meets the central convolutions. Behind this and belonging to the parietal lobe is the præcuneus, and still farther back the cuneus (of

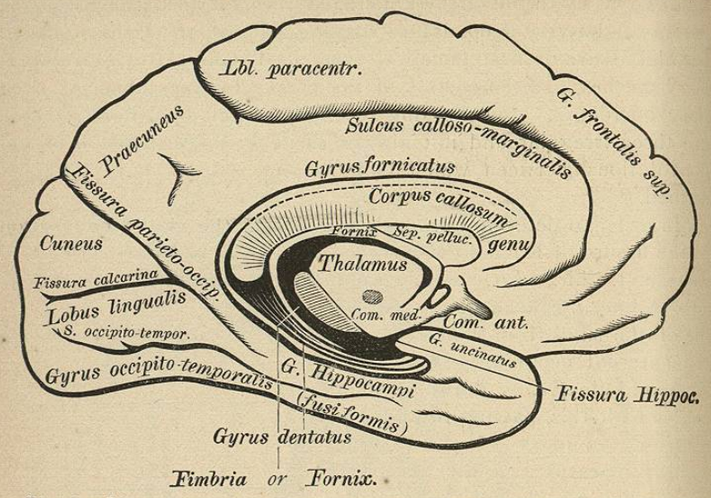

Fig. 40.—Convolutions and Fissures of the Median Aspect of the Brain. The posterior portions of the thalamus and the crus cerebri are cut off.

the occipital lobe). The latter is bounded by two deep fissures—below by the calcarine, in front by the occipito-parietal fissure.

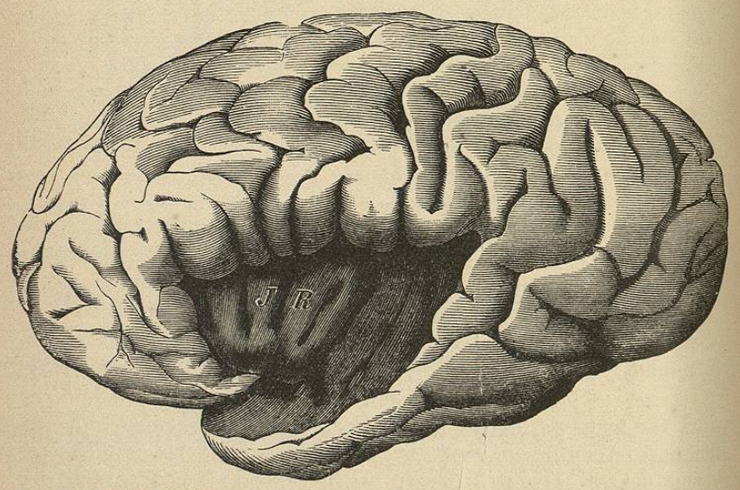

Fig. 41.—Convolutions of the Island of Reil (J. R.) made visible by removing the Operculum.

Extending from the posterior part of the corpus callosum to the uncinate gyrus is the hippocampal fissure.

Covered by the above-mentioned operculum, in the depth of the fissure of Sylvius, is the lobus intermedius s. opertus, the so-called island of Reil, on which five to seven small convolutions are seen. Their position is shown in Fig. 41, where the operculum has been removed.

In Fig. 42 the topographical relations between the surface of the brain and the surface of the skull are illustrated.

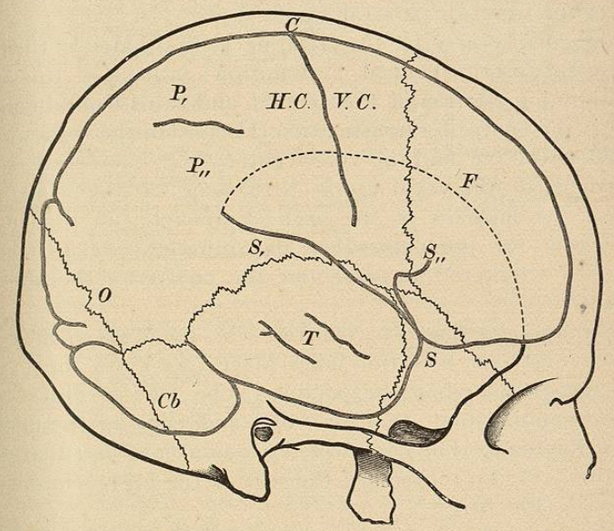

Fig. 42.—Topographical Relations between the Exterior of the Skull and the Surface of the Brain. (After Ecker.) *C*, fissure of Rolando. *V. C.*, anterior central, *H. C.*, posterior central convolution. *S S, S,,*, fissure of Sylvius. *T*, temporal lobe. *F*, frontal lobe. $P_{,}$, upper, $P_{,,}$, lower parietal lobe. *O*, occipital lobe. *Cb*, cerebellum.

