The electric condition of the paralyzed muscles in the limbs is normal from the outset, and no rapid atrophy occurs.

The situation of the pyramidal tract is seen in all the figures at *p*. In the upper part of the pons (Figs. 878

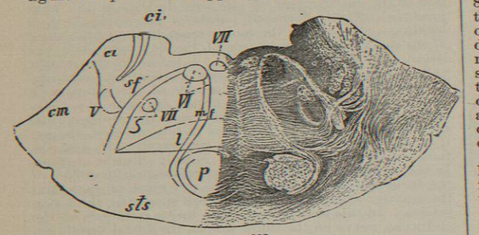

FIG. 882.

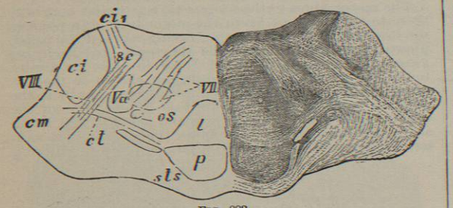

FIG. 883.

FIGS. 882 AND 883.—Sections through Pons. (Wernicke, × 2.) *stS*, Superficial transverse pons fibres from cerebellum through *cm*, middle cerebellar peduncle; *p*, pyramidal tract; *l*, lemniscus; *mf*, formatio reticularis; *VI*, abducens nucleus from which abducens nerve passes forward, lying near *p*; *VII*, facial nucleus lying deep in *mf*, sending its fibres backward toward the floor of the ventricle (Fig. 882), where they turn upward (Fig. 883), and then curve outward, thus forming a bend or knee around the *VI* nucleus; *Va*, ascending root of trigeminal nerve; *ci*, inferior cerebellar peduncle; *VIII*, auditory root; *8c*, external auditory nucleus; *cl*, corpora trapezoides; *os*, superior olive.

and 879) its fibres are seen to be split up into small bundles by the transverse pons fibres. In the middle of the pons, however (Fig. 881), they have collected together, and in the lower part of the pons (Figs. 882 and 883) they form a compact tract which continues downward in the anterior pyramid of the medulla (Figs. 884 to 887, *p*). At the lowest level of the medulla (Fig. 887) the pyramidal fibres decussate, and thus pass into the lateral columns of the spinal cord. About one-fifth of the pyramidal fibres do not, however, take part in the decussation, and these continue downward in the anterior median columns of the spinal cord. The ratio of decussating to non-decussating fibres was found by Flechsig to vary greatly, and in one case in sixty no decussation occurs. The relative sizes of one lateral pyramidal tract and of the opposite anterior median column of the spinal cord depend wholly upon the extent of the decussation of the pyramids in the medulla.

(3) *The Tract of Muscular Sense.*—This is called the lemniscus or fillet in the pons, and the interolivary tract in the medulla. As it conveys sensations upward we trace it in that direction. It arises from the nuclei gracilis and cuneatus on the dorsal surface of the medulla, in which the columns of Goll and Burdach of the spinal cord end (Figs. 887 and 888, *ft* and *fc*). It then crosses the median line, decussating with its fellow of the opposite side in the upper decussation of the medulla (sensory decussation of Meynert; pinniform decussation of Spitzka) (Fig. 887, *l*, *ft*). The fibres after decussating turn upward, enter the interolivary tract, and pass onward through the medulla beyond the upper level of the olives into the pons (Figs. 884 and 885, *l*). Here this tract broadens out, becoming almost ribbon-like in its appearance, and hence has been called the fillet. In the pons the fillet lies behind the deep transverse bundles of the pons, and just in front of the formatio reticularis (see Figs. 878 to 883, *l*).

As it passes up it receives additional fibres from the superior olive (Fig. 884, *o*, *s*), but these leave it again at the level of the posterior corpora quadrigemina (Fig. 878, *l*¹), while the main part of the fillet passes onward in the outer part of the tegmentum of the crus cerebri (Fig. 876, *l*), and thence into the posterior part of the internal capsule, whence it radiates to the parietal cortex of the brain.

Sensations of muscular sense being transmitted by this tract, a lesion in it causes ataxia in the limbs of the opposite side. A unilateral ataxia may indicate a lesion in the course of the fillet.

(4) The *Sensory Tract*, which transmits sensations of touch, temperature, and pain through the medulla and pons, is in the formatio reticularis. This portion of the medulla and pons lies just beneath the gray matter of the floor of the fourth ventricle, behind the tracts hitherto described. It is made up of nerve fibres passing in three directions: (*a*) transversely, the commissural fibres of the cranial nerve nuclei; (*b*) from the nuclei ventrad, the fibres of the cranial nerve roots and arciform fibres; (*c*) longitudinally, the sensory tract. The longitudinal fibres can be traced from the gray matter and various columns of the spinal cord, through the formatio reticularis to its upper level in the tegmentum of the crus, whence they issue to enter the posterior part of the internal capsule. In the medulla the formatio reticularis of each side is divided into two parts by the line of exit of the nerve roots of the twelfth (see Fig. 885). The inner part, near the median line, contains the continuation of the anterior and antero-lateral columns of the spinal cord, and the interolivary tract or lemniscus already described. The outer part contains the sensory tract now under consideration. In the inner two-thirds of this outer part the fibres pass which convey impressions of touch, temperature, and pain from the opposite half of the trunk

