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

but progressive improvement. Recovery takes place either rapidly or not at all in the large majority of cases.

The further course of the disease can be distinguished in no respect from that of cerebral hemorrhage. As we have stated in our discussion of the latter subject, even a post-mortem examination may fail to distinguish in old cases between the remains of a clot and an embolic process, inasmuch as the embolus itself may gradually have been absorbed and the original brain lesion replaced by a cyst or cicatrix.

For the changes in the symptomatology due to the varying location of the embolus, we must again refer the reader to the article by Dr. Starr, on *Brain Diseases: Diagnosis of Local Lesions*.

But we will here describe a very peculiar group of symptoms, apparently of bulbar origin, which has been observed in a number of instances in which the lesion was situated in another part of the brain. The clinical history of this form of disease is well shown by the following case reported by Berger:

"A man, aged sixty-one, suffering from small, contracted kidneys. In 1883 dysarthria and dysphagia developed suddenly without any violent apoplectiform symptoms. At the end of two days right hemiparesis and salivation were observed. Striking improvement after local faradization. About a year later an attack of tonic rigidity of the body, followed by an exacerbation of the disturbances of speech and deglutition, and also of the right hemiparesis. Paralysis of the orbicularis oris, particularly on the right side, and of the muscles of the tongue and deglutition. Inconstant condition of the glosso-labio-pharyngeal paralysis; no progressive course. No atrophy; normal electrical irritability of the paralyzed muscles."

Quite a number of autopsies have shown that symptoms of this character may be due to a bilateral or even unilateral lesion of the cerebral hemispheres in any position which interferes with conduction from the cortex to the centres of articulation and deglutition in the medulla oblongata, though the latter may be entirely intact. A case of this description, in which I was fortunate enough to obtain an autopsy, was seen by me only a few days ago. In this instance a spot of softening was situated in the anterior third of the posterior half of the right internal capsule, and also involved slightly the anterior part of the optic thalamus.

Diagnosis.—The diagnosis of cerebral embolism has been considered in part in the discussion of cerebral hemorrhage, so that very little need now be said on the subject.

One combination of symptoms, viz., the sudden occurrence of right hemiplegia and aphasia in a patient who has previously presented no cerebral symptoms, is extremely characteristic of embolism of the left middle cerebral artery. This group of symptoms is observed with comparative rarity as the result of any other lesion, though, of course, a hemorrhage occurring in the internal capsule may give rise to the same symptoms. In the latter event, however, the disturbance of consciousness is usually more severe than in the former, and, in addition, the onset of the disease is generally not so sudden.

The development, during an apoplectiform attack, of an embolism of the central artery of the retina is very significant of a similar affection of one of the cerebral vessels. Symptoms indicative of the formation of infarctions in the spleen, kidneys, and lungs may also aid us in coming to a conclusion.

Youthful age and the presence of valvular disease of the heart also point to embolism rather than hemorrhage. In very young children and infants, however, cerebral embolism is much rarer than hemorrhage.

If aphasia occurs as the sole symptom the diagnosis must be made from the attendant circumstances, such as the existence of cardiac disease, the occurrence of previous attacks of a clearer nature, the age of the patient, and the subsequent history of the disease.

Berger states that the pseudo-bulbar symptoms mentioned in the preceding section are distinguished from glosso-labio-pharyngeal paralysis by the following differential features:

1. Sudden apoplectiform development of the symptoms, which present no tendency to progression.
2. The presence of other cerebral disturbances.
3. The absence of atrophy of the paralyzed muscles, even after the disease has lasted a considerable time.
4. The absence of the reaction of degeneration.
5. The intact reflex irritability of the paralyzed muscles.
6. The absence of signs of progressive muscular atrophy.

Prognosis.—This depends mainly upon the situation and character of the artery—whether terminal or not—in which the embolus has lodged, and upon its complete or incomplete occlusion. The latter factor is of comparatively slight importance, since incompletely occluding emboli usually are converted into completely occluding ones from the secondary deposit of a thrombus. As a matter of course, the occlusion of one of the vessels supplying the pons or medulla may prove rapidly fatal.

