190

called the genu or knee of the capsule.

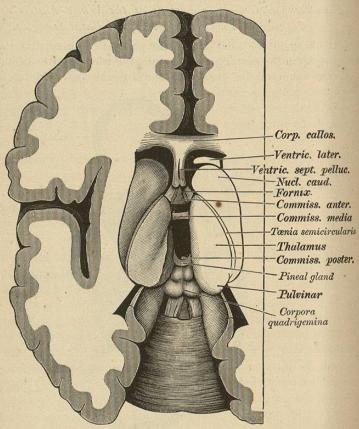


Fig. 48.—VIEW OF THE VENTRICLES ON HORIZONTAL SECTION. (After EDINGER.)

From the internal capsule the fibres reach the crusta (pes pedunculi cerebri), whence they pass through the lower (anterior or ventral) portion of the pons and enter the medulla oblongata as the anterior pyramids. At the lower end of the medulla most of them decussate and pursue a downward course in the lateral columns of the spinal cord on the opposite side. This, the most important of

all direct systems of fibres, was discovered by Deiters in 1865, and most carefully studied by Flechsig in 1876. It is generally known as the lateral pyramidal tract, and it represents the path for the voluntary movements. A lesion of it is therefore of grave consequence for the motor functions.

THE INTERNAL CAPSULE.

Fig. 48 represents a horizontal section which shows the relative position of the caudate nucleus to the optic thalamus, the corpus callosum, the fornix, the two white commissures, the anterior and the posterior, the gray middle commissure, the pineal gland, and the corpora quadrigemina.

Fig. 49 is a third horizontal section through the cerebrum at a lower plane. Both are taken from Edinger.

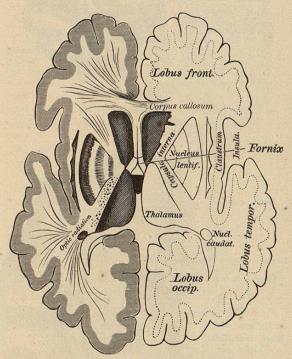


Fig. 49.—HORIZONTAL SECTION THROUGH THE BRAIN, ABOUT A FINGER'S BREADTH BELOW THAT REPRESENTED IN FIG. 48. (EDINGER.)

Figs. 50, 51, and 52 are three so-called frontal sections, of which the first is made through the anterior commissure, the second in front of, the third behind, the middle (gray) commissure. They also show the course of the internal and external capsule, and the situation of the so-called basal ganglia, the caudate and the lenticular nucleus (together known as the corpus striatum), and the optic thalamus.

In another frontal section, Fig. 53 (after Edinger), the direction of the fibres is illustrated diagrammatically.

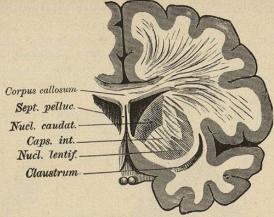


Fig. 50.

Pitres has recommended a series of frontal sections in order to facilitate in our descriptions of autopsies a more accurate localiza-

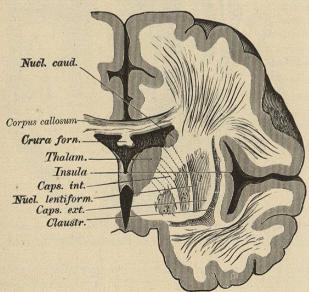
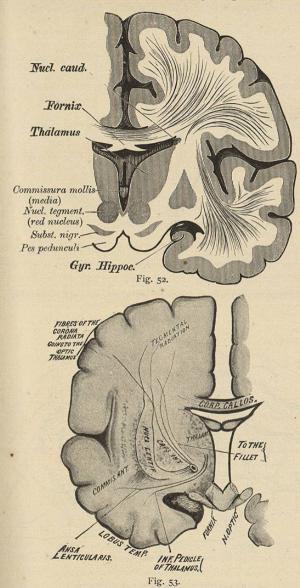


Fig. 51.

tion of lesions and tumors within the very extensive white matter of the brain. Nothnagel has modified somewhat these sections of

Pitres with regard to their position and designation. The table on page 194 contains the necessary explanation. With the help of these sections we need not content ourselves any longer with locating in the



Figs. 50-53.—SO-CALLED "FRONTAL SECTIONS" THROUGH THE BRAIN. Fig. 50, through the anterior commissure. Fig. 51, in front of the middle commissure. Fig. 52, behind the middle commissure. Fig. 53, immediately behind the chiasm. The radiating fibres are shown diagrammatically in the last illustration. (After Edinger.)

post-mortem accounts a tumor "in the anterior part of the brain," "in the temporal lobe," etc., but we give the one or more sections which correspond to the situation of the neoplasm, and so attain an accuracy which is indispensable for the after-use of our autopsy records.