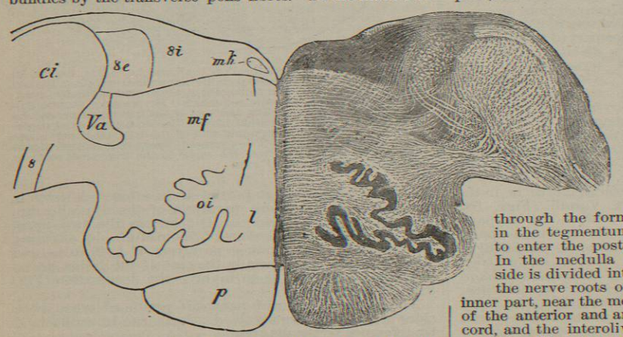

FIG. 884.—Section through Medulla. (Wernicke, × 4.) *p*, Pyramid; *l*, lemniscus or interolivary tract; *o*, olive; *mf*, formatio reticularis; *Va*, ascending root of trigeminal nerve; *8*, auditory root; *8c*, external auditory nucleus; *8i*, internal auditory nucleus; *mk*, upper end of hypoglossal nucleus; *ci*, inferior peduncle of cerebellum.

and limbs. In the outer third of this part is found a column of peculiar structure resembling the substantia gelatinosa of the posterior horn of the spinal cord, and in

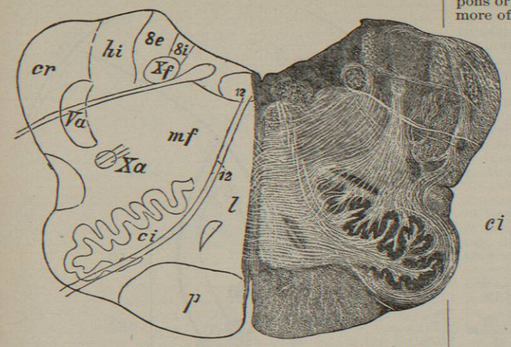

FIG. 885.—Section through Medulla. (Wernicke, × 4.) *p*, Pyramid; *l*, lemniscus or interolivary tract; *ci*, fibres from the olive which cross the raphé and pass outward and backward to enter the inferior cerebellar peduncle, *cr*; *mf*, formatio reticularis, in which *Xa*, the deep nucleus of the vagus (nucleus ambiguus), lies; *Va*, ascending root of trigeminal nerve; *12*, hypoglossal nucleus and root; *Xf*, respiratory bundle around which, in the floor of the ventricle, the vagus nucleus lies, and below which the vagus root issues; *8e* and *8i*, auditory nuclei; *hi*, nucleus cuneatus, from which fibres pass outward and enter *cr*.

this column terminate the fibres of the sensory part of the trigeminal nerve (Figs. 884 and 885, *Va*), which turn downward after entering the pons Varolii, and terminate at different levels in the pons and medulla (see Fig. 886). Thus the outer portion of the formatio reticularis contains the sensory tract from the face of the same side. It is evident from the diagram that a lesion which involves one-half of the formatio reticularis in the pons and medulla will produce an alternating anæsthesia, *i.e.*, loss of sensation on one side of the body and on the other side of the face and head (lesion at *B*, Fig. 886). Alternating anæsthesia is as characteristic a symptom of lesions of the formatio reticularis of the pons and medulla, as alternating paralysis is of lesions in the motor tract of the pons. In the upper part of the pons the sensory tract from the face crosses the median line, and hence a lesion in the formatio reticularis in the upper third of the pons, or in the crus cerebri, will produce a unilateral anæsthesia (lesion at *A*, Fig. 886; see Fig. 881, *Vdr*, *Vd*+). A lesion involving both halves of the formatio reticularis will produce bilateral sensory symptoms.

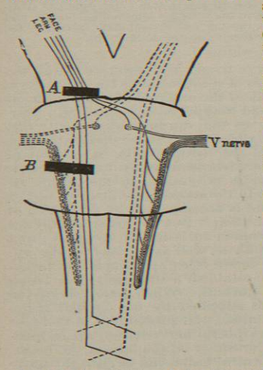

FIG. 886.—The Course of the Sensory Tract in Pons and Medulla. *A*, Lesion in tegmentum of crus cerebri, or upper quarter of pons, producing hemianæsthesia of the opposite side; *B*, lesion in formatio reticularis of pons or medulla, producing alternating anæsthesia.

It is evident that a lesion of any extent, either in the pons or in the medulla, will inevitably destroy one or more of these four tracts, and consequently will produce serious symptoms of wide extent and of considerable diversity.

