a part, and the influence of cold remains, in connection with this disease, as obscure as with all other nervous affections.

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PART III.

DISEASES OF THE BRAIN PROPER.

THE more autopsies we see the more the fact is brought home to us that brain lesions are frequently present which were not diagnosticated during life. This is by no means necessarily the fault of the diagnostician, for undoubtedly many focal lesions of the brain may exist without giving rise to any symptoms. Recently G. Schmid has published an interesting collection of such cases (Virchow's Archiv, 1893, cxxxiv, 1). On the other hand, of course, we frequently see cases presenting symptoms which make us at once suspect the existence of a brain lesion.

In such cases we have to ask ourselves two questions: (1) Where is the seat of the lesion? (2) What is its pathological nature? To the physician both of these questions are of interest; to the patient, more especially the latter.

The examination which searches for the seat of the lesion will give us the topical diagnosis (τοπος = place); the examination concerning the nature of the lesion, the pathological diagnosis.

The endeavor to localize cerebral lesions—that is, to make a topical diagnosis—has only of comparatively late years received attention, and much of the work so far done can not be called more than an attempt, in many cases indeed only a weak one. The celebrated discovery of Broca (1861), that certain disturbances of speech were often found associated with lesions in the third left frontal convolution, the discovery of Fritsch and Hitzig (1870) that stimulation of certain areas of the cortex produces contractions in certain definite groups of muscles on the opposite side of the body—these and various other, pathological, observations, to which reference will be made later, make it most probable, nay, almost certain, that definite parts or areas of the cortex are always connected with certain func-

tions of the brain; in other words, that these functions can be localized; and, notwithstanding the many uncertainties and numerous contradictions between the results of experiments on the one hand and those obtained from clinical observations on the other, it is this doctrine of cerebral localization which, though still undeveloped, must be considered as the basis of all further investigation in the field of cerebral pathology.

Equal stress must, however, be laid upon the examination into the nature of the lesion. A certain symptom—for instance, a persistent hemiplegia—is always the result of a lesion of the motor tract; a lesion, however, which can be produced in quite a variety of ways. It may be due to cerebral hæmorrhage, to a tumor, an abscess, etc. It is therefore, especially with regard to the prognosis, of the greatest importance to determine the exact nature of the lesion in a given case, but both questions ought always to be investigated with equal care if we wish to arrive at as exact a diagnosis as circumstances permit. In the following pages these two modes of diagnosis will be considered separately, and we shall first speak of what is known about cerebral localization, while in a later chapter the pathological side will be discussed.

I. The Study of Cerebral Lesions with Reference to their Seat—Topical Diagnosis—Doctrine of Localization.

Two classes of symptoms produced by cerebral lesions must be distinguished: first, general or diffuse (Griesinger), and, secondly, local. The former, so far as they concern the subjective feelings of the patient and the disturbances of the vegetative functions (temperature, pulse, respiration, condition of urine), are to be observed and described in this connection in the same way as in diseases of other organs. The latter—the local symptoms—may be divided into two classes, namely, the direct and the indirect. We call those symptoms direct which are produced by a persistent disturbance in the functions of a certain part of the brain. They are also called focal symptoms (Griesinger). By indirect symptoms we mean those which are only produced by transient conditions—changes in circulation, by compression, etc.—and which are in a way concomitant symptoms of the former. They may be entirely absent; on the other hand, they may be so prominent as to make a topical diagnosis impossible.

Destruction of a circumscribed area in the brain gives rise to symptoms of paralysis, or less frequently to symptoms of irritation. The former, where we have to deal with a loss of function, are also (after Goltz) called symptoms of destruction (*Ausfallssymptome*), and if the function is not lost but is only impaired, symptoms of impairment (*Hemmungssymptome*). The latter—namely, the irritative symptoms—are usually due to a so-called indirect action.

