sia; the faculty of repeating words and sentences being retained.

5. Interruption in the path *M m*. Variety of motor aphasia; the power of expressing thoughts in writing being retained.

6. Interruption in the path *A B*. A variety of sensory aphasia; the patient being, however, able to repeat spoken language, to read aloud, and write from dictation.

7. Interruption in the path *A a*. Inability to understand spoken language and to write from dictation, or to repeat spoken language. Nos. 6 and 7 have so far been observed only in rare instances (e. g., by Pick, Neurol. Centralbl., 1890, 21).

As to the occurrence of aphasia, it is most frequently seen as a sequence to an apoplectic attack, either as a direct or indirect symptom; in the latter case it is transient, and lasts, as we shall see later, a few minutes, hours, or days. In the former it persists, and may trouble the patient, though he may retain his full mental vigor to the end of his life. The most common form is motor aphasia, which appears in widely different gradations; thus, in some cases the patient's speech may be just a little thick, while in others it may be altered so that it is no longer intelligible. After what has been said, it is easy to understand that these defects chiefly occur after hæmorrhage in the left side of the brain—that is, with a right-sided hemiplegia; but it would be a decided error to suppose that they occur only or always in those cases, for motor aphasia may be found in connection with a left-sided hemiplegia, and it may be wanting in the right-sided form. Other diseases of the brain also may implicate the cortical speech centre and give rise to aphasia. Among these may be mentioned general paralysis, psychoses (Lloyd, Francis, Lancet, July 7, 1888), processes of softening, chronic meningitis, tuberculous deposits, etc., and traumatism of the left hemisphere, in which case aphasia may be the only symptom. Aphasia has also been observed in acute, especially infectious, diseases—e. g., typhoid and scarlet fever. Most instances of this latter form occur in children. It has also been observed in the puerperal state. Of special interest is that form of total or motor aphasia which sometimes suddenly, sometimes gradually, comes on after a fright. That after a fright, such as makes "the hair stand on end," the voice may refuse to perform its duty, even Virgil seems to have known full well, as we see from the verse, "*Steteruntque comæ, vox fau-*

cibus hæsit." The nature of this form is uncertain, still it is by no means impossible that, just as we find that vaso-motor spasm acting on the facial vessels will produce pallor, so we may have a similar condition in those finest distributions of the middle cerebral artery which supply the region of Broca. That the spasm in these vessels is usually of longer duration and produces more serious and more lasting consequences than the spasm of the cutaneous vessels, may be explained by the difference in their arrangement, as well as by the difference in the function of the parts which they supply.

It is not organic changes of the cortex which produce the symptoms in this case, the disturbances being entirely of a functional character, and this fright aphasia therefore constitutes a transition form to those instances in which, though the aphasia may have lasted for years, no changes are found at the autopsy, either in the cortical or subcortical area for speech. No doubt there is, besides the aphasia due to actual lesions in the cortex, also a functional form which we may imagine to originate in different ways, and it is at least probable that variations in the blood supply of the centres play an important part in this connection. Grashey (cf. lit.) has shown, in an ingenious piece of work, that we have to recognize a third form of aphasia, in which neither the centres nor the conducting paths are insufficient in their functions, but which is simply due to a diminished duration of the sensory impressions, giving rise to a disturbance in perception and association, and thus to an aphasic condition. Maybe it is this aphasia of Grashey which we find after concussion of the brain and after acute diseases, but it is difficult to diagnosticate it, and to differentiate a functional disturbance of the centres from a diminished duration of sensory impressions. A correct diagnosis is, however, of no small importance in the question of prognosis.

The outlook is absolutely unfavorable in cortical lesions where the centre is destroyed by processes of softening, tuberculous deposits, atrophy of the gray cortex, etc., but is, of course, materially better if the centres remain intact, and are only temporarily rendered unable to perform their function, for then speech returns gradually, if not wholly, partially, and it can not be denied that systematic exercise and regular instruction in speaking are capable of hastening an amelioration, nay, even a cure, especially if the patient be still young.

The aphasia of children, which we sometimes find after

acute infectious diseases, fright, or as a consequence of intestinal worms ("reflex aphasia"), in the course of acute infantile cerebral palsy, or of epilepsy, and occasionally, but very rarely, after a cerebral hæmorrhage, is in no other way to be distinguished from the aphasia of adults except in its prognosis. Children, *cæteris paribus*, always stand a better chance of improvement or recovery from aphasia than adults, no doubt because it is easier to educate in them the well half of the brain to perform the function of the damaged one. If the disturbance is only functional, as I saw in one case which was due to an overdose of santonin, in which the disorder in speech only lasted a few hours, the outlook is still more favorable, and complete recovery may be confidently expected; but if the function of one speech tract—that is, the left—be impaired by cortical or deep-seated lesions, even then it is in children usually not very difficult to educate the right side to some vicarious action, especially in cases where, before the lesion, the children have been taught to use both hands equally. The possibility of a cerebral disease should be thought of in the gymnastic cultivation and development of the body of children; the extremities of both sides should be exercised and strengthened equally, the children should be made ambidextrous; only then can, in a case of necessity, the right hemisphere fully take the place of the left.