2. *The Centres Lying in the Pons and Medulla.*—The cranial-nerve nuclei lying in the

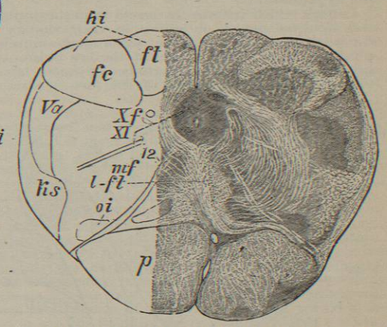

FIG. 887.—Section through Medulla, Level of Sensory Decussation. (Wernicke, × 4.) *ft*, Nucleus gracilis; *fc*, nucleus cuneatus, from which fibres curve around the central gray, and then decussate and turn upward (cephalad) in the interolivary tract, *l-ft*; *Va*, ascending root of the trigeminal nerve; *ci*, inferior peduncle of cerebellum, of which *hs*, direct cerebellar column of the cord, is a part; *Xf*, respiratory bundle; *XI*, medullary nucleus of spinal accessory nerve and its root; *12*, hypoglossal nucleus and root; *mf*, formatio reticularis; *oi*, olive.

pons and medulla must be briefly enumerated, since lesions affect them. They are located either in the gray matter of the floor of the fourth ventricle, or below this in the formatio reticularis.

Figs. 889 and 890 show the relative position of the cranial-nerve nuclei in the pons and medulla from the fifth to the twelfth, inclusive.

The third and fourth nerve nuclei and roots, together

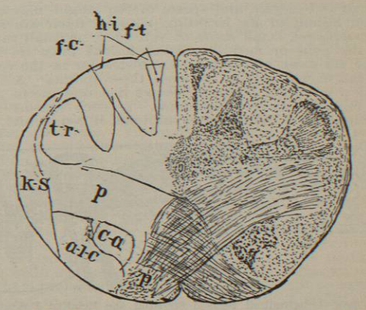

FIG. 888.—Section through Medulla, Level of Motor Decussation. *hi*, Posterior columns of spinal cord; *ft*, nucleus gracilis in column of Goll; *fc*, nucleus cuneatus in column of Burdach; *tr*, tubercle of Rolando, where posterior horn of spinal cord merges into *Va*, ascending root of *V*; *ks*, direct cerebellar column; *p*, pyramid decussating to reach lateral column; *ca*, anterior cornu of cord; *alc*, antero-lateral column of cord.

with the effect of lesions in them, will be discussed in the article on *Ophthalmoplegia*.

The cranial nerves from the fifth to the twelfth may be considered in two categories: first, the motor nerves; second, the sensory nerves.

(1) The *Motor Nerves* are the motor branch of the trigeminal which supplies the muscles of mastication, the abducens, the facial, the motor portion of the glosso-pharyngeal and of the vagus, the spinal accessory, and the hypoglossal.

Lesions which irritate the tracts from the cortical centres to these nuclei, or lesions in their vicinity which cause irritation, may produce spasm of the muscles supplied by them. Thus spasm of the jaw (trismus), with grinding of the teeth, nystagmus, conjugate lateral deviation of the eyes, spasm of the face, spasm of the

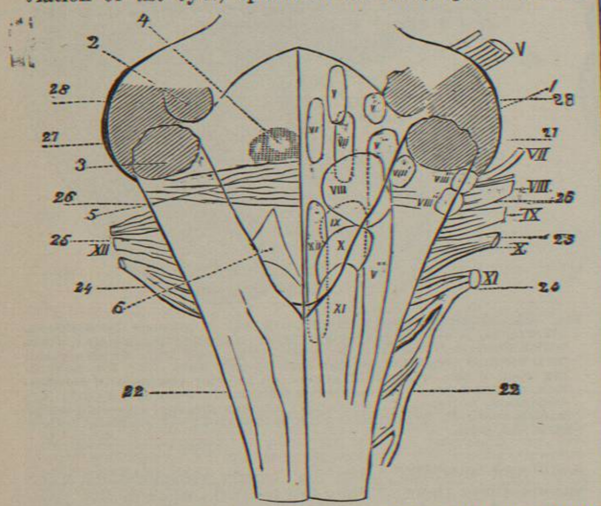

FIG. 889.—Diagram Showing the Position of the Cranial Nerve Nuclei upon the Floor of the Fourth Ventricle (Erb). *v* to *xii*, Nuclei; *V* to *XII*, cranial nerves; *v*, median of *V*; *v′*, motor nucleus of *v″*, ascending root of *V*; *viii*, inner nucleus of *VIII*; *viii′*, outer nucleus; *viii″*, *viii‴*, lateral nuclei of *VIII*; 27, middle cerebellar peduncle; 3, inferior cerebellar peduncle; 2, superior cerebellar peduncle; 4, eminenter teres; 5, striæ acusticæ; 6, ala cinerea.

throat and larynx, and spasm of the tongue may occur from lesions of an irritating nature. Such are small hemorrhages, small areas of softening, or small tumors in the pons or medulla. Such lesions must lie in or near the floor of the fourth ventricle, or in the course of the nerve roots, to produce these spasms. A review of recent literature shows that they are to be regarded as rare symptoms. If such spasms occur alone, without other symptoms, it is difficult to determine the nature of the lesion, or to decide that the spasm is not functional. If they occur in connection with other symptoms of pons or medullary lesion, they are valuable as evidence of the exact position of the disease.