It is not always possible to say whether a symptom is of a direct or of an indirect nature. For instance, if we find a patient in an unconscious state with a hemiplegia, this hemiplegia may be a direct focal symptom or it may have been produced indirectly. In the latter case it will disappear in a few hours or days, in the former it will be persistent. Or, if a patient suffering from the consequences of a cerebral hæmorrhage presents, as is often the case, disturbances in speech, this may again be a focal symptom or not. If, after consciousness has been regained, the speech becomes gradually but steadily better, then the aphasia was produced indirectly. If, however, speech remains unintelligible for months or years, it is clear that we have to do with a focal symptom. Therefore, in acute lesions we can only after a certain time has elapsed discriminate between direct and indirect symptoms.

The irritative symptoms consist either of general epileptiform convulsions or of partial, involuntary movements of the extremities (hemichorea, athetosis), of tremors, contractures, or forced movements of the whole body. We shall repeatedly have occasion to refer to these phenomena.

Not all of the symptoms have an equal value for the localization of a lesion. It is important first to note their mode of onset, whether this is sudden or gradual; whether several symptoms have made their appearance at the same time or one after the other, and so on, for in acute lesions, for instance, only those symptoms which appear synchronously are of importance. If a patient who has a hemiplegia presents a paralysis of the oculo-motor of the opposite side, and we learn that this latter has existed before the onset of the hemiplegia, nobody certainly will think of connecting the two or look upon them as being symptoms due to one focal lesion. This would only be allowable if both had set in at the same time (after an acute lesion).

But, even apart from the mode of onset, the symptoms are not of equal value in the localization. Some, it is true, as hemiplegia, together with contra-lateral oculo-motor paralysis, are almost pathognomonic (for a lesion in the crus), and their simultaneous appearance is therefore extremely important; while others, as the conjugate deviation in severe hemiplegia, are found in different lesions, and are therefore less significant; still others, as optic neuritis and all the general symptoms (headache, vertigo, unconsciousness), are absolutely valueless.

We see, therefore, that by no means all cases can be used for the study of the topical diagnosis, but only those in which the affection, in the first place, remains chronically stationary; secondly, in which it is circumscribed and isolated (Nothnagel); and, thirdly, where the surroundings of the focus are as little as possible implicated. These three conditions are best fulfilled in instances of hæmorrhage or embolus, or rather in the cases of softening produced by these accidents, and the largest contingent of cases which permit a topical diagnosis is therefore made up of these. They are rendered more suitable for our purpose the longer the time that has elapsed after the first onset (according to Nothnagel, six to eight weeks), as only then, as we stated above, are we able to separate the direct from the indirect symptoms. In other cerebral affections—e. g., meningitis, encephalitis, and especially tumors—a local diagnosis should only be attempted with the greatest circumspection, and even then errors can not be altogether excluded.

Symptoms Referable to Cortical Lesions.

In speaking of cortical lesions, "surface lesions," it must not be forgotten that the clinical meaning of the term is different from the anatomical one. Anatomically, it implies that the medullary layer situated below the cortex is intact, while clinically we speak of cortical lesions even if the white matter takes part in the pathological process as well; but so little attention has been paid to this difference in the autopsy reports, which have been published, that it seems an almost hopeless endeavor to distinguish whether the symptoms reported in a given case were due to changes in the cellular elements of the cortex itself, or to changes in the fibres of the medullary layer situated immediately beneath. Pick (Zeitsch. für Heilkunde, 1889, x, 1) has shown how important it is to make a microscopical examination; this is even more necessary if we find

secondary degenerations in the spinal cord—macroscopically the cortex may present no abnormality in such cases.

We possess quite a considerable amount of material, but it is by no means easy to make a judicious and successful use of it. Certain methods have to be employed in order to arrive at correct conclusions, methods which have been developed in such an excellent way by Exner (cf. lit.). It would, for example, be incorrect to assume a certain cortical area to be the centre for the motor function of an extremity simply because in many cases a lesion of this area was found where a paralysis in that extremity had existed during life. This "method of positive cases," as Exner has called it, is therefore uncertain, because there are quite different circumscribed cortical regions, a lesion in which gives rise to the same symptoms; and since, moreover, facts go to show that such a method may lead us to wrong conclusions, it ought to be discarded. Much more preferable, however, is the so-called "method of negative cases" (Exner), according to which "we have to mark out the lesions, found in all the cases in which a given function was not interfered with, and unite them on one hemisphere." If the number of cases is sufficiently large, while on the remaining parts of the cortex we find markings indicating lesions, the area for the functions in question will remain free.

