the inferior parietal lobule. As these areas for each part are distinct, cortical lesions of limited extent may affect one alone, or two adjacent areas; but it is only lesions of very great extent which can destroy them all. Monospasms, or monoplegia, are, therefore, prominent symptoms in disease of the motor region. An irritation beginning in one area may extend to adjacent areas, in which case a convulsion may commence in one part and then involve other parts. The relative position of the areas, then, determines the order of progress of the convulsion, face, arm, and leg being successively affected, or *vice versa*; and face and leg never being involved together without affection of the arm. When the entire side is involved the convulsion may become general. The seat of the initial irritation may, therefore, be indicated by the order in which the spasms extend. After such a spasm there remains a paresis in the muscles affected, those last and least involved recovering first (see *Epilepsy*). If the irritating lesion becomes a destroying lesion the monospasm is succeeded by monoplegia, and from the part of the body affected the area in the motor region which is destroyed can be determined. In cortical disease it is seldom that the lesion involves a single area without encroaching upon adjacent areas; hence, associated monoplegiæ of face and arm, or arm and leg, are more frequently met with than paralysis of one part alone. But even in these cases the disturbance of motion usually begins or is more marked in one part, rather than in both equally, and the order of extent of paralysis may indicate the direction in which the disease is progressing, and the place from which it started.

In paralysis from cortical lesion there is a loss or marked impairment of the muscular sense, and there may be some disturbance of general sensation. A loss of motor memories, *e.g.*, the motions involved in writing, playing an instrument, using a tool, occurs in cortical disease, and may indicate that the seat of the lesion is in the area of the arm. The limits of the region receiving impulses which awaken the perception of touch, temperature, and pain are not fully determined. It is thought, however, that the motor and sensory regions coincide, while it is probable that the sensory region extends beyond the motor and includes the parietal lobules which lie posterior to the motor area. Ferrier, however, teaches that the gyrus hippocampus is the region in which these sensations are received. Lesions affecting the posterior central convolution give rise to combined motor and sensory symptoms, the sensory areas lying in the same order as the motor areas, face, arm, and leg in the lower, middle, and upper thirds respectively. Lesions in the motor area anterior to the fissure of Rolando usually produce paralysis without anæsthesia. Lesions in the parietal lobules may produce anæsthesia but do not cause paralysis.

Each sensory area is in functional relation with the opposite limb to a much greater degree than with the limb of the same side. Monoanæsthesia may therefore occur from cortical lesion. The loss of sensation is rarely total, as it is probable that the decussation of sensory impulses is rarely complete. The degree of impairment of sensation is to be ascertained only by comparison of the affected limb with the other three. If the sensory area is not destroyed but is only irritated, subjective sensations in the limb whose area is affected occur, and such monoparæsthesiæ are valuable indications of cortical lesion, when disease in other parts is excluded.

Monospasm and monoplegia, monoparæsthesia or monoanæsthesia, are therefore the chief symptoms of cortical disease in the sensorimotor area. The two former indicate an affection lying anterior to the parietal lobules. The two latter may occur when these also are involved. No other local symptoms of disease in the parietal lobules are known, the disturbances of speech or

of sight which occur occasionally when the supramarginal gyrus is involved being due to a coincident lesion of the tracts passing beneath it.

3. Lesions of the three occipital convolutions and of the cuneus produce disturbances of vision (Fig. 871, D). Each occipital lobe is in anatomical connection with the like-named half of each retina, and hence a lesion of one occipital lobe produces an affection of vision in the opposite half of both visual fields. Irritation of the cortex of this region may cause hallucinations of vision, and if the irritation is in one lobe the subjective sights will appear upon the opposite side of the median line, and will move with the eyes of the patient. Destruction of the cortex will produce bilateral homonymous hemianopsia, *i.e.*, blindness in the opposite half of both eyes, and may also cause a loss of visual memories; the patient will fail to recognize familiar objects, and cannot recall scenes and faces formerly known. If the lesion is in the left occipital lobe near to its junction with the inferior parietal lobule (see Fig. 870), written and printed language also may no longer be recognized, and the patient, therefore, may lose the power of reading while speech remains. It is not yet possible to affirm the functional relation between various parts of the visual area in the occipital convolutions and various parts of the retinal surfaces, although a few cases indicate that such a relation may exist. The relation of the angular gyrus, which lies anterior to the occipital lobe, to vision is undetermined. While it was formerly supposed to be the centre of the visual area by Ferrier, this view has been opposed by German authorities, who hold that the disturbances of vision which have occurred occasionally when it was diseased have been due to a lesion of the visual tract which lies beneath it and which passes to the occipital lobe (see *Hemianopsia*).

