of the cerebellum. The old belief, however, that the cerebellum is in any physiological or anatomical connection with the visual centres, has long since been discarded. Similarly, anosmia, the loss of sense of smell, is not a focal symptom, but is dependent upon the pressure of hydrocephalic fluid, a frequent consequence of cerebellar disease, upon the olfactory bulbs.

The other cranial nerves may occasionally suffer indirectly.

About the state of the tonus of the muscles, the reports are almost diametrically opposite. According to some there is a normal tonus or even a hypertonia; according to others a unilateral or bilateral hypotomia; and according to still others there is at one time some rigidity, at another flaccidity.

In keeping with this, the reports about the reflexes, particularly the knee reflexes, are equally contradictory. They are reported to be present or exaggerated, or absent or diminished. Jackson and others report variability of the findings, and I have found it so in the patient whose picture is here reproduced, and whose tonus and reflexes were frequently examined.

Disordered trophic functions are reported by the experimental physiologists. But such symptoms have not been ascertained clinically. Occasionally the pes cavus is mentioned in this connection, but this is evidently the result of the static ataxia.

Visceral functions are rarely disturbed. Control of the sphincters is damaged only when the state of consciousness is interfered with. In this connection it is well to draw attention to the symptom complex that the French call syndrôme bulbo-protuberantielle — which consists of attacks of bradycardia and Cheyne-Stokes respiration.

These symptoms are evidently referable to pressure on the medulla, and will occur in any lesion in this neighborhood. The same may be said of the occasionally observed glycosuria and polyuria.

The mental functions are primarily not altered in cerebellar disease, and the old conception of the intimate connection of the cerebellum with the sexual instinct is without foundation.

The speech is frequently altered. The disturbance is of the dysarthric type, commonly called cerebellar dysarthria, and occasionally reminds one strongly of the syllabic speech.

Diagnosis.—The diagnosis of disease of the cerebellum is made without difficulty when the focal symptoms are pronounced. Frequently, however, the picture is considerably obscured by a number of distant symptoms or by compensation. *Joseph Fraenkel.*

BRAIN: CEREBRAL HEMORRHAGE.—Etiology.—The most frequent cause of non-traumatic cerebral hemorrhage is the occurrence of miliary aneurisms in the vessels of the brain. These aneurisms are 0.1–1 mm. in size and are found with extreme rarity in other organs. Charcot and Bouchard, who were the first to recognize them as the cause of hemorrhage, regard them as due to chronic periarteritis, which leads to an excessive proliferation of nuclei in the lymphatic sheaths and walls of the vessels, and sometimes to atrophy and disappearance of the muscular coat. These writers believe that the implication of the tunica intima, which is always present in such cases, is secondary to the affection of the outer walls. Almost all the more recent writers attach the chief importance to a primary affection of the tunica media. According to Roth, the intima is first attacked by waxy degeneration. Roth states that, in the beginning, we find diffuse cylindrical dilatation of the vessel, then there is degeneration of the muscular coat, occasionally of a waxy character. Next, all three layers of the vessel undergo aneurismal dilatation, and finally the intima and adventitia become thickened in order to compensate for the atrophy of the media.

Löwenfeld claims that various vascular changes may lead to rupture, viz., simple atrophy, fatty and granular degeneration or ordinary atheroma. There is very little doubt, however, that these views obtain, if at all, only in exceptional cases.

Miliary aneurisms are always very minute, and sometimes cannot be seen without the aid of a lens. Charcot and Bouchard noticed that they manifested a predilection for certain parts of the brain, being found with diminishing frequency in the following regions: optic thalamus, corpus striatum, cerebral cortex, pons Varolii, cerebellum, centrum ovale, peduncles, and medulla oblongata. In the very large majority of cases they are situated in the corpus striatum and optic thalamus, thus explaining the great preponderance of hemorrhages in this locality.

It has long been thought (and many clinicians and pathologists hold this view at the present time) that atheroma of the cerebral vessels is one of the main causes of cerebral hemorrhage. But this view is erroneous. Atheroma of the brain is confined usually to the arteries of the circle of Willis, and it is not uncommon to find these vessels as rigid and unyielding as pipe stems, without a trace of hemorrhage in the brain. In rare cases, even the vessels of the pia mater have been found to be exquisitely atheromatous, although no vascular rupture had occurred. On the other hand, it is not uncommon to find extensive hemorrhage, although atheroma of the vessels is entirely absent or very slight.

Age is an important factor in causation, the large majority of cases occurring after the age of forty-five years. But cases are not very infrequent at an earlier period, and they may also occur in infancy and childhood. Billard found a clot in the left corpus striatum in an infant three days old.