Still better results are obtained by the method of percentages (Exner). The cortex is divided into arbitrary fields, and for each of these fields we determine, first, how often it has been diseased in a given number of cases; secondly, in how many of the cases the symptom which we are studying was present. The ratio between these two results is best given in percentages. Only through this indispensable, although somewhat tedious, method can we ascertain that the fields of the right cortex are different from those of the left, and that certain areas exist of which a lesion always, and others of which a lesion not always but frequently, produces a certain symptom. The former Exner calls "absolute," the latter "relative," cortical areas.

We do not always find cortical lesions at the autopsy in cases in which certain symptoms, which would have led us to suspect their existence, have been noted in life. On the other hand, they are found in cases where we have hardly felt justified in expecting them. There is no doubt that no inconsiderable part of the cortex can be diseased without giving rise to

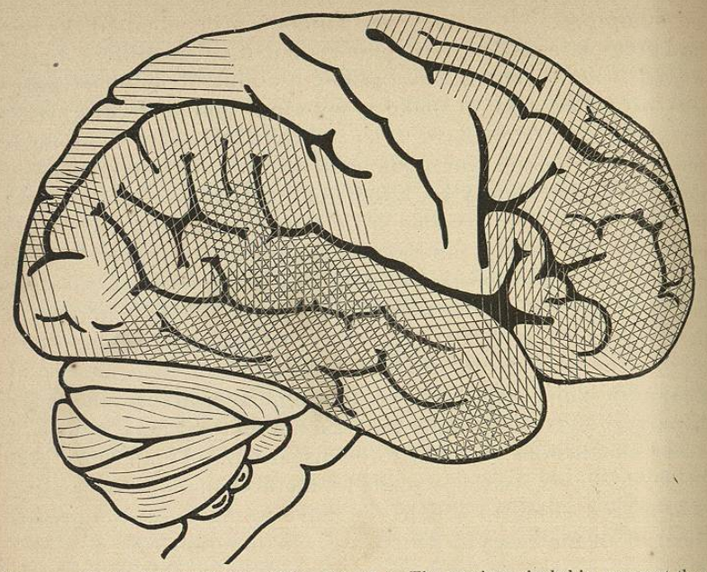

Fig. 35.—RIGHT HEMISPHERE. (After EXNER.) The portions shaded in represent those parts of the cortex which can be injured without giving rise to sensory or motor disturbances; the blank areas are motor and sensory.

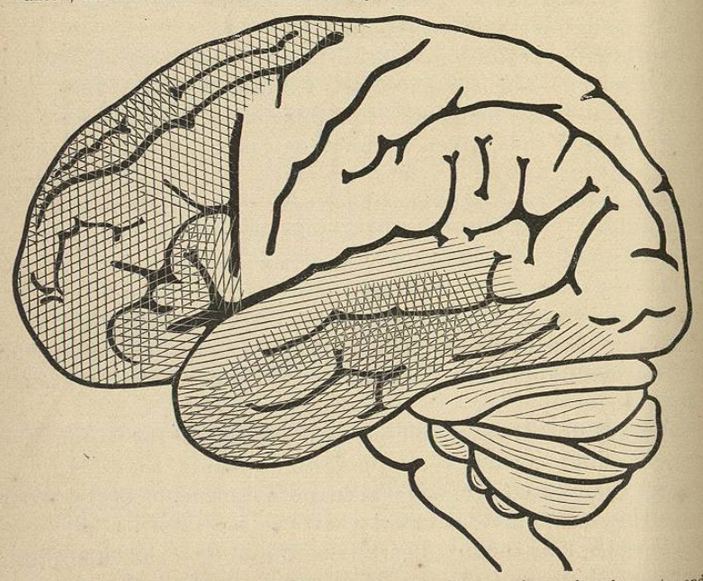

Fig. 36.—LEFT HEMISPHERE. (After EXNER.) This diagram shows that the motor and sensory areas are of greater extent on the left than on the right hemisphere.