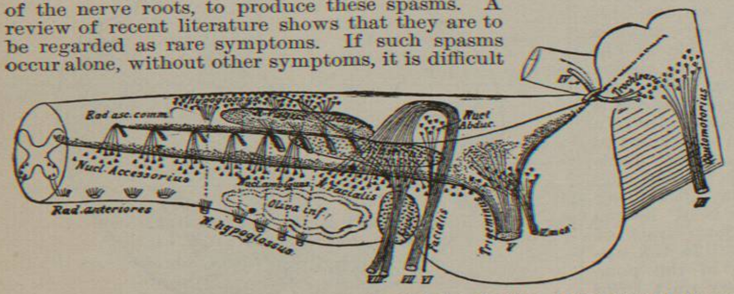

FIG. 890.—Longitudinal Section Showing the Relative Position of the Cranial Nerve Nuclei (Edinger).

Lesions which interrupt the tracts from the cortical centres to the motor-nerve nuclei, which destroy those nuclei, or which interfere with the conduction of impulses along

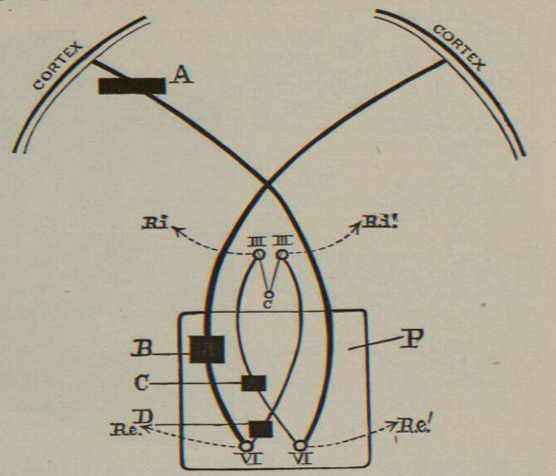

FIG. 891.—The Innervation of the Muscles of the Eye. (After Hunnius, "Zur Symptomatologie der Brückenerkrankungen.") P, Pons Varolii; III, oculomotor nuclei, with R.i., nerve to left internal rectus, and R.i.′, to right internal rectus; VI, abducens nuclei, with R.e., nerve to left external rectus, and R.e.′, to right external rectus; tract from cortex to VI conveys voluntary impulses of conjugate deviation of the eyes; tract from VI to III unites the action of one external rectus with the opposite internal rectus; C, centre for convergence of the eyes which is independent of VI and is actually in connection with the cortex by independent tracts not shown; A, situation of a lesion in the cerebrum producing loss of voluntary conjugate deviation of the eyes to the right; B, situation of a lesion in the pons producing loss of voluntary conjugate deviation of the eyes to the left; C, situation of lesion in the pons in the posterior longitudinal bundle, causing paralysis of conjugate motion to the right; D, situation of a lesion in the pons in the posterior longitudinal bundle, and involving the VI nucleus, causing loss of conjugate motion to the left, with permanent deviation of the eyes to the right.

the nerve root and nerve trunk, will produce paralysis. Paralysis of the muscles of mastication is the rarest form of paralysis. Paralysis of the external rectus muscle, or of the facial muscles and tongue, is very common. Paralysis of deglutition or of the vagus is occasionally met with. It is easy to determine in any case whether the paralysis is due to lesion of the central tracts, or to lesion of the nerve nuclei. In the first case atrophy does not follow the paralysis, and there is no change in the electric reaction in the muscles. In the second case atrophy occurs soon after paralysis, and is accompanied by a reaction of degeneration. But the latter symptoms are common to lesion of the nerve root and trunk as well as to lesion of the nucleus; hence other symptoms are necessary to establish a diagnosis of a lesion in the nucleus. When paralysis of the muscles supplied by one cranial nerve occurs alone, the probability is in favor of a peripheral nerve lesion, *e.g.*, a meningitis or tumor on the base of the brain; and a central lesion can be thought probable only when an implication of other muscles in the domain of other nuclei ensues.

Paralysis of the external rectus is so common that it requires some attention. It is usually due to a lesion on the base of the brain outside of the pons, involving the sixth nerve. It may be due also to lesion in the pons between the nucleus of the sixth nerve and the exit of the root (see Fig. 882). In both cases there will be a permanent deviation of the eye inward and an inability to turn it outward, with contraction of the pupil.