A treatment for the aphasia as such does not exist. In cases of functional aphasia the only thing necessary is to convince the patient that his condition is not serious. If this does not lead to any improvement, we should try hypnotism, from which astonishing results have sometimes been obtained. If, on the other hand, the aphasia is due to organic changes, such as hæmorrhage or embolism, in our treatment we must be guided by the principles discussed in the chapter dealing with these conditions.

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We have repeatedly pointed out that the motor disturbances produced by cerebral diseases are either due to destructive or irritative lesions. The former consist of paralyses and pareses, the latter of involuntary movements in different groups of muscles—the so-called spasms. Those disturbances which are due to affections of the cortex (cortical motor disturbances)

present much that is characteristic and interesting. They will be considered presently.

The motor centres, the motor area of the cortex, comprise, as has been stated above, the two central convolutions, the paracentral lobule, and the parts lying immediately adjacent. Upper and lower extremities, neck and face, have their own

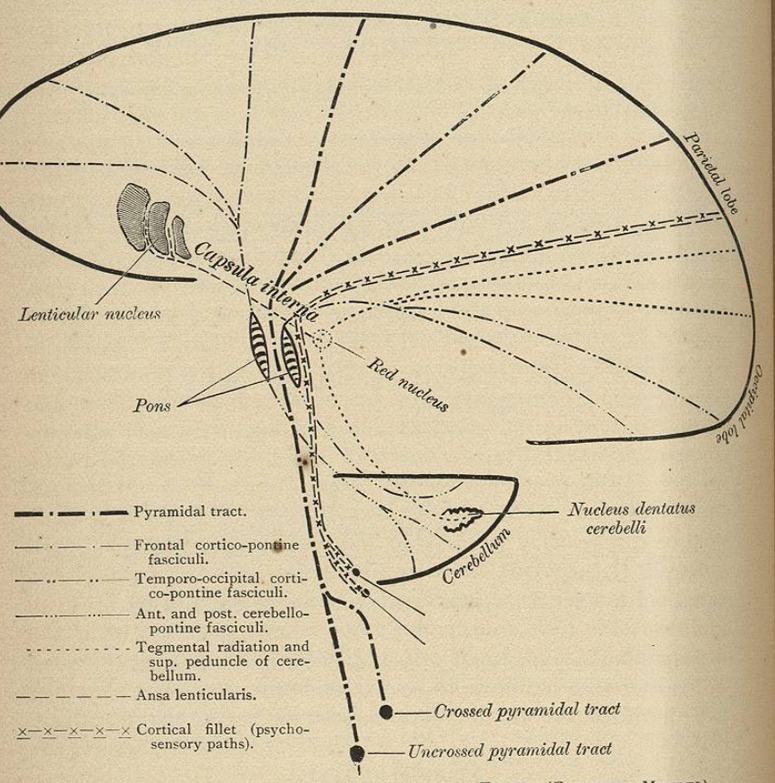

Fig. 46.—Diagram showing the Direct System of Fibres (Flechsig, Mendel).

special centres, which are distinctly separated from one another in the central convolutions. Thence fibres converge, as is shown in Fig. 46, into the anterior two thirds of the posterior division of the internal capsule. One thing at once becomes apparent from this arrangement, namely, that in cortical lesions the paralysis or paresis may easily be confined to one extremity, an arm or a leg, while if the lesion affects

the tract lower down in the brain—for instance, in the region of the capsule—the paralysis must extend over the whole half of the body. A paralysis of one extremity only is called a monoplegia (monoparesis), in contradistinction to a hemiplegia, which means paralysis of one side (half) of the body, and it is a perfectly warrantable conclusion, sufficiently confirmed by post-mortem evidence, that, if the patient presents a paralysis of only one arm or one leg, we are dealing with a cortical lesion. A hemiplegia is only then likely to be of cortical origin if its development indicates that the lesion beginning in one motor centre has gradually encroached upon another. It goes without saying that in diagnosticating cortical lesions we must not rely on a single symptom, but all must be considered, and especial care must be taken not to confound a paralysis of cortical with one of peripheral origin. One great distinction between these two is to be found in the manner of onset. While a paralysis of cortical origin may develop quickly in a few hours, a peripheral one will be more gradual, and only reach its full extent after weeks or even months. Moreover, the latter, the peripheral, is easily recognizable by the changes which take place in the electrical excitability—e. g., if reaction of degeneration and visible atrophy in the muscles can be demonstrated. The absence of cerebral symptoms, which are rarely entirely wanting in cortical affections, is also characteristic of peripheral disease. Great pain may be entirely absent in the central, but is commonly present to a greater or lesser degree in the peripheral variety. Remembering, then, these points, and making it a routine practice never to omit the electrical examination in doubtful cases, we are not likely to make an error in the diagnosis.