any symptoms. It is this part which has been called the cortical area of latent lesions (Exner), and it is certainly a matter worthy of note that the extent of this area is smaller on the left than on the right hemisphere, whereas the motor area—that is, the area in which a lesion is followed by motor disturbances—is larger and more developed on the left than on the right side (cf. Figs. 35 and 36). The first represents the right, the second the left hemisphere. On both all the lesions are indicated which have produced neither sensory nor motor disturbances. The blank fields are therefore sensory and motor—their greater extent on the left side is at once apparent.

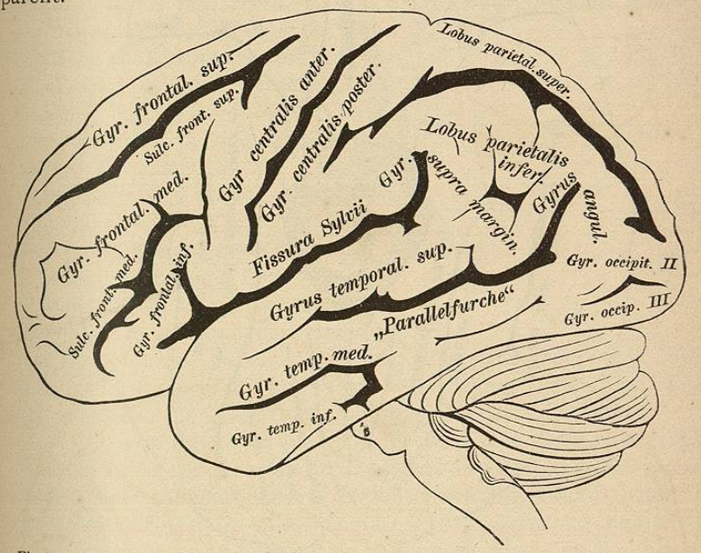

Fig. 37.—CONVOLUTIONS AND FISSURES OF THE LATERAL ASPECT OF THE BRAIN. (After ECKER.) Parallelfurche = parallel fissure, or first temporal fissure.

Before we go into the description of the individual lesions of the cortex we will briefly refresh our memory on the anatomy of the parts.

The thin gray covering which surrounds the white matter, and which has been called brain cortex, presents on each hemisphere three surfaces—a lateral, a basal, and a median. The two lateral form the convexity, the two basal the base of the cerebrum. The cerebrum is divided into lobes, which can again be subdivided into convolutions or gyri. To be able to localize and correctly describe

cortical lesions we must be thoroughly familiar with the position, as well as the names, of the different convolutions. The following illustrations are intended to facilitate the study of the convolutions and the fissures or sulci separating them. Fig. 37 represents those on the lateral surface (convexity), Fig. 38 those on the basal, and Fig. 40 those on the median aspect of the cerebrum.