4. Lesions of the first and second temporal convolutions produce disturbances of hearing, but whether each lobe is related to the opposite ear alone, or to both ears, is undetermined (Fig. 871, E). Irritation of these convo-

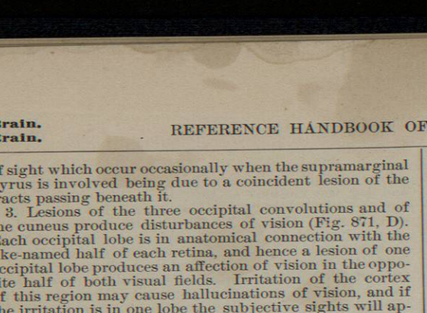

Fig. 871.—The Projection Tracts Joining the Cortex with Lower Nerve Centres. Sagittal section showing the arrangement of tracts in the internal capsule. A, Tract from the frontal lobe to the pons, thence to the cerebellar hemisphere of the opposite side; B, motor tract from the central convolutions to the facial nucleus in the pons and to the spinal cord; its decussation is indicated at K; C, sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe; D, visual tract from the optic thalamus (O.T.) to the occipital lobe; E, auditory tract from the internal geniculate body (to which a tract passes from the VIII. nerve nucleus [J]) to the temporal lobe; F, superior cerebellar peduncle; G, middle cerebellar peduncle; H, inferior cerebellar peduncle; CN, caudate nucleus; C.Q., corpora quadrigemina; Vt., fourth ventricle. The numerals refer to the cranial nerves.

lutions may cause hallucinations of hearing, and destruction of them may cause deafness. If the lesion is upon the left side and involves the first convolution, the symptom produced is word-deafness, or loss of memory of the sound of words, with consequent inability to recognize the meaning of spoken language or to recall the words necessary to speech. This is also known as sensory or amnesic aphasia. It can be distinguished from motor or ataxic aphasia by the inability of the patient to understand what is said to him (see *Aphasia*).

5. Lesions at the apex of the temporo-sphenoidal lobe may produce disturbances of taste and smell, but further investigations are needed to establish the localization of these functions.

6. Lesions of the island of Reil have caused disturbance of motion in the face and arm of the opposite side, and also have caused aphasia when the left island was involved. It is not certain, however, to what extent these symptoms were dependent upon the affection of adjacent convolutions, of tracts beneath the island, or of the basal ganglia (Figs. 872 and 873). The associating tract which joins the sensory with the motor speech centres lies just under the convolutions of the island of Reil and destruction of this tract would cause paraphasia. Lesions of the island of Reil would be especially liable to affect the circulation in the motor area, since the branches of the middle cerebral arteries pass over it. While, therefore, it can be stated that hemiplegia and aphasia may be produced by lesions here, it is probable that they would be indirect and not direct local symptoms. The function of the island of Reil is unknown.

The facts which have been stated regarding cortical lesions are based upon autopsies and are independent of any physiological considerations regarding the functions of the various parts, although they agree with the results of experiment on animals. It is evident that disease which affects an extensive region of the cortex may produce a number of symptoms arising from the implication of several areas at the same time. Such widespread disease is usually attended by general symptoms, marked mental disturbance, loss of memory, and lack of self-control, as well as by the local symptoms.

II. Cerebral Tracts.—1. The white matter of the brain consists of fibres of two varieties: association fibres which join the different convolutions and functional regions with one another, and projection fibres which join the different convolutions with the basal ganglia and with the gray matter of the pons, medulla oblongata, and spinal cord. These fibres are so interlaced in the centrum ovale that neither can be injured without affecting the other. An interference with the passage of impulses through association fibres produces symptoms of a mental character. An example of this has been cited in describing lesions of the island of Reil, viz., paraphasia. The patient suffering from this disease can recall the desired words, and is able to initiate the motions necessary to speech; but the associating tract between the memory of a definite word and the memory of its motion being broken, he does not speak the desired word but replaces it by another. Thus, in a case known to the writer, whenever the patient spoke she said, "Ah, dear me, I don't know," much to her own annoyance, as she understood what was said to her and knew what she ought to say in reply. The forms of paraphasia are numerous, but are all to be referred to lesion of association fibres. It is very probable that many defects of memory, and much of the apparent stupidity in brain diseases, is to be ascribed to a failure of function in these fibres by which ideas are associated. Nothing more definite can be stated regarding their injury, and the mental symptoms, aside from paraphasia, do not indicate the location of the disease.

2. Lesions of projection fibres produce well-marked local symptoms. It is by means of these fibres that all parts of the body are joined to connecting parts of the brain, so that in imagination a map of the body can be projected upon the cortex of the brain. An interference with any separate bundle of fibres will therefore produce symptoms in the organ with which it is joined, and therefore will cause effects somewhat similar to those produced by a lesion of the corresponding part of the cortex. It is necessary to consider the lesions of the various tracts,

as they produce different symptoms. The projection fibres passing inward and downward from the extensive cortical surface of each hemisphere converge, and are

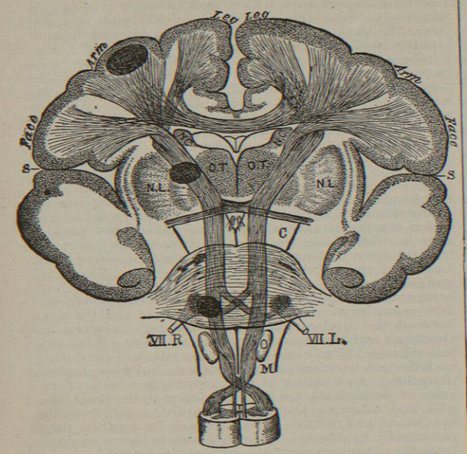

Fig. 872.—The Motor Tract. S, Fissure of Sylvius; N.L., lenticular nucleus; O.T., optic thalamus; N.C., caudate nucleus; C, crus; P, pons; M, medulla; O, olivary body. The tracts for face, arm, and leg gather in the capsule and pass together to the lower pons, where the face fibres cross to the opposite VII. nerve nucleus, while the others pass on to the lower medulla where they partially decussate, to enter the lateral columns of the cord; the non-decussating fibres pass to the anterior median columns. The effect of a lesion situated at three points in the tract is shown on the left side of the figure.

finally collected into a compact tract which lies between the basal ganglia and is known as the internal capsule. Many fibres pass into the basal ganglia, but as their function is only conjectured, the effect of their destruction is unknown. Others pass between the ganglia, through the capsule, and issuing from its basal portion enter the crus cerebri, and traversing it go down to the pons, medulla, and cord.