But abducens paralysis may also be due to a lesion of the abducens nucleus in the pons. In such cases there is the added symptom of inability to perform conjugate movement of both eyes toward the side of the lesion, and a permanent deviation of both eyes to the opposite side. But this deviation does not prevent the voluntary act of convergence of both eyes to a near point. Lastly, abducens paralysis may be due to lesions in the crus, or between the crus and the cortex, in the course of the tract conveying voluntary impulses from the cortex to the sixth-nerve nucleus. Such lesions will interfere with voluntary conjugate movement of both eyes to one side, viz., toward the side whose abducens is paralyzed. But since such a lesion may lie in the pons after the decussation of this tract, or in the cerebrum before its decussation, it cannot be said that the deviation is uniformly either toward or away from the lesion. In this case there is no permanent deviation of the eyes to either side; for the abducens nucleus being uninjured, it maintains the tone of the external rectus muscle, and during rest the eyes are found directed forward. If a lesion in the course of the central tract is of an irritative nature, however, it may cause forced motions of the eyes by sending impulses to the sixth nucleus. Under these circumstances there will be conjugate deviation of both eyes toward the side of the abducens paralysis.[1] The act of conjugate movement may also be interfered with, without paralysis of the abducens nerve. That act is governed by the sixth nerve nucleus, or by a special nucleus in its immediate vicinity as yet undetermined. It involves an innervation of one external rectus muscle and of the opposite internal rectus muscle. But the internal rectus muscle derives its innervation from the third nerve. A crossed connection must therefore be maintained between the abducens of one side and the oculo-motorius of the other side. This connection is maintained by means of the posterior longitudinal fasciculus (Figs. 877 to 883, *hl*). A lesion in the pons in the course of this bundle will interfere with the act of conjugate movement without paralyzing either abducens or oculo-motor nerve alone. In none of the cases mentioned is voluntary convergence of the eyes to a point suspended. These conditions may be understood by reference to Fig. 891.

A very extreme contraction of the pupils is mentioned as a symptom in cases of sudden lesion of the pons. It has not been explained.

Paralysis of the facial muscles is very common, but is usually due to lesions in the nerve outside of the pons. The symptoms which are produced when the facial nerve root or its nucleus is invaded in the pons, resemble those of Bell's palsy; for all the facial muscles are affected, and an atrophy with reaction of degeneration ensues. The situation of the lesion in the pons can be determined only when other symptoms of pons disease, such as alternating paralysis (Fig. 880), are present.

Paralysis of the motor portion of the glosso-pharyngeal nerve, with dysphagia or difficulty of swallowing, is a not infrequent symptom of pons disease. It occurs after sudden lesions, such as hemorrhage or embolism, also as the result of tumors, and most frequently of all in progressive bulbar paralysis.

Paralysis of the motor and vaso-motor mechanisms, governed by the vagus nerve, is a constant symptom of disease in the lower part of the pons and in the medulla. Arrest of respiration and heart action, from lesion of this nucleus, is the cause of sudden death, which so frequently attends sudden lesions in the medulla and pons. When the lesion is, however, of a slowly progressive nature (tumor, sclerosis about a hemorrhage), symptoms of irritation may occur from gradual invasion of the pneumogastric centres. Projectile vomiting; slow or very rapid, or intermittent, irregular pulse; dyspnœa; loss of voice; polyuria and glycosuria, are symptoms which have been ascribed to such lesions. The two last-named symptoms have been associated with tumors in the lower part of the medulla[2] and upper part of the spinal cord, which have involved the fibres from the vaso-motor centre on their way downward to their exit from the cord at the eighth cervical segment. The intra-medullary nucleus of the spinal accessory nerve is so closely adjacent to that of the vagus that they are usually involved together, when difficulty of respiration and articulation ensues. When the spinal portion of the nerve is affected, spasms of the neck occur; but this symptom is characteristic of meningeal affections, and has not been observed in central lesions.

An affection of the hypoglossal nerves or their nuclei is very frequently observed in medullary disease, partly because the nerves pass through the middle of the medulla, and partly because their nuclei are large. If the lesion produces irritation of one nucleus, fibrillary twitchings and spasm in one-half of the tongue may be caused. It is more usual to find paralysis than spasm, in which case the tongue, when protruded, will deviate toward the paralyzed side, and will, therefore, point toward the lesion. But when the tongue is affected by disease in the medulla, it is not unusual for both halves to be involved, and then it is found that the power of protruding it is suspended, and motions in the mouth are also difficult. Hence talking, chewing, and swallowing are interfered with. An electric examination in these cases will demonstrate the reaction of degeneration in the intrinsic muscles of the tongue, and prove that the lesion is medullary, and not cerebral. Anarthria or dysarthria, *i.e.*, imperfect articulation, is, according to Nothnagel, a frequent and important symptom of medullary disease.