In cortical lesions the loss of motion is usually not absolute, and we find more frequently a paresis than an actual paralysis. The disorder does not necessarily affect a whole extremity, an arm or a leg; it may be confined to the distribution of special nerves, or even to portions of these, the so-called dissociated hemiplegias (cf. also Pick, Prag. med. Wochenschr., 1891, 25–27). Sometimes the affected arm or leg can be moved *in toto*, though a strong effort may be required, and it is only in the fingers and toes that the loss of power is complete.

A characteristic symptom is the inability of the patient to execute complicated movements, such as buttoning his coat, counting money, and so forth, acts which are performed awk-

wardly and with difficulty, owing to a loss of the motor images. This condition has been called ataxia, and in these cases we have a "cortical ataxia." The lesion has to be referred to that part of the cortex which contains the sensory area (*Fühlsphäre* of Munk) for the affected, that is, the ataxic extremity. The trouble is very distressing to a patient in a brachial as well as in a crural monoplegia, and becomes almost unbearable if the sensory disturbances, which we shall shortly describe, are superadded (cf. lit., Observations of Bernhardt).

In infective tumors, gummata, tubercles at the surface of the brain, we occasionally meet with symptoms of irritation, such as monocontractures, which depend upon an irritation in the corresponding portion of the motor path (Wernicke). They are not seldom accompanied by sharp pains. In such cases the differential diagnosis between a hysterical and a true organic cortical lesion may cause considerable difficulty (cf. chapter on Hysteria).

Of the greatest practical importance are the epileptiform attacks which occur, either with or without loss of consciousness, as a consequence of direct or indirect irritation of the cortex. If they occur in the further course of the monoplegia, the onset of which was apoplectiform, the diagnosis of a cortical lesion can be made with a high degree of probability. In some cases the convulsions are not general, but only appear as localized twitchings or spasms, confined to one half of the body or one extremity; they may be clonic (that is, an alternation in quick succession of contractions and relaxations) or tonic (that is, steady contractions lasting for some time), and may be of considerable intensity; their occurrence later in parts already paralyzed would indicate a disease of the brain surface, though we may not always be able to say whether the irritation of the cortex depends upon a direct or—as, for instance, in tumors, which cause an increase of the intracranial pressure—an indirect action. In the latter case, also, general or partial convulsions may ensue.

The use of the term cortical epilepsy (or Jacksonian epilepsy, after Hughlings Jackson, who first described these conditions) is liable to give rise to misconceptions, and it must be remembered that the so-called cortical epilepsy has nothing in common, except the name, with the classical genuine epilepsy.

The epileptiform seizures due to cortical lesions show certain fundamental differences from the classical attacks. Con-

sciousness is retained, a feature which gives the whole attack an entirely different aspect. A certain kind of aura occurs here also; the patient knows when the convulsions are coming on, either by slight twitching in the fingers or toes, or by formication and other symptoms, which occur only in the affected extremity. But all the other symptoms—the cry, the fall, the biting of the tongue, etc.—are absent. The patient sees and watches the twitching of his extremity; not rarely he has violent pains; he tries to hold the extremity in a fixed position or asks others to do so, and attempts to avoid injuring himself. After the convulsions he feels weak and unstrung, but only in consequence of the increased muscular work. Headache and all the various post-epileptic symptoms are absent, or, at any rate, are not connected with the attack as such.

The degree, the duration, and the frequency of the attacks vary considerably; sometimes only a more or less marked twitching appears in the affected limb; sometimes, however, the attack manifests itself in shaking movements, which may become so violent that the bed shakes and the patient anxiously cries for some one to assist him and to hold him. If violent pains have been present during the movements, they are wont to persist after the attack, and, combined with the motor weakness in the affected extremity, are often productive of great suffering. The duration also varies. I have seen cases in which the attack was over in from a quarter of a minute to one minute; on the other hand, I have seen instances in which it has lasted for a quarter of an hour. If such prolonged attacks occur at frequent intervals—two, three, or six times a day—the state of the patient may be very pitiable; and, indeed, the carrying on of the individual's occupation may be interfered with by this partial epilepsy much more than it often is in cases of the classical disease. In other instances months intervene between the attacks. The whole course of the malady is eminently chronic; the patient may suffer for years, or tens of years, without there being any other symptom present. Death occurs either from an extension of the brain lesion or as a result of some intercurrent disease. Pitres has called attention to the fact that so-called equivalents may occur in Jacksonian epilepsy also, and has pointed out that they may be of a sensory or of a psychical nature (Revue de méd., 1888, viii); the former belong to Charcot's *épilepsie partielle sensitive* (Leçons du Mardi à la Salpêtrière, 1889, pp. 20 and 368); the latter manifest themselves

in visual, auditory, or olfactory hallucinations without any marked signs of motor irritation.