If improvement occurs it usually takes place within one or two weeks. Otherwise the symptoms remain stationary or are complicated at a later period with secondary contractures, etc., as in cerebral hemorrhage. According to our experience, there is less danger of mental impairment than in cerebral hemorrhage. Relapses may occur at any time from the continuance of the primary cause. Death occurs much less frequently than in hemorrhage of the brain, unless the embolus lodges in the basilar artery.

CEREBRAL THROMBOSIS.—*Etiology.*—Thrombosis of the cerebral arteries is generally the result of atheromatous degeneration of the vessels. In such cases the current of blood is also usually slowed on account of weakness of the heart's action, and coagulation, therefore, takes place upon the roughened parts of the inner coat of the vessel. The deposit of coagulated blood gradually increases in extent until, as a rule, the lumen is entirely occluded. In rare instances, however, a parietal thrombus forms, leaving part of the lumen free for the passage of blood.

As we have stated previously, it is quite common to find, also, that a secondary thrombus is deposited upon a cerebral embolus, whatever the origin of the latter may have been.

A syphilitic affection of the walls of the vessels may lead to thrombosis in the same way that atheroma does, but the syphilitic vascular lesions have been discussed under a separate heading (see *Brain: Syphilis*).

As atheroma of the vessels is usually one of the accidents of advancing age, so thrombosis is also most frequent after middle life.

Another variety of cerebral thrombus is that known as the marasmic thrombus. This develops in intact vessels as the result of simple retardation of the current of blood (perhaps associated with a change in its chemical constitution), and is observed usually after protracted, exhausting diseases or severe attacks of an acute infectious disease. This variety is also observed in childhood, from similar causes.

Another form of thrombosis is the result of direct compression of the vessel, either from a mass of surrounding exudation, particularly in tuberculous meningitis, from the growth of a tuberculous new formation through the wall of the vessel, or from the pressure of a tumor of the brain. In such cases, however, the clinical history of the thrombus is usually lost in that of the primary disease.

Clinical History.—Cerebral thrombosis is frequently preceded by prodromal symptoms, which vary greatly in character and duration, lasting at times for many months. These symptoms are due probably to the impaired nutrition of the brain, on account of the slowly increasing diminution of the elasticity and calibre of the affected vessel.

Dizziness is one of the most common prodromal manifestations; it may be constant or intermittent, and is often aggravated by any sudden change in position. Violent headache is more infrequent, and is usually diffused over the entire head. The patient grows moody and morose, the memory is weakened, and impairment of speech may also be noticed. Sometimes intercurrent attacks occur, in which the individual partly loses consciousness, experiences numbness in an arm or leg, and, perhaps, has a slight loss of power in these parts.

In addition to the cases of mental weakness, Monakow describes a form of more decided psychical disturbance. This may assume the shape of senile mania or melancholia or the delusions of persecution. In the latter event, the patient believes himself ill-treated by his family and friends, threatens lawsuits in order to defend his rights, etc.

Others exhibit increased excitement, with vivid hallucinations and occasionally, as in ordinary acute mania, sing aloud, gesticulate violently, and exhibit complete confusion of ideas; destructiveness may also be manifested. This condition rapidly gives place, however, to a quiet dementia.

Melancholic conditions are rarer. They are characterized by mental confusion and depressive excitement; delusions of a religious tinge are sometimes noticed, and there may be a tendency to suicide. This condition also terminates quickly in dementia.

After a longer or shorter duration of the prodromal stage, the symptoms proper of the thrombosis usually develop with great rapidity. Sometimes, indeed, the attack is as sudden as one caused by embolism or hemorrhage, and if no previous symptoms have been observed, it may be impossible to make a differential diagnosis. The symptoms of the attack itself can usually be distinguished in no respect from those of cerebral hemorrhage, but there is some difference to be noted in the subsequent history. Recovery from the paralysis which has been produced is extremely exceptional. On the contrary, the impairment of motion grows more marked with time. This feature is explained in part by another peculiarity of the affection, viz., that very commonly the mental power becomes steadily impaired after the attack of paralysis. If several seizures occur (as is not infrequently the case), the patient gradually sinks into a condition in which he forgets almost everything, and even fails to attend to the wants of nature; his speech is thick and indistinct, etc.

The symptoms due to thrombosis of the basilar artery are so peculiar that they merit separate consideration.

The symptoms develop suddenly or gradually, according to the mode of development of the thrombus.