PITRES-NOTHNAGEL FRONTAL SECTIONS.

Designation.	Points where sections are made.	Called by Pitres	Comprises, according to Nothnagel,
A	Immediately in front of genu of corpus callosum.	Coupe préfrontale.	Centri ovalis pars frontalis anterior.
В	Starting at the beginning of fissure of Sylvius.	Coupe pédiculo- frontale.	Pars frontalis media.
B ₁	Between anterior cen- tral and frontal con- volutions.		Pars frontalis posterior.
С	Through the fissure of Rolando.	Coupe frontale.	Pars centralis anterior.
D .	Through ascending parietal convolution.	Coupe pariétale.	Pars centralis posterior.
Е	Through parietal lobe 3 cm. posterior to the fissure of Rolando.	Coupe pédiculo-pa- riétale.	Pars parietalis.
F	Through occipital lobe.	Coupe occipitale.	Pars occipitalis.

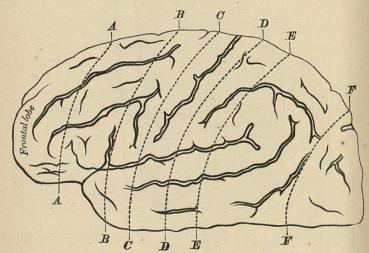


Fig. 54.—Points at which the Pitres-Nothnagel Sections are made. They all run parallel with the fissure of Rolando.

Figs. 55-60 represent diagrammatically Pitres' sections. From Fig. 54 we can get an idea of the points on the surface of the brain

at which the sections are to be made (cf. Pitres, Recherches sur les lésions du centre ovale des hémisphères cérébraux étudiés au point de vue des localisations cérébrales, Paris, 1877).

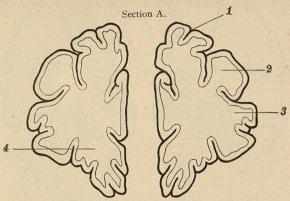


Fig. 55.—1, 2, 3, first, second, and third frontal convolutions. 4, præfrontal fasciculus of the centrum semiovale.

We can likewise avail ourselves of the charts of the human brain published by Exner (two plates, with twelve diagrams, Wien, Braumüller, 1888). On the plates the discovered lesion can be easily marked off, and thus the extent and situation of it represented.

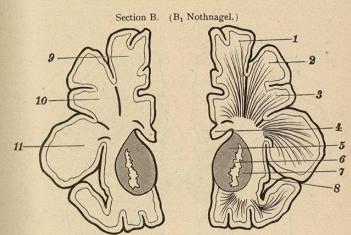


Fig. 56.—1 and 2, first and second frontal convolutions. 3, middle pediculo-frontal fasciculus. 4, corpus callosum. 5, nucleus caudatus. 6, internal capsule. 7, lenticular nucleus. 8, island of Reil. 9, 10, 11, superior, middle, and inferior frontal fasciculi.

With reference to the lesions in the centrum ovale, it should be stated that, as a rule, the symptoms produced by them are similar to those which we find in lesions of the corresponding

Fig. 57.—1, first frontal convolution. 2, 3, 4, superior, middle, and inferior frontal fasciculi. 5, corpus callosum. 6, nucleus caudatus. 7, optic thalamus. 8, internal capsule. 9, lenticular nucleus. 10, claustrum.

area of the cortex. Thus we shall meet with motor disturbances if the fronto-parietal fasciculi of the corona radiata, which

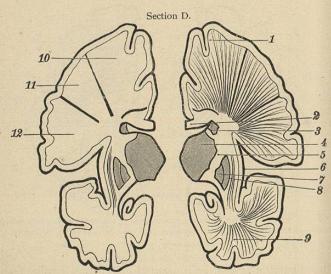


Fig. 58.—1, anterior central convolution. 2, corpus callosum. 3, caudate nucleus. 4, optic thalamus. 5, internal capsule. 6, island of Reil. 7, lenticular nucleus. 8, external capsule. 9, temporal fasciculus. 10, 11, 12, superior, middle, inferior parietal fasciculi.

take their origin in the motor area, are diseased; while lesions in the præfrontal or occipital bundles may, and indeed very often do, not evoke any symptoms. If the left (inferior) pedic-

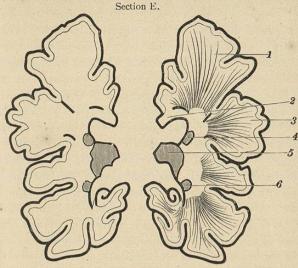


Fig. 59.—1, superior parietal fasciculus. 2, corpus callosum. 3, inferior parietal fasciculus. 4 and 6, caudate nucleus. 5, optic thalamus.

ulo-frontal bundles in addition are affected, the patient will also be aphasic, the aphasia, however, being of long duration only if the lesion extends close up to the cortex. Lesions in

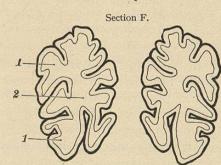


Fig. 6o.—1, occipital convolutions. 2, occipital fasciculus of the centrum semiovale.