As stated above, the localization of the motor centres by Fritsch-Hitzig and that of the speech centre by Broca paved the way for a number of discoveries which, based partly upon clinical observations, partly upon the less trustworthy experiments on animals, eventually will lead to a complete and accurate physio-pathological topography of the brain cortex. Thus far our knowledge is scanty and uncertain, and the centres which we shall here describe as being determined are almost all relative, in the sense of Exner (cf. page 165), the only exception being the so-called motor region of the cortex. On the right hemisphere, the paracentral lobule, the anterior central,

and the upper half of the posterior central convolution, on the left hemisphere, the paracentral lobule, the upper three fourths of both central convolutions, and a part of the upper parietal lobule, constitute the absolute cortical area for the upper extremities (Exner). The absolute cortical area for the lower extremities is situated, on the right hemisphere, in the paracentral lobule, and in the upper third of both central convolutions; on the left hemisphere, in the paracentral lobule, the upper half of the posterior central convolution, and the greater part of the superior parietal lobule.

The cortical area for the facial nerve is situated in the lower end of the anterior central convolution; in front of this and in the adjoining portion of the second and third frontal convolutions is the centre for mastication (Hirt). In the region of the island of Reil we find the voice-centre—i. e., the centre for the movement of the vocal cords (Rossbach); in the frontal lobe that of the muscles of the neck (Fraenkel); in the angular gyrus that for the external ocular muscles. Haab (Zürich, 1891) has attempted to determine the centre for the pupillary reflex.

Of the so-called sensory centres—i. e., the areas in the cortex where conscious sensation takes place—we know the psycho-optic to be situated in the occipital lobe; the psycho-acoustic in the temporal lobe; that for smell and taste in the uncinate gyrus (Ferrier). The cortex of the frontal lobe and that of the temporo-occipital region are the seat of the higher intellectual processes (Flechsig).

The so-called thermic centre discovered by Eulenburg and Landois corresponds to the motor region, and the tactile regions for the different parts of the body also are identical with the motor centres (Exner, Tripier).

Further investigations must show whether the centres which we have been wont to regard as being situated in the medulla oblongata—for example, the centre for salivation, that for deglutition, that for the movements of the stomach and intestines (vomiting and defecation), for sneezing, coughing, etc.—are also situated in the cortex. The results of the treatment by suggestion make the assumption of such centres necessary. Nevertheless, while the "area of latent lesions" (Exner) is still as large as it is at present, an explanation of this kind is premature.

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The speech centre, which, as we have seen, is situated partly in the frontal, partly in the temporal lobe of the left hemisphere, is certainly of larger extent than is commonly supposed. It is well known that after Bouillaud in 1825 had pronounced the frontal brain, and Marc Dax in 1836 the left

hemisphere, to be the seat of speech, Broca claimed that the posterior part of the third left frontal convolution, the pars opercularis, or, as it later was called, the region of Broca, contained the speech centre; and, indeed, in speech disturbances a lesion of this very region has most frequently been found at the autopsy. There are, however, other parts of the cortex, as the island of Reil, the central convolutions, and, above all, the temporal lobe, more especially its upper convolution, which are connected with speech and which are of no less importance. Thus we have, after much laborious work and after many careful observations and comparisons, come to the conclusion that a different form of speech disturbance (aphasia) is produced according as the lesion is one of the frontal or of the temporal lobe—(of the left side only). In the former case the patient knows the word which he wishes to pronounce, but can not do so because he has lost the memory for the movements necessary for speech—i. e., he no longer knows how to use his tongue and lips in the act of speaking—motor aphasia. If the lesion is situated in the third left frontal convolution (Broca's region), we speak of cortical motor aphasia; if it is situated in the white matter of the hemispheres, in the posterior portion of the internal capsule, or in the left crus, we speak of subcortical motor aphasia.

In the latter case—i. e., if the lesion is in the temporal lobe—the patient knows exactly what he wants to say, and he has no difficulty in repeating it if it is spoken for him; but he can not find the expression for himself, he has "forgotten" the word—sensory aphasia. That the understanding of words is situated in the temporal lobe, more particularly in the first temporal convolution, was first stated by Wernicke, who also originated the terms motor and sensory, cortical and subcortical, aphasia. The anatomy of the subcortical sensory aphasia has as yet been only imperfectly studied.