Sometimes merely a severe apoplectic attack is produced, accompanied often by contraction of the pupils and irregular breathing, the attack proving fatal in a period varying from a few hours to several days.

But usually very characteristic phenomena are developed. If hemiplegia is present (and this is usually the case), it is generally of the so-called "alternate" type (Gubler). In this form of hemiplegia the body is paralyzed on one side and the face on the opposite side (the paralysis of the facial nerve involves the occipito-frontalis and orbicularis palpebrarum, and is therefore similar to peripheral facial paralysis). In addition, there is another very rare form of alternate hemiplegia, in which the face and body are paralyzed on the same side, the motor oculi communis on the opposite side. Leyden proposes to call the former variety inferior alternate hemiplegia, the latter variety superior alternate hemiplegia.

In addition, disturbances of speech and deglutition are frequently produced, the former more commonly than the latter. The difficulty of speech is shown by the thick, muffled articulation, and is due to paresis of the muscles of articulation; in other words, it does not present the characteristics of any of the forms of aphasia. The tongue is usually moved with difficulty. The bodily temperature is apt to rise very high in these cases shortly before death, and Eichhorst has reported a case in which the temperature reached 108° F.

Despite the gravity of the lesion, some patients make a tolerably complete recovery, and again become able to walk about, until a further extension of the disease produces a second and fatal attack.

Diagnosis.—As we have on several occasions remarked, the symptoms of an apoplectic seizure due to cerebral hemorrhage, embolism, and thrombosis may be identical. But the attendant circumstances are often of such a nature that we shall be enabled to distinguish cerebral thrombosis from the two other affections mentioned.

When the prodromal stage is very long and the attack itself develops slowly, the recognition of thrombosis is usually quite easy. But when these conditions do not hold, the diagnosis depends chiefly upon the age of the patient, the condition of the blood-vessels (particularly the radial and temporal arteries), and the subsequent history of the case.

Furthermore, it is characteristic of this disease that it is very often attended by a gradual, pronounced failure of the mental powers, so that the patients finally sink into a demented condition.

In a considerable proportion of cases, however, it is impossible to make a positive diagnosis between cerebral hemorrhage and thrombosis.

Prognosis.—The prognosis, as regards complete recovery, is extremely unfavorable, not so much on account of the severity of the lesion itself, but from the fact that the vital powers are usually at such a low ebb that restoration of function in the affected parts is impossible.

Sometimes a considerable interval elapses between the individual attacks, but the disease is always progressive, and unless carried off by an intercurrent disease, the patient gradually sinks into a condition of imbecility, finally gets up bed-sores, etc.

Treatment.—During attacks of cerebral embolism or thrombosis very little can be done beyond keeping the patient perfectly quiet. When indicated by the pulse, stimulants should be administered to rouse the failing action of the heart.

After the primary attack has passed over, the treatment of the residua of embolism will be essentially the same as that advised concerning cerebral hemorrhage.

After thrombosis, the paralysis and other symptoms do not yield to treatment, and little can be done beyond keeping up the general nutrition of the patient.

Leopold Putzel.

BRAIN: FUNCTIONS OF CEREBRAL CORTEX.—It is now conceded that the physiological processes underlying sensation and perception, emotional expression, language, voluntary movement, memory, the association of ideas and intellection are to be sought within the encephalon. It is admitted, also, that the structures which are thus prerequisite to every manifestation of mental life are the neurones of the cortex of the cerebral hemispheres. Exner's statement that a "physiology of the cerebral cortex in the sense in which there is a physiology of the muscle, etc., scarcely exists at the present time" is doubtless as true now as it was two decades ago. And yet the functions of the cerebral cortex, both of its entirety and also of differentiated portions, are at the present day in large measure ascertained and comprehended.

A bare recital of the facts of cerebral localization conduces only to confusion. It would be difficult to point to another department of science in which facts as facts have so little significance. Before a fact or reputed fact can be comprehended and made to serve as the basis for conclusions concerning the nature and localization of the cerebral functions, it must not only be critically examined on its own merits; it must also be connected with a great multitude of other complicated phenomena and interpreted in relation to general theories of mental and neural activity. The comprehension of the functions of the cerebral cortex is mainly the result of psychological interpretation and analysis.

Six different methods of investigation or lines of in-