Figs. 55-6o.—Pitres-Nothnagel Sections, the position of which is made clear by the table on page 194.

the white matter of the occipital lobe may produce hemianopia, in the temporal lobe auditory disturbances. Whether, however, diseases in the parietal lobes ever produce sensory changes—

developed, is unknown.

The idea that the basal ganglia were true motor centres, and that the common form of hemiplegia was due to lesions thereof, has been given up, and we have learned from the investigations of Flechsig and Wernicke that direct connections between the motor centres of the cortex and these basal ganglia do not exist. Moreover, it has been proved by numerous thoroughly reliable observations that destruction of the lenticular or of the caudate nucleus does not necessarily give rise to a motor paralysis. One or both lenticular nuclei have repeatedly been found destroyed in cases in which there was no sign of paralysis (Lépine, Nothnagel, Edinger, Hebold). In order that this may not ensue, it is only necessary that the internal and perhaps also the outer capsule remain intact. As soon as the former (the inner capsule) is either directly or indirectly implicated, we have a hemiplegia which is either transient or persistent, according to the nature of the lesion in the capsule. Whether the lenticular or the caudate nucleus alone is diseased can not be determined from the symptoms.

There is no doubt but that lesions of the thalamus, especially of its anterior and middle part, may occur without symptoms, and it is impossible to say whether motor paralysis is ever produced by lesions of the thalamus, for in all instances in which this may have been the case the motor paralysis may also have been a result of damage to neighboring parts (pedun-

cles, internal capsule). Better founded is the idea that lesions of the pulvinar, the posterior part of the thalamus, give rise to defects in sightcrossed amblyopia or homonymous bilateral hemianopia; but the possibility that the posterior part of the optic tract is interrupted can even then not be excluded. The athetoid movements and symptoms of motor irritation (hemichorea, posthemiplegic tremor, athetosis) are, even if a connection actually exists between them and lesions of the thalamus (Greif, cf. lit.), certainly not characteristic of such lesions. The same holds good for the disturbances in the muscular sense which have been observed in diseases of the thalamus (Meynert, Jackson). The relation between these latter and loss of the movements of facial expression in the course of central facial paralysis has been spoken of in Part II, Chapter V. Recently Nothnagel has again published a clear case of this kind (Zeitsch. f. klin. Med., 1889, xvi, 5, 6, p. 424).

THE CORPORA QUADRIGEMINA.

Lesions of the internal capsule produce symptoms varying according as the anterior or posterior limb is attacked. Pure cansule lesions—i. e., those in which the caudate as well as the lenticular nucleus remain intact—have rarely if ever occurred. Fissures have been occasionally known to occur without having necessarily produced any motor disturbances in life (cf. Nothnagel, loc. cit., p. 272). The functions of the anterior limb of the internal capsule are obscure, and lesions of this part do not produce any symptoms. With the posterior limb we are better acquainted, and, above all, this one fact is well established, that a lesion of the anterior two thirds of the posterior limb gives rise to the usual typical hemiplegia, with paralysis of the lower facial branches. A very small lesion at the knee may produce an isolated facial paralysis. If the posterior portion of the anterior two thirds is the chief seat of the disease. the paralysis is most marked in the leg. The posterior third of the posterior limb is occupied by the sensory fibres (le carrefour sensitif of Charcot), and lesions of that region cause a loss of sensation on the opposite side of the body ("hemianæsthesia," Oppenheim, Charité-Annalen, 1889, xiv, p. 396), in which often the nerves of special sense are implicated, and hearing, smell, and taste (on the anæsthetic side) are, if not lost, at least diminished. Often hemiplegia is accompanied by hemianæsthesia, because, if the one portion of the capsule is affected, an indirect and transitory implication of the other may occur. Usually such a hemianæsthesia soon disappears in the same way as the indirect motor disturbance often soon passes off in cases of persistent hemianæsthesia. Whether the symptoms of motor irritation (the so-called post-hemiplegic chorea, for instance), which are a not rare accompaniment of hemiplegia, are due to disease of the internal capsule or to disease of the neighboring basal ganglia, is as yet undecided.

By the corpora quadrigemina we mean that peculiar eminence which by a crucial furrow is separated into four parts (bodies), and forms the posterior boundary of the third ventricle. In front it is bounded by the commissure which unites the two thalami; on it tests the pineal gland (conarium). The anterior pair of bodies, which are called the nates, are larger than the posterior, the testes. The appearance and structure which these two pairs of bodies present in the lower mammals, justifies the conclusion that they are to-