Since the fibres passing through the capsule are gathered from distant and widely separated regions of the cortex, a small focus of disease in the capsule may produce as serious and widespread symptoms as disease of great extent in the cortex. Extensive disease in the cortex, or in the centrum ovale, produces considerable mental impairment, but this is not true of capsular disease. Hence in any case in which the symptoms are extensive, but the mind unimpaired, the probability is in favor of a small lesion in the brain tracts rather than of a large lesion in the cortex. The brain tracts in the capsule may be injured either directly, by lesions in their course, or indirectly, by lesions in the basal ganglia in whose vicinity they pass. In both cases the initial symptoms will be the same if the disease begins suddenly; but in the latter case recovery may follow, while in the former the symptoms may increase in number, owing to secondary degenerations following a lesion of a tract. If the disease is a slowly progressive one (*e.g.*, tumor), general symptoms may precede local symptoms, and indirect local symptoms will be succeeded by direct local symptoms.

The internal capsule is divided into two halves, an anterior and a posterior division, by the projection of the apex of the lenticular nucleus, which lies on its outer side. Through the anterior division pass the projection fibres from the frontal lobes (Fig. 871, A), and the fibres which join the anterior parts of the cortex with the basal ganglia. Nothing is known as to the exact function of either of these bundles of fibres, and no symptoms of their injury can be stated. Extensive lesions may occur in the white matter of the frontal lobes, affecting both association and projection tracts without producing any symptoms, although in many such cases the symptoms present in cortical lesions of the frontal lobes do occur. Through the posterior division of the internal capsule pass several important tracts. These are (1) the motor tract from the lower third of the central convolutions, which curves over the lenticular nucleus and passes down in the anterior part of the posterior division of the capsule, enters the second quarter of the crus, thence passes into the median part of the ventral half of the pons, and turning downward ends in the facial and hypoglossal nuclei (Fig. 874, *Fa.*); (2) the motor tract from the other thirds of the central region (Fig. 871, B), which is known as the pyramidal tract, since its fibres, after passing through the third quarter of the crus and the ventral portion of the pons, make up the pyramid of the medulla (Fig. 874, *M.*); (3) the tract conveying general sensations from the entire body (Figs. 871, C, 874), which lies just behind the pyramidal tract, and having come from the tegmentum of the crus, and passed through the posterior third of the capsule, radiates toward the parietal convolutions (Fig. 874, *S.*); (4) the visual tract (Fig. 874, D), whose fibres, issuing from the pulvinar of the thalamus, pass upward and turn backward in the capsule on their way to the occipital lobe (Fig. 874, *O.*); (5) the auditory tract, which passes through the lower posterior segment of the capsule on its way from the auditory nucleus to the temporal lobe (Fig. 871, E). Lesions of the internal capsule which affect one or more of these tracts cause distinct local symptoms. Lesions in the motor tract produce paralysis, whose distribution will depend upon the extent to which the tract is involved. Usually the entire tract is affected and hemiplegia results; but occasionally the facial and hypoglossal muscles are only slightly affected. In all cases the upper branch of either facial nerve escapes, since its cortical centres are bilateral. In lesions of the pyramidal tract the arm and leg of the opposite side are always paralyzed together. Lesions lying in the sensory tract cause hemianæsthesia, which is rarely absolute, but usually in the form of great impairment of sensation on the opposite side from the lesion. Lesions lying still farther back may affect the visual tract and cause blindness in the opposite half of both eyes. It is

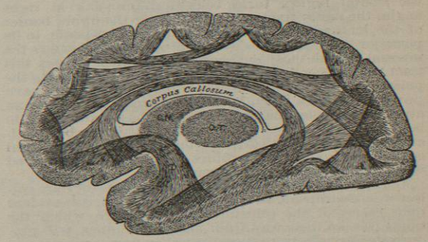

Fig. 873.—The Association Fibres in the Centrum Ovale. A, Between adjacent convolutions; B, between frontal and occipital lobes; C, between frontal and temporal lobes, the cingulum; D, between temporal and frontal lobes—lesion of this tract causes paraphasia; E, between occipital and temporal lobes—lesion of this tract causes word-blindness; C.N., caudate nucleus; O.T., optic thalamus.

possible that lesions in the lower part of the capsule may produce loss of hearing in the opposite ear. Whether taste and smell are ever affected by capsule lesions is undetermined.