(2) The *Sensory Nerves* of the pons and medulla are the trigeminal, the auditory, and the glosso-pharyngeal. Lesions which affect the trigeminal nerve and produce anæsthesia of the face have already been considered. Trophic disturbance of the eyeball has not yet been observed in cases of lesion of the trigeminal root or nucleus. The auditory nerve is occasionally affected in diseases of the pons and medulla. Two sets of symptoms are then produced: one referable to the implication of the fibres conveying auditory impressions, the other due to the irritation or destruction of fibres concerned in the sense of equilibrium. Deafness has been observed in pons disease on the side of the lesion,* and is probably due to destruction of the auditory fibres passing inward to the inner auditory nucleus on the floor of the fourth ventricle, or to an implication of that nucleus (see Fig. 884). The central course of the auditory tract is believed to be as follows: From the inner and outer auditory nuclei fibres cross the median line in the corpus trapezoides (Fig. 883, *ct*) to the opposite superior olive, thence turn upward with the lemniscus, pass beneath the posterior corpora quadrigemina in the lower lemniscus (Fig. 877, l^1), and thus reach the internal geniculate body, from which a tract passes outward through the lower posterior part of the internal capsule and corona radiata to the temporal lobe of the brain (Figs. 876, 884). The course of this tract has been traced by anatomists, and has not yet been confirmed by pathological observation. Lesions at any part of it should produce deafness in one ear. Disturbance of equilibrium may occur from lesions in the lateral portion of the pons, if such lesions interrupt the part of the auditory nerve conveying such sensations to the cerebellum. This part of the auditory nerve passes either directly into the cerebellum through the inferior peduncle, or terminates in the external auditory nucleus, which is connected by fibres with the cerebellum (Figs. 882, 883, *e*, ci^1). Vertigo, which is a frequent symptom of pons lesion, and rotatory movements of the body toward one side, which is an occasional symptom of pons disease when the lesion involves the lateral portion of the pons, and the inferior and middle cerebellar peduncles, are probably due to an involvement of this portion of the auditory nerve in the lesion. The rotatory movement is probably due to a voluntary attempt to correct a subjective sense of motion. The patient really suffers from vertigo, and thinks that he is turning or falling

* It is recorded in five cases in twenty-six. In other cases it may have been present, but was not recorded (Starr: "The Sensory Tract in the Central Nervous System," Jour. of Ment. and Nervous Disease, July, 1884).

toward one side; he therefore tries to prevent this falling by turning toward the other side. The symptom may be so serious that such rotatory movements are constantly kept up until the patient dies of exhaustion. The patient turns toward the side opposite the lesion (?).

The sensory portion of the glosso-pharyngeal nerve receives the sensations of taste,[3] and in two cases in which its nucleus has been involved a loss of taste has occurred. This symptom is so rarely elicited that it is not known whether lesions elsewhere in the pons or medulla will produce it.

The Vaso-Motor Centre.—Lesions in the upper half of the medulla produce marked vaso-motor symptoms.[4] They consist of a general vaso-motor paralysis with flushing of the surface and sensation of heat, and of abnormal sweating. The vaso-motor centre is bilateral, and each centre controls the circulation on the same side of the body. Lesions in its area are so likely to cause sudden death that but few cases can be found in which it was affected (eight cases in my collection). But the symptoms of a vaso-motor character are to be looked for in any case of medullary lesion, and when such symptoms are limited to one lateral half of the body, and are associated with other symptoms of bulbar disease, they are valuable as signs of the situation of the lesion.

There are a few general symptoms that have been not infrequently observed in pons disease which require mention. General convulsions are the most constant of these. The majority of sudden lesions (hemorrhage, embolism) in the pons are ushered in by general convulsions, followed by coma. Nothnagel established the fact that in animals irritation of the pons produces general convulsions, and hence authors have spoken of a convulsive centre in the pons. It is hardly warrantable to hypothecate such a centre. But it is probable that lesions in the pons are capable of producing general convulsions by irritating the motor tracts which pass through it. This is borne out by the fact that when the lesion is a tumor or a sclerosis, *i.e.*, a gradually increasing lesion, convulsions do not occur. Headache, disturbance of vision, vertigo, and psychical changes have been frequently observed in connection with diseases of the pons and medulla, but they are to be ascribed to changes in the circulation or internal cranial pressure, and not to any special local lesion.

It is evident from this review that the symptoms of lesions of the pons and medulla are very numerous, very complex, and very various, in accordance with their extent and the manner of their occurrence. The symptoms in bulbar paralysis are very different from those of softening from embolism. And as almost every case so far recorded has differed from every other in important particulars, it is necessary to study carefully and fully all the symptoms occurring in any case of central lesion. If the cranial nerves are involved, or if there is present alternating paralysis or anæsthesia, the possibility of pons or medullary disease must be considered, and an investigation of the various symptoms here detailed should not be neglected. *M. Allen Starr.*

[1] See Report of Clinical Society, Lancet, March 18th, 1887.
[2] Archiv für Psychiatrie, xiii., S. 658 u. S. 671.
[3] C. L. Dana: Jour. Nerv. and Ment. Dis., xiii., 65, 1886.
[4] M. A. Starr: The Sensory Tract in the Central Nervous System. Jour. Nerv. and Ment. Disease, xi., July, 1884; also, Vaso-Motor Neuroses, Pepper's System of Medicine, vol. v.

BRAIN: EMBOLISM AND THROMBOSIS.—EMBOLISM.—*Etiology.*—Cerebral embolism is almost always the result of an endocarditis, either acute or chronic, of the left side of the heart. In acute ulcerative endocarditis the emboli are usually very small, and lodge in the capillaries. This form of the disease will not be discussed in this article.