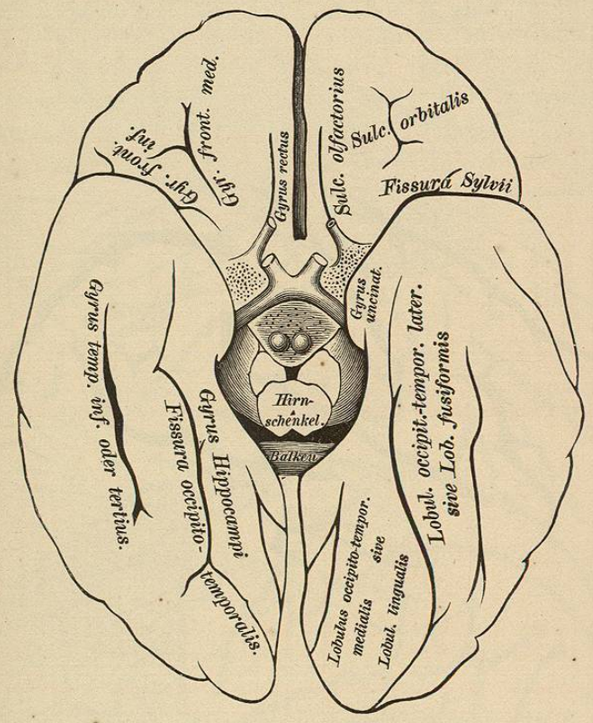

Fig. 38.—Convolutions and Fissures at the Base of the Brain. (Diagramatically after Ecker.) Hirnschenkel = crura cerebri. Balken = corpus callosum.

In Fig. 37 are included the frontal, parietal, temporal, and occipital lobes, so far as their convolutions and fissures belong to the lateral surface—in other words, belonging to the frontal lobes, the three frontal and the anterior central convolution (gyrus centralis anterior, *pli frontal ascendant*), and belonging to the parietal lobe, the posterior central convolution (gyrus centralis posterior, or *pli parietal ascendant*); between the last two is seen the fissure of Rolando. Further, a part of the upper and the entire lower parietal lobe are shown, which latter is subdivided into the supra-marginal

convolution in front and the angular gyrus (*pli courbe*) behind; belonging to the temporal lobe we have the three temporal convolutions, of which the first (uppermost) lies between the fissure of Sylvius and a very deep fissure running parallel to it, the so-called parallel fissure or first temporal fissure. The fissure of Sylvius has two branches, and the portion of the cortex between them is called the "operculum." Belonging to the occipital lobe, finally, there are three irregular and not always easily distinguishable occipital convolutions, between which two occipital fissures have been described.

In order to determine from the outside of the skull the position of the fissure of Rolando we proceed, according to Köhler (Deutsch. Zeitsch. f. Chir., 1891, xxxii, 5, 6), in the following manner (cf. Fig. 39): A line, *a*, is drawn over the middle of the skull from the forehead to the external occipital protuberance. A second line, *b*, is drawn at right angles with this, passing through the anterior boundary of the external auditory meatus; parallel with this second line we draw a third line, *c*, passing through the posterior margin of the mastoid process, so that it cuts the sagittal line, *a*, two inches behind the line *c*. A fourth line, *d*, starting from the point where *a* and *c* intersect, and running obliquely downward so that it meets the line *b* two inches above the external auditory meatus, will indicate the direction of the central fissure.

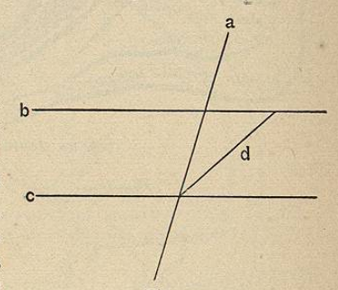

Fig. 39.

On the basal aspect we see those parts of the three frontal convolutions which are included in the base, of which the first (uppermost) is here called the gyrus rectus; then the tractus, with the sulcus olfactorius; next the uncinate gyrus, which belongs to the gyrus fornicatus, and which will be better seen on the median aspect; further, the basal part of the third temporal convolution (gyrus temporalis inferior) and two lobules, which belong to both the temporal and occipital lobe, the inner (median) one called the lingual lobule, the one more external the fusiform lobule.

The median surface (Fig. 40) shows in the middle the corpus callosum (in front the genu, behind the splenium); immediately below is the "septum lucidum," immediately above the gyrus fornicatus, the temporal part of which is called the hippocampal convolution, and is continuous with the uncinate gyrus. Above the gyrus fornicatus, and separated from it by the calloso-marginal fissure, are the frontal convolutions; farther back, the paracentral lobule, which