Heredity also appears sometimes to play a certain part in etiology, but it acts by inducing the development of miliary aneurisms, and not by giving rise to any defect in the cerebral tissues proper.

The disease also occurs quite frequently, during the course of Bright's disease, associated with hypertrophy of the heart without valvular lesion. It has been supposed that the cardiac hypertrophy and consequent increased arterial pressure are the active factors in this condition, but it is more probable that they act only as exciting causes, the real cause being found in the changes in the walls of the vessels (arteriosclerosis), which constitute an integral part of Bright's disease.

In scurvy, leukæmia, hæmophilia, and severe infectious diseases, minute cerebral hemorrhages may occur as the result of the nutritive changes induced in the walls of the vessels, but these will not be considered in this article.

Violent muscular strain, mental excitement, cold baths, etc., are also adduced as causes, but they prove efficient only if a lesion of the vessels is present. They probably act by causing active or passive cerebral congestion.

The large majority of cases occur in the male sex, the predisposing and exciting causes being more frequent in males than in females.

Pathological Anatomy.—If the patient dies soon after the occurrence of a cerebral hemorrhage, a soft, black clot is found, which can be readily removed from its cavity, and is usually mixed with shreds of brain tissue. The surrounding tissues are irregularly torn, and are softened and blood-stained for some distance around the clot. Very little fluid blood is present. The size of the clot varies from a minute speck (capillary hemorrhages) to a mass of enormous size, which may occupy a considerable portion of one lobe. In the latter event the blood usually ruptures into the lateral ventricle, may then pass into the opposite lateral ventricle, and also force its way through the third into the fourth ventricle. More rarely the hemorrhage breaks through the cortex and appears under the pia mater, usually upon the convexity. When the hemorrhage is large in amount, pressure effects are distinctly perceptible. The convolutions may be flattened, the falx cerebri pushed forcibly toward the opposite side of the brain, and even the nerves flattened at the base of the brain. In such cases the cerebral tissues are usually very dry.

If the loose clot is gently removed and the tissues

slowly moved to and fro under water in order to remove the more adherent shreds, we shall usually be able to find the artery upon which the ruptured miliary aneurism (sometimes more than one) is situated.

After a variable period, depending chiefly upon the size of the clot, the latter begins to contract, finally breaks down, as it does in other localities, and absorption then begins. The extent to which this occurs varies in different cases. Sometimes the absorption is complete and perhaps nothing will be left at the site of the hemorrhage but a narrow, waxy, yellowish cicatrix, which contains a variable number of hæmatoidin crystals. The latter may also be scattered through the surrounding tissues, giving them a reddish-yellow color. In other cases the serum is not absorbed, and a cavity remains which may be lined with a delicate cyst wall. After the lapse of time it often becomes difficult, sometimes impossible, to distinguish these cysts from the remains of spots of softening. (Fig. 2, Plate XV.)

The changes described run their course in a short time. The clot remains soft for three or four days; absorption then becomes active and reaches its maximum toward the close of the second week. If absorption is not complete, the cavity contains serum by the twentieth day, and the cyst wall is fully formed in from four to six weeks.

Hemorrhages within the region of the pyramidal tracts, in any part of their course (and also those in the motor regions of the cortex), give rise to secondary descending degeneration, which extends down through the crus, pons, and medulla into the antero-lateral column of the opposite side of the spinal cord. In very rare cases the degeneration finally extends into the anterior gray horns of the cord.

Among twenty cases of descending degeneration of the spinal cord due to unilateral lesions of the brain, Pitres found four cases in which the degeneration was present in both lateral columns, though more markedly on the side opposite to the cerebral lesion. He attributes this phenomenon to very incomplete decussation of the pyramidal tracts in the medulla oblongata—the variability of which has been clearly demonstrated by Flechsig.

After hemorrhages into the occipital lobe secondary degeneration is found in the optic radiations and, at a much later period, in the external geniculate body, pulvinar, and anterior corpora quadrigemina. After the lapse of years this degeneration may involve the optic tract and nerve.

After hemorrhage into the second or the third frontal convolution we find degeneration of the dorsal bundle of the lenticulo-striate section of the internal capsule. This is followed by degeneration of the median segment of the cerebral peduncle, of the fibres to the median nucleus of the optic thalamus, and of the anterior portion of the lateral nucleus of the same ganglion.

After hemorrhage into the temporal convolutions degeneration of the most lateral portion of the peduncle has been observed in several cases.