The embolus consists generally of small, soft particles of fibrin which have been detached by the current of blood from the vegetations on the valves of the heart. But the embolus may also be composed of calcified particles, or of pieces of the valves which have been separated by the ulcerative process from the main part of the valve. Less frequently the embolus is detached from a cardiac thrombus, situated often in the left auricular appendix, or near the apex of the left ventricle.

In rare instances the embolus is derived from the right side of the heart, and such cases have been explained either by the patency of the foramen ovale, or by the transmission of the embolus through one of the pulmonary veins, which are said to constitute a direct communication between the right and left sides of the heart. Cohnheim reports a case of embolism of the middle cerebral artery owing to thrombosis of the veins of the lower limb. In this case the foramen ovale readily admitted three fingers. So far as we are acquainted with the literature of the subject, this is the only case of the kind on record.

Myocarditis may also give rise, though very rarely, to cerebral embolism by causing rupture of the endocardium, and the consequent admixture of the products of inflammation with the blood. This is also true of gummatous or other growths in the substance of the heart, which proliferate through the endocardium.

Another source of embolism is found in atheromatous degeneration and calcification of the inner coats of the aorta, with subsequent deposit of fibrin, and in aneurism of the same vessel. Embolism may result also from thrombosis of one of the arteries in the circle of Willis, a portion of the thrombus becoming detached and giving rise to an embolus in one of the more peripheral vessels in the brain.

Cerebral embolism may also result from gangrenous or other processes in the lungs, which have caused ulceration and finally perforation of a vein, and thus permit the entrance of the gangrenous or other material into the blood. This mode of development has been referred to previously in the discussion of abscess of the brain.

Tumors may give rise to embolism in the same way. Another equally rare cause of embolism is the existence of wounds, phlegmonous inflammations involving the subcutaneous adipose tissue, and complicated fractures of the bones in which inflammatory processes are set up in the medulla of those organs.

Von Dusch maintains that many of the cases of sudden death in pleurisy during aspiration, or while the pleura is being washed out, are due to the formation of thrombi in the pulmonary veins. He believes that the development of paralysis, which is observed sometimes under such circumstances, may be explained by the secondary occurrence of cerebral embolism. Indeed, in one instance of this kind, the cerebral emboli were discovered at the post-mortem examination. Finally, mention should be made of the capillary pigment emboli of the brain, seen occasionally in severe forms of intermittent and remittent fever. These emboli may be so numerous as to give the brain a chocolate color. All the cases of the kind which have come under our observation have originated in Central or South America.

In our experience cerebral embolism occurs much more frequently in males than in females, but, according to Gowers, it is more frequent in women, owing, as he claims, to the greater frequency of mitral stenosis in females.

Pathological Anatomy.—Cerebral emboli (with the exception of the capillary emboli, which will not be discussed in this article) are usually single, and, in the majority of cases, are situated in the left middle cerebral artery. This predilection is explained by the fact that the current of blood from the aorta passes by a straighter course into the left carotid than into the right carotid, and that, furthermore, the left middle cerebral artery is the direct continuation of the carotid. It has been claimed by Gelpke, on the basis of statistics, that the left middle cerebral presents only a slight preponderance over the right middle cerebral in this respect, but this is probably owing to the fact that, on account of the usual situation of the embolus in the vessel on the left side of the brain, the history of such cases is not generally reported.

In a certain proportion of cases the emboli are situated

in other vessels, and even two or more may be present at the same time.

The following is a brief *résumé*, according to Duret, of the distribution of the blood-vessels to the brain. These vessels may be divided into two classes: First, those which are given off at the base of the brain and at once enter the organ to supply the parts situated above (basal ganglia, etc.); and secondly, the continuations of these vessels which supply the cortex. We shall first describe the former.

The anterior cerebral and anterior communicating arteries send off a number of small branches which supply the anterior part of the corpus striatum.

The posterior cerebral artery sends branches to the walls of the third ventricle, the optic thalamus (mainly the posterior portion), the tegmentum of the crus cerebri, and the corpora quadrigemina.

The middle cerebral artery (the most important artery of the brain) gives off a number of branches before it supplies the cortex: (1) The internal striate arteries to the first and second divisions of the lenticular nucleus and the internal or white capsule; (2) the lenticulo-striate arteries which supply the anterior part of the third division of the lenticular nucleus and of the internal capsule, and also pass to the corpus striatum; (3) the lenticulo-optic arteries which pass to the posterior part of the third division of the lenticular nucleus and the anterior portion of the optic thalamus.

Following is the cortical distribution of these vessels:

The anterior cerebral artery supplies the convolutions on the inferior surface of the frontal lobe, the first and second frontal convolutions, the paracentral lobule, and the præcuneus.

The posterior cerebral artery supplies the gyrus uncinatus and hippocampus, the inferior surface of the temporo-sphenoidal lobe, and the occipital convolutions.