It is evident that an extensive lesion in the capsule may

involve two or more of these tracts; and hemiplegia, with hemianæsthesia and hemianopsia, could not be produced by a single lesion lying in any other part. The initial effects of a hemorrhage or thrombosis, which involves the internal capsule, may be extensive, as several tracts may be affected. If but one, however, is really destroyed, the affection of the others will be temporary, and the symptoms will decrease in extent and severity until they are limited to the affected tract. It is only after the temporary effects have subsided that the disease can be located accurately. On the other hand, a slowly progressing lesion, tumor, or abscess may involve one tract after another in succession, and the progress of the case will determine the localization of the lesion.

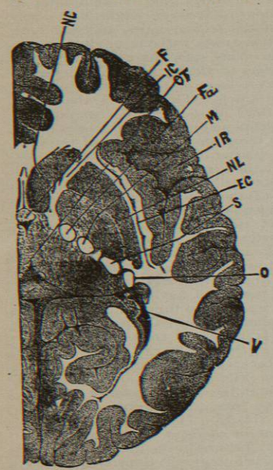

FIG. 874.—Horizontal Section through the Brain. (After Strümpell.) *NC*, nucleus caudatus; *F*, fornix; *IC*, anterior half of internal capsule; *OT*, optic thalamus; *Fa*, facial tract; *M*, motor tract; *S*, sensory tract; *O*, visual tract, in posterior half of internal capsule; *IR*, island of Reil; *NL*, nucleus lenticularis; *EC*, external capsule; *V*, lateral ventricle, posterior cornu.

3. Aphasia may be due to a lesion in the tract which joins the third frontal convolution with the facial and hypoglossal nuclei in the pons. Two courses have been described as taken by this tract. One corresponds to the course of the fibres from the face area of the motor region through the internal capsule, already detailed. The other is as follows: The aphasic tract passes inward from the frontal region, turns backward, crossing at right angles the motor tract, and going along the upper border of the lenticular nucleus, in the external capsule, to its posterior limit, it curves over the nucleus and enters the internal capsule near the sensory tract, then crosses this tract to the second quarter of the crus, and so reaches the pons. Lesions in the course of either of these tracts have produced aphasia when in the left hemisphere. Such an aphasia resembles ataxic or motor aphasia, and is not to be distinguished from it. It is more liable to be associated with agraphia than when the lesion is in the frontal convolution, as association fibres are likely to be destroyed when the lesion is in the centrum ovale.

4. Lesions of the cortex and of the tracts as they are collected in the capsule having been considered, it is evident that lesions of the centrum ovale, through which the tracts pass on their way from one to the other, will produce similar effects to those in either part, according as it lies nearer one or the other. The association fibres which pass through the centrum ovale are shown in Fig. 873. Any interference with them will produce defects in the association of concepts and ideas, and hence be shown by mental defects, especially defects of memory. Lesions of the centrum ovale have no distinctive symptoms by which they can be differentiated from lesions in the cortex or capsule, unless the affection of association fibres as well as of projection fibres adds distinct mental symptoms. If the lesion is so situated as not to affect any of the projection tracts it will not produce any local symptoms.

III. LESIONS OF THE BASAL GANGLIA are very frequent and give rise to many symptoms. These symptoms are, however, to be ascribed to a coincident affection of the tracts just mentioned, which pass through the internal capsule between the ganglia (Figs. 874 and 875), and hence the local symptoms of lesions in the lenticular or caudate nucleus and optic thalamus are those of lesions of the internal capsule opposite those bodies, viz., in its posterior division. If the symptoms are permanent, the capsule is probably injured. If the symptoms pass away, the capsule was incidentally affected. And the effects of the lesion may entirely subside while the lesion remains, if it is limited to either of these ganglia. It is therefore evident that we cannot locate a lesion in the ganglia from any direct local symptoms; for, as we are ignorant of the function of the ganglia, we do not know what is the effect of their destruction. The hemichorea and hemiathetosis which occasionally remain after lesions of the thalamus are to be regarded as symptoms of irritation constantly exerted upon the motor tract passing near it. There are few facts to support the assertions that the optic thalamus is a sensory ganglion, except that lesions of the pulvinar cause hemianopsia. The basal ganglia, doubtless, have important reflex functions, and comparative anatomy indicates that some functional relation exists between the lenticular nucleus and the arm; between the caudate nucleus and the leg; between the thalamus and the organs of sensation. The effects of a lesion limited to any one of these ganglia, and producing no influence upon adjacent parts, are, however, beyond detection. As a matter of fact, seventy per cent. of the cases of hemiplegia are due to a lesion in the basal ganglia and affecting the internal capsule; and from the symptoms and nature of the disease its location there can usually be affirmed. The diagnosis is, however, made from the capsular symptoms, as already detailed.

IV. LESIONS OF THE EXTERNAL CAPSULE AND OF THE CLAUSTRUM cannot yet be located (Figs. 872 and 874). If on the left side, they usually produce paraphasia like the lesions of the island of Reil.

V. LESIONS OF THE CORPORA QUADRIGEMINA are very rare. If the anterior pair is involved, blindness, loss of pupil reflex, strabismus, and nystagmus may be produced. If the posterior pair is involved disturbances of coordination may be marked. As both pairs are usually affected together, the combination of these symptoms may aid a diagnosis. Blindness is such a common symptom of cerebral disease that it is only when it is not due to choked disc, optic atrophy, or neuritis, and when it is not of the nature of hemianopsia, that it is to be thought a local symptom of quadrigeminal lesion. A defective action of the same branches of the oculo-motor nerves on both sides is rather more characteristic of quadrigeminal disease than the total affection of one nerve. Cases are yet too few to warrant more accurate statements.