After large hemorrhages into the tegmentum of the pons, secondary degeneration of the fillet takes place, not alone in an ascending but also in a descending direction. This extends to the nuclei of the posterior columns on the opposite side after the lapse of years, and the ganglion cells there undergo atrophy.

Almost all cases of old hemorrhage which have come under our observation on the post-mortem table have exhibited, irrespective of the site of the lesion, an atrophy of the brain. This is observed throughout the entire cerebral hemisphere on the side of the hemorrhage, and in the opposite hemisphere of the cerebellum. The atrophy of the latter is proportionately less marked than that of the cerebrum, but it appears to affect all parts alike. The cause of this phenomenon is obscure, but it is due probably to partial disuse of the structures implicated.

Symptomatology.—In the majority of cases there are no precursory manifestations of this disease. When they are present, they consist of vertigo, headache, ringing in the ears, general mental inertia, occasional lapses of memory, and defective speech. Sometimes slight numbness and a feeling of weakness may be felt upon one side of the body. The latter symptoms are generally dependent on a slight hemorrhage which has already occurred. The other prodromal symptoms are supposed to be due to congestion of the brain.

As a rule, however, the symptoms begin quite suddenly. In a considerable proportion of cases the attack develops during sleep, the patient going to bed in his usual condition of health and awaking in the morning to find himself paralyzed on one side of the body. When the attack occurs during waking hours, the patient generally experiences a strange sensation in the head and an increasing feeling of numbness and weakness on one side. This is followed rapidly by loss of power on that side, and the patient, if standing, falls to the ground, and in a certain proportion of cases becomes unconscious. It must be remembered, however, that a large number of patients retain their consciousness throughout the entire seizure. Thus it has been noticed that even twenty-four hours have elapsed from the onset of the symptoms to the development of complete hemiplegia and aphasia, the patient retaining consciousness and the control of his mental faculties during this entire period. The disturbance of consciousness in different attacks presents all possible grades from this condition to complete and profound coma.

In the most severe cases the patient lies motionless, with a turgid, sometimes livid face, the pupils varying but usually presenting no characteristic appearance, the cheeks flaccid and flapping loosely with respiration. The pulse is usually slow, full, and hard; the arteries of the neck pulsate visibly. Respiration is usually slow, labored, and attended with stertor; sometimes Cheyne-Stokes respiration is observed and is generally of fatal import. All the limbs may be in a condition of complete resolution, but more commonly the non-paralyzed side manifests a slight rigidity.

A not infrequent symptom of the apoplectic seizure is the so called conjugate deviation of the eyes and head. Both eyes are usually turned away from the paralyzed side, "as if looking toward the site of the lesion." Nystagmus movements may be noticed at times, but the axes of the eyes are not directed beyond the median line. The head is also turned in the same direction as the eyes, and offers distinct resistance when an attempt is made to restore it to the normal position. In extremely rare cases this symptom has been observed in attacks of cerebral hemorrhage unattended with loss of consciousness.

This peculiar phenomenon is usually noticed when the hemorrhage occurs in the parietal lobe (especially near the inferior parietal lobule), although it is also observed at times when other parts of the brain are implicated. According to Hughlings Jackson, conjugate deviation is of sinister prognostic import with regard to the recovery of the paralysis.

When the hemorrhage ruptures into the ventricles, or through the cortex beneath the pia mater, contractures or convulsions are frequently noticed. Pitres states that this symptom depends upon the point of rupture of the hemorrhage into the ventricle, and is produced only when the fronto-parietal fibres are involved by the lesion.

It has been stated that severe hemorrhages are attended invariably by an initial depression of temperature (one-fourth to one-half hour after the beginning of the seizure), followed in a few hours by a rapid rise, which continues until death in fatal cases, and subsides after a few days in the non-fatal cases. In some instances the temperature may rise from the very beginning, even though the disease has a rapidly fatal termination.

The cutaneous and tendon reflexes are usually abolished or greatly diminished during the period of coma. But this condition soon gives place to an increase of reflex excitability, with the exception of the cremaster and abdominal reflexes, which are found to be diminished.

As a general rule, the patient slowly rallies from the comatose condition, consciousness being restored usually in a period varying from an hour or more to a couple of

EXPLANATION OF PLATE XV.

EXPLANATION OF PLATE XV.