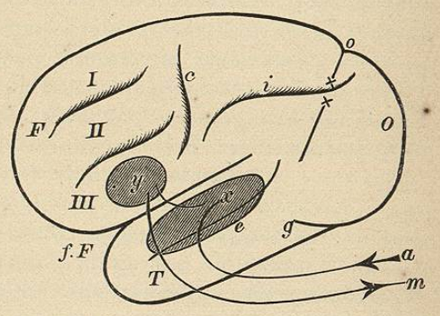

Fig. 43.—Wernicke's Schema for the Cortical Mechanism of Speech.

In the diagram of Wernicke which is shown in Fig. 43, *y* represents the motor, *x* the sensory speech center; the latter

is the terminus of the centripetal path of the auditory nerve *a x*, the former is the beginning of the centrifugal path *y m* going to the muscles used in speaking; *x y* an assumed association path between both; *y* is situated in the third frontal, *x* in the first temporal convolution. According as one or the other of these centres or the connection between them, or both centres, were destroyed, Wernicke distinguished four cardinal types of aphasia:

1. Destruction of the centre *y*—motor aphasia. Mobility of the muscles used in speech is retained, but the patient can either not speak at all or only say a few words or syllables. Understanding and memory of words are intact.

2. Destruction of the centre *x*—sensory aphasia, "word deafness" (Kussmaul). The patient can use as many words as ever, but in speaking they are mixed up. The understanding of words is lost, although the power of hearing is not interfered with.

3. Destruction of the association path *x y*, situated in the insula (?)—the so-called conduction aphasia of Wernicke. The patient can use as many words as ever, but in speaking they are mixed up. The understanding of words is retained.

4. Destruction of both centres, *x* and *y*—total aphasia. Power and understanding of speech are lost.

If we then consider as proved that a certain group of motor and sensory memory pictures are localized in the brain; if we further agree that the former correspond to certain groups of muscles which serve a common purpose, the latter to the distribution of a sensory nerve—it is not difficult to conceive that this same arrangement may exist for all the muscles and for all the sensory nerves. It is certainly easy to understand the occurrence of other motor defects in cases of aphasia. Thus there may be loss of simple movements (e. g., of the power to put out the tongue), or more complex ones (e. g., writing may become impossible—agraphia; *aphasie de la main*, Charcot). Again, we have a patient who, in consequence of a cortical lesion in the central termination of the optic nerve, no longer recognizes his letters, and has thus lost the faculty of reading ("alexia"); or the visual memories may be lost altogether (not only those of letters), and a condition ensue which Munk calls psychical blindness.

In examining a patient affected with aphasia, with a view of determining which path has become interfered with, we may

meet with considerable difficulty, and the diagnosis of the particular type of aphasia with which we are dealing is often not easy, for the cases are not so sharply defined or so well characterized as we might be led to expect from the simplicity of the schemata. On the contrary, we often meet with combinations of the different types or with transition forms of aphasia in which even the most experienced clinician will venture a differential diagnosis only with much reservation. Take, for instance, the different degrees of that form of speech disturbance known as ataxic aphasia, in which the patient is unable to pronounce a word, though it constantly is floating, as it were, before his mind. This inability may go so far that the patient can only pronounce a few words or syllables (monophasia), that he involuntarily confounds words without being in the least uncertain about their meaning; or it may, on the other hand, only amount to a slight disturbance, shown by some misplacement or omission of some letters, as in saying *dy* instead of *dry*, *turk* instead of *truck*, and the like. In the latter case we speak of syllable-stumbling (*Silbenstolpern*). Likewise we have different degrees of the so-called amnesic aphasia, where there may be loss or only slight impairment in the memory for words (sometimes only for words of foreign languages which have been learned later in life). As the faculty of writing and reading may often be more or less altered, it is important that it should be minutely examined into; the patient is asked to spell individual words, then to read sentences without spelling, then to write spontaneously and to dictation, and finally to copy words. In the case of a patient who is left-handed, his ability to write with the left hand should always be tested. Every case of aphasia must be carefully studied by itself, and each one gives opportunity for interesting observations.

In general we may be guided by the following rules:

1. If we find a patient whose sanity is established, who possesses a normal acuteness of hearing and understands what is said, but is unable to repeat sentences or to speak spontaneously, and can only utter individual words and syllables, we may assume a lesion of the third frontal convolution, possibly of the lowest part of the anterior central convolution.

2. If a patient, although able to speak without difficulty, does not understand simple questions, then the first temporal convolution is diseased (*in toto*). If the understanding of words is only impaired, then only a part is affected.

3. If the patient has lost the faculty of reading, although there is no motor aphasia to be noted, we have to deal with a lesion of the cortical centre for vision (cf. page 172).