VI. LESIONS OF THE TEGMENTUM OF THE CRURA CEREBRI, which lies beneath the corpora quadrigemina. Since the sensory tracts pass through this region, anæsthesia may be produced by such a lesion, and the proximity of the corpora quadrigemina would give rise to indirect local symptoms of their affection. Lesions of the red nucleus cause the symptom of incoordination which occurs when the posterior pair of the corpora quadrigemina are involved. They also cause paralysis of the third nerve, which passes through this nucleus. Lesions of the foot of the crus cerebri, in which the motor tract passes, cause hemiplegia of the opposite side. As the third nerve issues through the foot of the crus, a lesion here causes a paralysis of this nerve on the side of the lesion. Hence hemiplegia of one side, with third-nerve paralysis of the other side, indicates a lesion of the foot of the crus cerebri on the side of the third-nerve paralysis. Lesions on the base which press upon this part will produce the same combination of symptoms.

VII. LESIONS OF THE CEREBELLUM, if located in the hemispheres and not in the median or vermiform lobe, and if of such a nature as not to exert pressure on surrounding parts, may not produce any symptoms. If

the lesion is in the vermiform lobe, disturbances of coordination known as cerebellar ataxia occur, which consist in an inability to walk without staggering like a drunken man. The ataxia exists only while the patient is in an upright position; it rarely affects the motions of the arms, and when it does it never interferes with the fine adjustments, but only with extensive movements in space, *e.g.*, grasping objects at a distance. These patients can stand with eyes closed without swaying. In these respects the ataxia differs from that of posterior sclerosis. A second characteristic symptom of cerebellar disease located in the vermiform lobe is vertigo. This may be very severe, but as it may occur without ataxia, and ataxia may be present without vertigo (though rarely), the two are not to be considered as interdependent. Vertigo is increased by rising to an erect position, but may persist when the patient is in bed. It decreases somewhat when the patient has remained fixed in any position for some time; but is always increased when he closes his eyes. The vertigo is usually an early symptom of cerebellar disease. It is more constant and persistent in cases in which the intracranial pressure is increased. It may gradually pass off in other cases. Cerebellar vertigo does not differ from vertigo in Ménière's disease, and is probably due to an affection of the terminal fibres of the eighth nerve, from the semicircular canals or their nuclei. The vertigo of Ménière's disease is, however, usually accompanied by deafness. Ataxia and vertigo together afford strong presumption of disease in the vermiform lobe, although neither alone is sufficient for a diagnosis.

The indirect local symptoms of cerebellar disease may be numerous. They are due to an affection of the tracts and nerve nuclei in the pons and medulla (see Fig. 871). Various forms of paralysis and anæsthesia, vaso-motor disturbances, obstinate vomiting of a projectile character, general symptoms of intracranial disease, *e.g.*, headache, optic neuritis, are usually present with tumors, abscesses, or hemorrhages in the cerebellum, especially if they are in the median lobe. The combination of cerebellar ataxia and vertigo with these and other symptoms of pons disease (see Section IX.) affords clear evidence of disease in the cerebellum.

Lesions of the cerebellum have no apparent effect upon the mental powers when they occur in adults. A deficient development of the cerebellum is, however, a frequent cause of congenital idiocy. When one cerebellar hemisphere fails to develop, the opposite olivary body in the medulla and the opposite hemisphere of the cerebrum usually present an atrophic appearance.

Lesions of the middle peduncles of the cerebellum, the crura cerebelli ad pontem, produce characteristic symptoms. These consist in a tendency on the part of the patient to assume a forced position, to turn toward or fall toward one side in walking, or even to revolve constantly about one axis of his body. The forced movements may be made by the eyes (conjugate deviation in one direction), by the head, or by the entire body. In a case seen by the writer, in which the autopsy showed a tuberculous tumor in the left middle peduncle and in the vermiform lobe, in addition to ataxia, vertigo, vomiting, and headache, the patient lay constantly on his left side, and when he turned upon his back or toward the right side the vertigo became so excessive that he was obliged to resume at once his former position. In walking, this patient showed a tendency to fall toward the right side, and found it impossible to turn around toward the right. Such patients may lose their balance in moving in one direction, *e.g.*, forward or backward, and in attempting to regain it they may be obliged to hasten their movements. This has been interpreted wrongly as a tendency to compulsory walking in one direction, *e.g.*, backward. It is really due to the vertigo. Lesions of the other peduncles of the cerebellum do not produce any known characteristic symptoms aside from those of cerebellar disease.