FIG. 1.—"The inner surface of the dura mater (right half) is covered with a pseudo-membrane which is colored a pale red in some parts, a dark red in others, and which clings firmly to the dura. On the left side there is presented an irregularly shaped yellowish-green deposit which rests upon an older layer of organized exuded material of a dark red color and infiltrated with blood. The explanation of these conditions is this: An infectious fibrino-purulent inflammation has, in this case, been grafted upon an older proliferative process, a complication which is observed only in rare instances. The pseudo-membrane, which is applied quite uniformly over the inner surface of the dura mater, is composed of a fibrous connective tissue which in some parts is rich in cells and everywhere is liberally supplied with blood-vessels (pachymeningitis vasculosa). In many places throughout the layers of newly formed tissue there may be seen hemorrhagic exudations, some of older and others of more recent date. It is safe to assume that these hemorrhages have come from the imperfectly developed new blood-vessels, through a process of diapedesis." (Bollinger.)

FIG. 2.—*Apoplexy of the Right Hemisphere of the Brain (nucleus caudatus and nucleus lentiformis; capsula interna).* "In the right cerebral hemisphere, at a point corresponding to the location of the nucleus lentiformis, and extending from this outward as far as to the capsula interna, is an irregularly shaped cavity, somewhat larger than an English walnut, which is filled with dark, reddish-brown masses of clotted blood. Its walls in some places show a rusty red discoloration. Some bloody serum is present in the right lateral ventricle. The rest of the cerebral parenchyma is tough, the brain as a whole being atrophic (it weighs 1,220 gm.). The large arteries at the base of the brain are in a condition of marked atheromatous degeneration; the arteria pro fossa Sylvii is obliterated.

"Among the other pathological conditions found in this woman, who was sixty-nine years of age, the following may be mentioned: Chronic interstitial nephritis and hypertrophy of the heart (both ventricles), which weighed 475 gm. (the normal weight being from 250 to 300 gm.)." (Bollinger.)

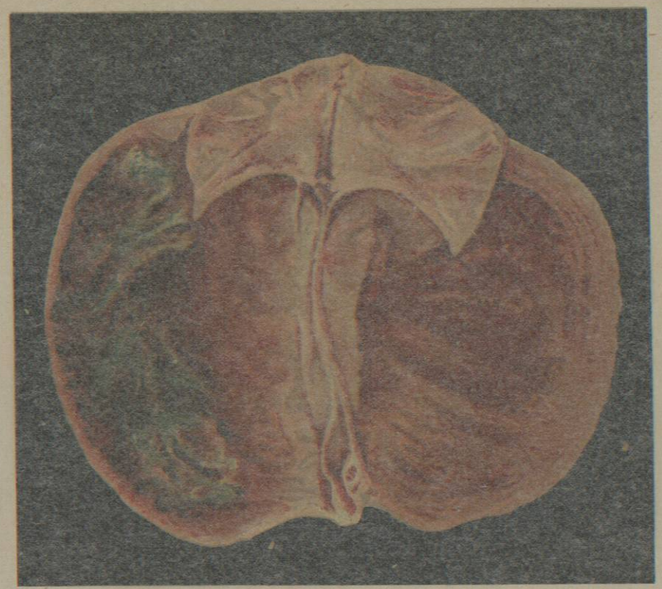

FIG. 1.

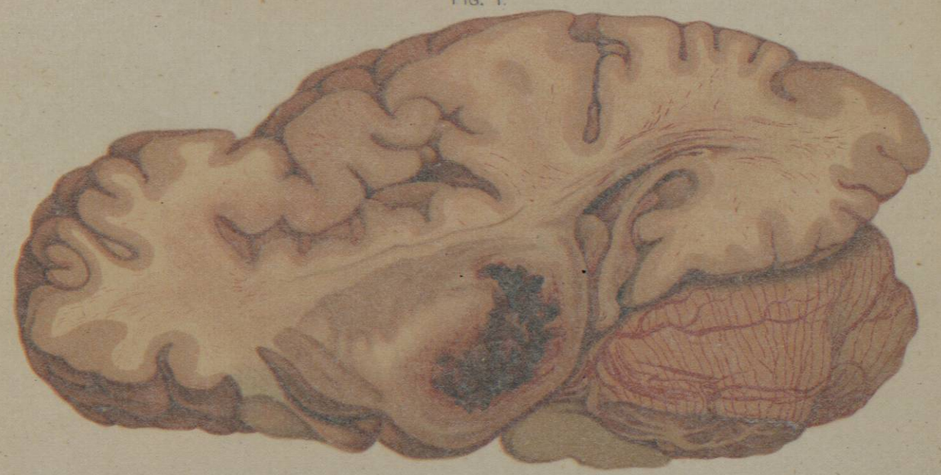

FIG. 2.

Intracranial Pathological Conditions. (After Bollinger)

Fig. 1. Pachymeningitis Interna Hæmorrhagica. Fig. 2. Apoplexy of the Right Hemisphere of the Brain.