4. A disease of the cortical speech centre does not exist if the patient gradually regains speech which he had suddenly lost; if in such a case the hemiplegia, which has simultaneously appeared after an apoplectic stroke, persists, the white substance near the cortex is usually diseased (Gowers).

We should be going beyond the limits of this work if we attempted to discuss the aphasic symptom-complex in all its difficult and not rarely obscure details; there exist a large number of interesting special articles on this subject, to the most important of which references will be found at the end of this chapter. While recognizing the steady advance which has been made toward the interpretation of these most complicated disturbances, we are ever reminded, by the constant difficulties which arise, how far we are from a complete understanding of them. Almost every case shows peculiarities which do not fit into any of the schemata; and while to-day a successful investigator claims to have cleared up some obscure point in the difficult field of aphasia, to-morrow another one proves that this conclusion was after all too hasty, and that only he, the second investigator, has really settled the question. In a word, there is hardly a single point in the problem of aphasia which is not still the subject of controversy. The tendency to schematize is very prevalent in Germany, and in opposition to these too schematic conceptions of aphasia, English and French investigators have pointed out the variations of the inner speech —i. e., of the thinking processes necessary for speech—and the differences which may be bound up with the individual peculiarities of the person who speaks, writes, or reads. But these objections are slow to be appreciated in Germany. Whether a person reads by spelling, or whether after considerable practice one may read without spelling, whether the optical images of letters are necessary in the process of writing or not—these and many similar problems still await their solution, and can be cleared up only by untiring, careful observation of cases.

For the beginner it is not only desirable but necessary to have the matter presented to him somewhat dogmatically, and this, according to our experience, will be best and most easily accomplished with the aid of schemata, of which, besides

the above-mentioned one of Wernicke, quite a number have been brought out. The one we have deemed most suitable and the best fitted for teaching purposes is probably that which Lichtheim has developed (Arch. f. Psych., 1884, xv, 3). It has been here given in Figs. 44, 45.

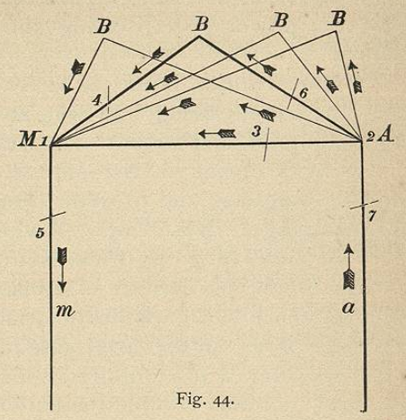

Fig. 44.

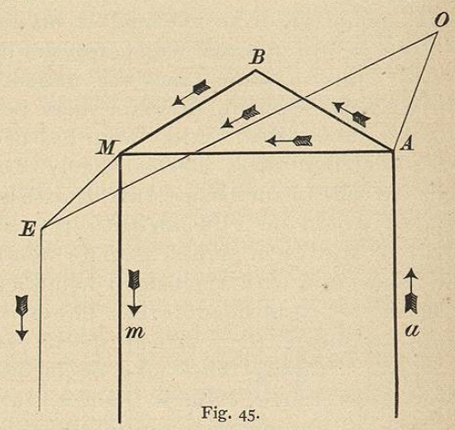

Fig. 45.

Figs. 44, 45.—LICHTHEIM'S SCHEMA ILLUSTRATING THE SEVEN DIFFERENT FORMS OF APHASIA. *a*, *A*, centripetal path for auditory impressions. *A*, centre for auditory images. *M*, centre for motor images. *M*, *m*, centrifugal motor path. *B*, the place where concepts are formed. *O*, the centre for visual images. *E*, centre from which the organs of writing are innervated. (The seven different forms of aphasia have been indicated in Fig. 44 by the numbers 1–7.)

The reflex arc necessary for repeating words contains the centre for auditory images of words, *A*; the centre for motor images, *M*; the centripetal path for auditory impressions, *a A*; the connecting path, *A M*; the centrifugal motor path, *M m*. *B* is the place where concepts are formed—voluntary speech necessitates a centrifugal path from *B* (brain cortex) to *M*. *O* is the centre for the visual images of letters. *E* is the centre for the innervation of the muscles required in writing. Now, according to the path affected, we distinguish seven different forms of aphasia.

1. Interruption in the point *M*. Broca's (motor) aphasia.

2. Interruption in point *A*. Wernicke's (sensory) aphasia.

3. Interruption in the path *M A*. Conduction aphasia (Wernicke).

4. Interruption in the path *M B*. A variety of motor apha-