VIII. LESIONS UPON THE BASE OF THE BRAIN, tumors, chronic meningitis, may produce local symptoms by affecting the parts lying near them. Thus, all the cranial nerves may be involved either separately or in various combinations, and the nerve first or most severely affected may indicate where the lesion began. If such a lesion affects the optic nerve of one side, the optic chiasm, or the optic tract, blindness, either in the form of amaurosis of one eye or in that of hemianopsia of some variety, will be produced. If the lesion affects the crus cerebri or pons, the symptoms of lesion of this part (*vide supra* and Section IX.) may be present in addition to cranial-nerve paralysis. In all cases progressive bulbar paralysis is to be excluded before the diagnosis is made. The local symptoms of lesion of those parts of the cortex which lie on the base of the skull are undetermined, but a tumor of some extent which invades the left Sylvian region may cause aphasia from pressure upon the island of Reil. The variety of symptoms possible in basal disease can be determined by the study of the organs upon the base of the brain (see *Brain*), and any unusual combination of symptoms in intracranial diseases, especially if of syphilitic origin, should lead to a question as to the possibility of a lesion in this locality.

IX. LOCAL LESIONS IN THE PONS VAROLII AND MEDULLA OBLONGATA.—The pons Varolii and medulla ob

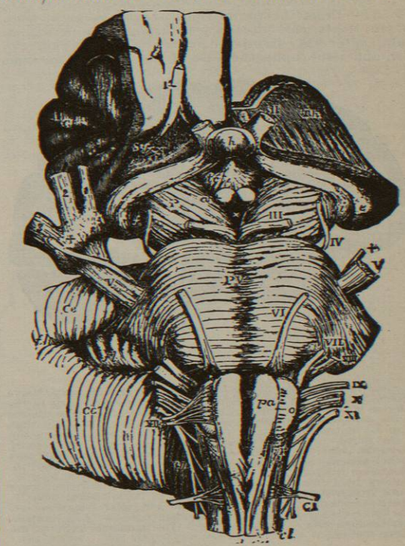

FIG. 875.—The Base of the Brain and the Cranial Nerves. Pons and Medulla. *I* to *XII*. The cranial nerves; *Th*, optic thalamus; *h*, pituitary body; *tc*, tuber cinereum; *a*, corpora albicantia; *P*, pes pedunculi; *i*, internal, and *e*, external, geniculate body; *PV*, pons Varolii; *pa*, anterior pyramid of medulla; *o*, olive; *d*, decussation of anterior pyramids; *ea*, anterior column of spinal cord; *cl*, lateral column of spinal cord; *Cc*, cerebellum; *fl*, flocculus of cerebellum.

longata lie upon the base of the brain, between the cerebrum and its crura above, the spinal cord below, and the cerebellum behind. Through them pass important tracts which join these organs with one another, and by means of which motor, sensory, vaso-motor, and trophic impulses are sent from the brain to the body, and from the body to the brain. Diseases in the pons and medulla cause symptoms of wide extent and serious nature, by interfering with the transmission of these impulses. The pons and medulla are not, however, merely organs of

transmission. They contain centres of great importance, and preside over numerous functions. In them are located the nuclei of origin of the cranial nerves from the fifth to the twelfth inclusive, and through them the cranial nerves pass outward from these nuclei to the surface. In them are also situated complex automatic centres, which preside over the acts of combined movement of the eyeballs, deglutition, articulation, respiration, and cardiac inhibition, and which regulate the tone of the entire vaso-motor system, and the secretion of saliva, perspiration, and urine. Disease in the pons and medulla, by affecting these various centres, produces important symptoms, and the complexity of those symptoms is evident from the number of normal functions which will be interfered with in any case of extensive lesion. These functions and their affections must be considered separately.

1. *The Tracts Passing through the Pons and Medulla.*—These may be studied by the aid of a schematic drawing (Fig. 880), and by comparing a number of sections made at different levels through the pons and medulla (Figs. 877 to 888).

(1) *Tracts Connecting the Cerebrum and Cerebellum* (Fig. 876, A).—From the frontal lobe of each cerebral hemisphere tracts pass downward through the corona radiata, enter the anterior division of the internal capsule, and issue upon the base of the brain in the inner

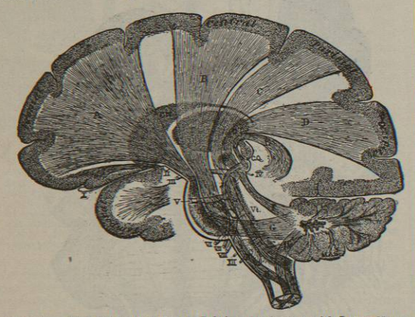

FIG. 876.—The Projection Tracts Joining the Cortex with Lower Nerve Centres. Sagittal section showing the arrangement of tracts in the internal capsule. A, Tract from the frontal lobe to the pons, thence to the cerebellar hemisphere of the opposite side; B, motor tract from the central convolutions to the facial nucleus in the pons and to the spinal cord; its decussation is indicated at K; C, sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe; D, visual tract from the optic thalamus (O.T.) to the occipital lobe; E, auditory tract from the internal geniculate body (to which a tract passes from the VIII. nerve nucleus [*d*]) to the temporal lobe; F, superior cerebellar peduncle; G, middle cerebellar peduncle; H, inferior cerebellar peduncle; CN, caudate nucleus; C.Q., corpora quadrigemina; Vt., fourth ventricle. The numerals refer to the cranial nerves.

third of the pes pedunculi (Fig. 877, *p*). From the parietal, occipital, and temporal lobes of each hemisphere tracts pass downward through the corona radiata, enter the posterior division of the internal capsule, and issue upon the base of the brain in the outer third of the pes pedunculi (Fig. 877, *p*). A section through the crura cerebri at the level of the anterior corpora quadrigemina (Fig. 877), shows the pes pedunculi ($p+p'$) separated from the tegmentum by the substantia nigra (*sn*).