With reference to the diagnosis, it should be mentioned that, just as in the case of genuine epilepsy, cortical epilepsy may be simulated by uræmic attacks if the latter are confined to one side (Chauffard, De l'urémie convulsive à forme de l'épilepsie Jacksonienne, Arch. gén. de méd., July, 1897). Furthermore, attacks which resemble very closely those of Jacksonian epilepsy may occur in hysteria; in these cases the presence of other hysterical manifestations will prevent an error in diagnosis. Mendel has repeatedly observed cases of general paralysis in which Jacksonian epilepsy was the initial symptom. The foci which were found at the autopsy were in each case situated in the right psycho-motor region, and the (paralytic) speech disturbance occurred in the terminal stage, whereas usually this is one of the early symptoms of general paralysis.

The sensory disturbances which are produced by the affections of the brain cortex are remarkable, and by no means fully understood. As we have seen before, they do not, as a rule, cause pain, but rather manifest themselves in alterations of sensation, known as paræsthesias. Thus the patient may speak of a curious numbness or deadness; or, again, he may have a sensation as of ants crawling under the skin, a feeling as if the part had gone to sleep, etc. There may also be a distinct increase in pain perception, a slight "analgesia," a diminution or loss of pressure, touch, and temperature sense, and oftener, as it seems, in diseases of the parietal lobes a more or less pronounced disturbance of the muscular sense, in consequence of which the patient can with closed eyes either give no account at all or only a very imperfect one of the position of his extremities. If, as often happens, the above-described awkwardness in motion (ataxia) coexists with these changes, we may be tempted to refer the trouble not to the cortex, but to the spinal cord; more especially are we liable to think of tabes, although the ataxia is produced in an entirely different manner in the two diseases. However, the differential diagnosis will in most cases present no difficulties if we take into consideration all the symptoms, and examine into the condition of the patellar reflexes, the reaction of the pupils, and ascertain whether there are bladder symptoms and whether lancinating pains are present or not. These sensory changes, we must not forget, are

by no means always observed in cortical lesions, and in the cases in which they existed the white matter of the brain has often been found to be likewise the seat of disease; they are therefore in no way to be regarded as pathognomonic, and we have to be cautious in using them for diagnosis. The same is true to a greater degree of the vaso-motor and trophic changes, the relation of which to the brain cortex is still obscure.

Symptoms Referable to Lesions of the White Matter of the Hemispheres and Lesions of the Basal Ganglia.

Looking at the anatomy of the parts, we notice that the fibres coming from the cortex pass through the white matter of the hemisphere, which in the region of the frontal and parietal lobes is

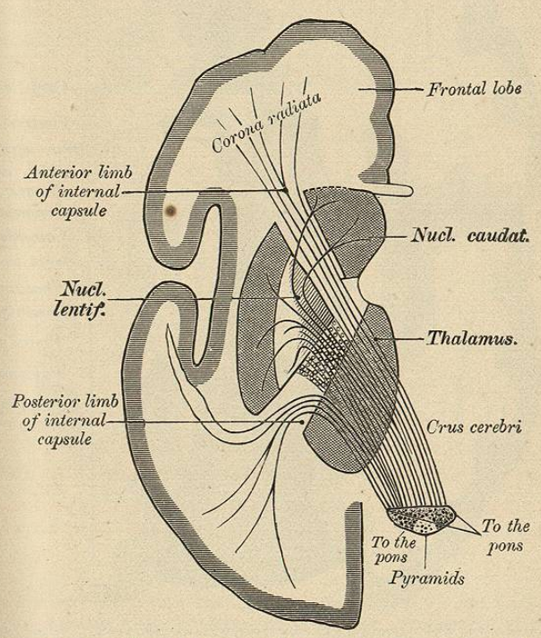

Fig. 47.—Course of the Fibres from the Internal Capsule to the Crus Cerebri. The thalamus is represented as transparent. (Diagrammatic after Wernicke and Edinger.)

designated centrum semiovale Vieussenii. Turning toward the brain-stem, in its neighborhood they appear arranged in bundles placed side by side, completing by their convergence what has long been known as the corona radiata. With this corona radiata begins the