The middle cerebral breaks up into five branches: (1) To the third frontal (Broca's) convolution; (2) to the ascending frontal convolution; (3) to the ascending parietal convolution; (4) to the inferior parietal and superior temporo-sphenoidal convolutions; (5) to the first and second temporo-sphenoidal convolutions.

The pons Varolii and medulla oblongata are supplied in the following manner:

The basilar artery gives off vertical branches, which supply the nuclei of origin of the nerves and the ependyma of the fourth ventricle. The nuclei are also supplied by small vessels, which enter with the roots of the nerves.

The inferior cerebellar artery supplies the lateral parts of the medulla and the inferior cerebellar peduncle. The remaining portions of the pons and medulla, and the cerebellum, are supplied by the transverse and cerebellar arteries.

The vessels which enter the substance of the brain from the base are terminal arteries, *i.e.*, there is no anastomosis between the distribution of one vessel and the adjacent ones. A certain amount of anastomosis exists between the vessels which ramify in the pia mater, but this is very often insufficient to compensate for the anæmia produced by the plugging of one of these arteries.

When a terminal artery is completely obstructed by an embolus, the supply of arterial blood to the distribution of the vessel is shut off. According to Cohnheim, the reflux of blood from the corresponding vein will gorge the capillaries with blood. Litten believes that the filling of the capillaries is due to the influx of blood from the capillaries of adjacent regions. If the blood pressure is too feeble, however, this feature is not observed.

As a result of the cessation of circulation in the territory supplied by the obstructed vessel, the nutrition of the capillaries and veins becomes impaired, and they therefore permit the escape of red blood globules. Then the tissues which have been deprived of blood undergo simple fatty degeneration, and an infarction is produced. Finally, absorption occurs, leaving a cyst containing clear fluid.

If a reflux of blood into the vessels does not take place, the infarction has a yellowish or whitish color from the beginning, but in other respects the course is the same as that just described. The retrogressive changes (necrobiosis) probably begin at the end of thirty-six or forty-eight hours. Some authorities think that these changes begin at the end of two or three hours.

Unlike what takes place in embolism of other organs of the body, embolism of the brain does not often produce a hemorrhagic infarction, but almost always results in a patch of yellow or white softening. The reason for this difference is not very clear.

When the embolus is lodged in one of the vessels supplying the cortex, the effect produced varies according to the size of the vessel and the extent to which it anastomoses with surrounding ones. Infarctions in this region are usually of small size, though they occasionally attain much larger dimensions than those situated within the brain, particularly if a large branch of the middle cerebral artery has been occluded. The color of the infarction is yellow or brown, and the external surface is often extremely hard. It diminishes in extent toward the interior, and sometimes involves only the gray matter of the convolutions. On the other hand, the white matter below the convolutions may alone be affected, while the cortex escapes. The pia mater above the lesion is usually infiltrated with fluid, and is readily detached from the surface.

In old cases in which the motor regions have been involved, either within the brain or in the cortex, descending degeneration of the pyramidal tracts occurs as it does in cerebral hemorrhage. Moreover, I have occasionally seen atrophy of the unaffected parts of the hemisphere after extensive lesions of this kind, though not to so marked an extent as that which I have described as occurring in cerebral hemorrhage.

Clinical History.—From the nature of the disease, the symptoms of cerebral embolism always begin suddenly. The attack may or may not begin with a disturbance of consciousness, and, as in cerebral hemorrhage, this may vary from a passing vertigo or feeling of confusion to complete and profound unconsciousness. General or unilateral convulsions also constitute a not infrequent accompaniment of the attack. The symptoms during the stage of unconsciousness differ in no respect from the corresponding ones of cerebral hemorrhage, but they are not apt to be so prolonged, and, moreover, there are no characteristic phenomena connected with the bodily temperature. There is usually a slight rise of temperature soon after the development of the seizure, but afterward it varies irregularly from time to time. In fatal cases the temperature usually rises steadily until death.

As the embolus is situated commonly in one of the arteries of the left side of the brain (usually the middle cerebral), right hemiplegia usually results, and in a considerable proportion of cases is associated with aphasia. In fact, the sudden occurrence of right hemiplegia and aphasia, without previous head symptoms, is *prima facie* evidence that we have to deal with an attack of embolism of the left middle cerebral artery.

The character of the aphasia differs greatly in individual cases according to the situation of the lesion, and all the different varieties of aphasia may thus be produced (*vide* the article on *Aphasia*, in Vol. I.). In some cases, indeed, aphasia is the only symptom produced, and may remain permanent without being followed by any other symptom. In a case which came under my observation it was associated with epileptiform convulsions, which recurred at irregular intervals.

At other times the aphasia is a temporary symptom which soon disappears, but is followed later by an apoplectiform attack, attended with hemiplegia, which then runs the usual course.

The duration of the hemiplegia varies within very wide limits. If recovery does not take place within a few weeks, the paralysis will probably persist for the remainder of life. Unlike the hemiplegia of cerebral hemorrhage, the paralysis is not so apt to undergo slow