The outer and inner thirds of each pes (exclusive of the median fasciculus p^1) are made up of these tracts from the cerebral lobes. After passing through the pes these tracts enter the pons Varolii, where they are split up into small bundles by the transverse fibres of the pons. Sections through the pons Varolii at various levels (Figs. 878 to 883) show that the basal half of the pons consists of longitudinal and transverse bundles of fibres interlaced, and that considerable masses of gray matter, consisting of large polygonal cells, lie between the fibres. It is in these gray masses that the longitudinal bundles

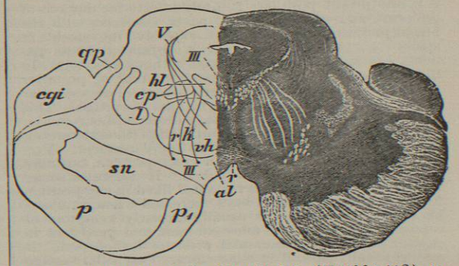

FIG. 877.—Section through the Crura Cerebri. (Wernicke, × 2.) *p*, Pes pedunculi; p_1, median fasciculus; *sn*, substantia nigra; *cgi*, corpus geniculatum internum; *qp*, corpus quadrigeminum posterior; *l*, lemniscus; *rk*, red nucleus of tegmentum through which pass fibres of oculomotor nerve, *III.*, from its nucleus in the gray matter below the aqueduct of Sylvius, to its exit between the crura; *cp*, fibres in the formatio reticularis which have come from the posterior commissure; *hl*, posterior longitudinal bundle; *V*, descending root of the trigeminal nerve; *vh*, upper part of the decussation of the superior peduncles of the cerebellum, which end in the *rk*; *r*, raphé; *ar*, lenticular loop.

under consideration from the cerebral lobes terminate. Hence, in the sections through the lower portion of the pons (Figs. 882 and 883), fewer longitudinal bundles are seen than in the upper sections. From the masses of gray matter in the pons, the transverse fibres originate and pass laterally to the cerebellum. The majority of the fibres from one-half of the pons pass to the opposite hemisphere of the cerebellum, and thus cross the median line, where they interlace with those of the other side. The gray masses of the pons are thus interposed in a continuous tract between the cerebrum and the cerebellum;

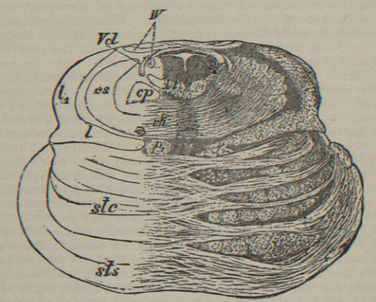

FIG. 878.—Section through the Pons. (Wernicke, × 2.) *sts* and *stc*, Superficial and deep transverse bundles of fibres intersecting the longitudinal bundles, and terminating in the masses of gray matter of the basal portion; p_1, median fasciculus of the pes; *l*, lemniscus; l_2, lower lemniscus; *rk*, red nucleus of tegmentum; *es*, superior peduncle of cerebellum, some fibres of which are decussating in the raphé; *cp*, fibres in formatio reticularis, which have come from the posterior commissure; *hl*, posterior longitudinal bundle; *Vd*, descending root of the fifth nerve; *IV*, fourth nerve decussating in the valve of Vieussens.

each cerebral hemisphere being connected with both cerebellar hemispheres, but to a far greater extent with the opposite hemisphere than with the one upon the same side.

Lesions of the frontal, parietal, and occipital lobes, if extensive, are followed by degeneration in the tracts from these lobes to the masses of gray matter in the pons.

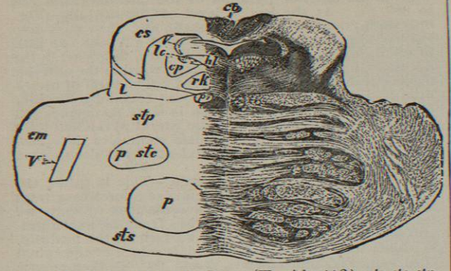

FIG. 879.—Section through the Pons. (Wernicke, × 2.) *sts*, *stc*, *stp*, Superficial and deep transverse bundles of the pons, intersecting the longitudinal bundles, and terminating in the mass of gray matter of the basal portion; *p*, pyramidal (motor) tract from the middle third of the pes; p^1, median fasciculus of the pes, here seen to join the lemniscus, *l*; *V*, fifth nerve root; *cm*, middle peduncle of cerebellum; *cs*, superior peduncle of the cerebellum with *cb*, valve of Vieussens; *rk*, *hl*, *cp*; *V*, as in Fig. 878; *lc*, locus ceruleus.

Congenital absence of the cerebral cortex (Porencephalie) results in congenital absence of these tracts. Absence or atrophy of the cerebellum is attended by absence or atrophy of the transverse fibres of the pons, which join the cerebellar hemisphere with the gray masses of the pons. Congenital deficiency of one cerebral hemisphere is occasionally attended by atrophy of the opposite cerebellar hemisphere, and *vice versa*.

The function of the tract from the cerebrum to the cerebellum is doubtless to maintain a communication between these organs. But the exact function is unknown, and lesions in the course of these tracts in the pons do not produce any known symptoms.

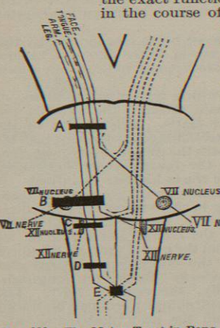

FIG. 880.—The Motor Tract in Pons and Medulla. Lesion at *A* causes hemiplegia of side opposite to lesion; lesion at *B* causes alternating paralysis; lesion at *C* causes paralysis of tongue on side of lesion and of extremities of opposite side; lesion at *D* causes paralysis of extremities of opposite side; lesion at *E* causes paralysis of extremities of both sides.

(2) *The Motor Tracts.*—These enter the upper border of the pons from the middle third of each pes pedunculi (Fig. 877), having come from the central convolutions through the corona radiata and anterior part of the posterior division of the internal capsule (Fig. 876, 2). Each motor tract, at its entrance into the pons, contains fibres whose destination is to the motor centres for the face, arm, and leg of the opposite side. But the centres of the facial nerve lie in the pons Varolii (Figs. 882 and 883). In its passage through the pons, therefore, the facial division of the motor tract leaves the remainder and crosses the median line. This crossing is made by way of the raphé, fibres leaving the longitudinal bundles in the ventral half of the pons, turning dorsad, entering the raphé, decussating with similar fibres from the opposite side, and then turning laterad to reach the facial nucleus (see Figs. 882 and 883). From the facial nucleus the facial nerve passes outward to the side of the pons, having first passed inward and curved around the abducens nucleus in the so-called knee of the facial nerve. The accompanying diagram indicates the course of the motor tract in the pons and medulla. As a result of the division of the motor tract of one side, lesions in the pons produce different forms of paralysis, according to their location. A lesion in the motor tract in the upper third of the pons causes hemiplegia of the side opposite to the lesion (Fig. 880, lesion at *A*). A lesion in the middle or lower third of the pons causes alternating paralysis, *i.e.*, paralysis of the face on the side of the lesion, with paralysis of the arm and leg on the opposite side (Fig. 880, lesion at *B*). In the first case (hemiplegia) the upper branch of the facial nerve is not affected, the patient can close his eye on the paralyzed side, and the facial muscles contract to the faradic electric current. In the second case (alternating paralysis) all branches of the facial nerve are involved, the eye remains open, and the reaction of degeneration develops soon after the lesion occurs. In this case it is usual to find a paralysis of the abducens nerve on the same side as the facial paralysis, in which case the eyeball will be turned in.

The motor fibres to the tongue take a similar course to those to the face, but leave the motor tract at a point in the lower third of the pons. Having reached the hypoglossal nucleus in the medulla they end, but the hypoglossal nerve passes outward, lying in close proximity to the motor tract in the medulla, as shown in the diagram. A lesion in the lower third of the pons or in the medulla may thus cause paralysis of one side of the tongue and of the opposite arm and leg (Fig. 880, lesion at *C*). In the medulla the motor tracts pass in the anterior pyramids (see Fig. 875, *pa*), and before entering

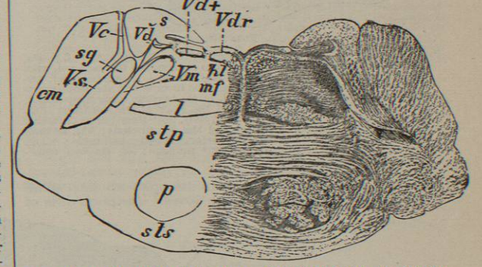

FIG. 881.—Section through the Pons. (Wernicke, × 2.) *sts*, *stp*, Superficial and deep transverse pons fibres from the cerebellum through *cm*, the middle cerebellar peduncle; *p*, the pyramidal (motor) tract; *l*, lemniscus; *mf*, formatio reticularis; *hl*, posterior longitudinal bundle; *Vm*, motor nucleus and root of the trigeminal nerve; *Vs*, sensory root of the trigeminal nerve; *Vc*, cerebellar root of the trigeminal nerve; *Vd*, descending root of the trigeminal nerve (see Figs. 879, 880, and 881); *Vd+*, crossed descending root of trigeminal nerve; *Vdr*, fibres from the raphé to the trigeminal root; *sg*, substantia gelatinosa of *V* root.

the spinal cord undergo partial decussation. A lesion in one pyramid will cause paralysis of the opposite arm and leg (Fig. 880, lesion at *D*); but as the two pyramids lie side by side, lesions of the medulla, by affecting both at once, may produce paralysis of both arms and legs; and this is especially the case if the lesion lies so near the spinal cord as to involve the decussation (Fig. 880, lesion at *E*).

The various forms of paralysis produced when both sides of the pons are involved at once can be ascertained by reference to the diagram.

In all these affections of the motor tract a condition of spastic rigidity may follow the paralysis, and an increase of tendon reflexes is usually observed. In a few cases of pons lesion, however, the tendon reflexes have been lost.