Pseudo-cephalocele sometimes results from syphilitic or tuberculous caries of the skull and perforation of the dura mater, and from complicated fractures accompanied with prolapsus cerebri. Such cases have been reported by Bennett, Tavignot, Bryk, Podrazki, Spring, Hawkins, Talko, Bruns, and Kusmin. In one case only did the dura mater protrude.

These tumors vary from two to five inches in diameter, and are usually of a soft, elastic consistence. The skin over them is either smooth, glossy, and pale red, or inflamed, indurated, and presenting fistulous openings. Sometimes there are pulsation and fluctuation. Dizziness, retardation of the pulse, and even unconsciousness can, as a rule, be produced by pressure upon these tumors with the finger. Partial replacement may be brought about by compression, and in one case there was complete return of the hernia by gradual pressure. When the brain has protruded, this was doubtless due in many cases to the formation of cerebral abscess or to collections of pus in the cavity of the skull; in some cases, probably to œdema or encephalitis with tumefaction of the brain in the neighborhood of the diseased or injured skull.

Treatment.—In pseudo-meningocele continuous and equable pressure should be employed, when practicable, but this should be done early. If the tumor is large and tends to increase in size, operative interference is allowable, and this is best carried out according to the methods described by von Bergmann below. Strict antiseptic precautions should be observed, and a compress subsequently applied. Irritative injections are not advisable. If the opening in the skull remains patent, it is well to keep it shielded by a plate and bandage for the prevention of a renewal of the tumor or injury to the brain. Tonics, nutritious diet, and lime preparations are indicated.

Other forms of pseudo-cephalocele are treated according to the nature of the bone disease or injury. In suppurative processes we must be on the lookout for pus deposits in the brain or skull cavity, and remove them, if feasible, with careful asepsis. It is best not to operate on the prolapsed brain itself, but in this we must be governed by what we have recently learned with regard to the physiological value of the different convolutions. Hitherto such heroic measures have been carried out usually as a result of error in diagnosis, and in these cases death has followed from meningitis, encephalitis, or hemorrhage. Compression should naturally be resorted to in these cases only when there are no symptoms of inflammation or suppuration. In the rare pseudo-cephaloceles of adults we may cautiously puncture and make use of the compress, but we should always be on the lookout for symptoms of inflammation and abscess.

Bayenthal (*loc. cit.*) advances the singular theory that the presence of a pseudo-meningocele may be of *advantage* to the patient as a safety valve for increased intracranial pressure, and that it might be more advantageous to *enlarge* it, than to remove it, in cases showing cortical irritation.

Von Bergmann (on "Cephalhydrocele Traumatica," *St. Petersburg. med. Woch.*, 1897, N. F., xiv., 61) says that in these cases he always presupposes a fracture of the skull—in the majority of the cases of the lateral aspects of the skull—the lined fractures being vertical. The interval often observed between the bones in such cases is doubtless due to subsequent absorption. A laceration of the dura has always been found at autopsy. He considers compression of service only in a few cases, and only when applied early. Puncture is successful in a few; but meningitis is to be feared. In 33 cases of Rahm, where puncture was employed, 6 were cured and 9 died. Of 3 cases treated by puncture and iodine, 2 died and 1 recovered with prolapsus cerebri. Two cases treated by incision and drainage died. He cites Krönlein's two cases, in which the sac was removed and drainage employed. Both recovered. One had to wear a plate, and in the other epileptic convulsions returned seven to eight months after operation. Von Bergmann concludes as follows:

1. Puncture should not be practised. Injections with iodine are still more inadvisable.
2. Compression should first be tried (with compress and bandage or with a plate). If the tumor is thereby kept small and flat, radical operation can be delayed.
3. If the tumor increases in size, the method of Slajmer and Dembrowski is simple and less dangerous than the method of Krönlein. This consists of making a flap of skin, periosteum and bone to cover the defect in the skull, first emptying the sac and dissecting it away from the edge of the bone.

Frederick Peterson.

BRAIN: CEREBELLAR DISEASE.—An etiological factor with specific predilection for the cerebellar tissue does not exist. Whatever causes disease of other organs and tissues of the body, produces occasionally also cerebellar lesions. Chronic purulent otitis media is of particular etiological significance; cerebellar abscess, a common affection of the cerebellum, is one of its frequent complications.

Prenatal intrauterine and intrapartum accidents and mishaps may occasionally lead to disease of the cerebellum, although this occurs more rarely here than in the other parts of the nervous system.

Ataxie héréditaire cerebelleuse (Marie, Nonné) is believed to be a hereditary disease of the cerebellum; the final proof that the cerebellum only and primarily is the cause and seat of this disorder is, however, still wanting.

Cases of congenital atrophies and scleroses of the cerebellum are on record. Their etiological explanation is still very obscure. For some of these cases, possible intrauterine inflammations or traumatisms or intrapartum accidents may furnish a probable etiology, analogous to similar results wrought by the same factors on the brain, and occasionally on the cord.

Specific acute infectious diseases like poliomyelitis and poliencephalitis do not occur in the cerebellum.

If any damage is done to the cerebellum in the course of acute infectious diseases, it has not been recorded.

The chronic infectious diseases—syphilis and tuberculosis—are quite frequent causes of cerebellar disease. Neither appears to injure the cerebellum directly, and the metasyphilitic toxins, of so great an etiological importance for the brain and cord, show no affinity for the cerebellar tissue.

The cerebellar degenerations found, in cases of tabes, are manifestly secondary to the primary disease.

Solitary tubercles are frequently found in the cerebellum, and localized basal tuberculous meningitis may occasionally damage the cerebellum.

Formation of gummatous tumors is apparently rarer than that of the tuberculous variety, and the syphilitic meningitis, in the vicinity of the cerebellum, is more frequent than the tuberculous.

Finally, it is to be remembered that syphilis, by injuring the blood-vessels, is occasionally at the bottom of cerebellar mischief. It is true, this is vastly less often the case here than in the brain or cord, partly on account of causes explained further on.

Exogenous intoxications do not seem to harm the cerebellum, although the picture of acute alcoholism is believed to be caused by cerebellar disturbance.

General pathological conditions (anæmias, diabetes, carcinosis, etc.) do not directly influence the cerebellum.

Arterio-capillary fibrosis is the only general state of etiological significance.

Hemorrhages, embolisms, and thromboses of cerebellar blood-vessels are not very frequent.

Embolism of cerebellar arteries is apparently a great rarity. This is in part due to the anatomical peculiarities of the cerebellar vascular supply. All three cerebellar arteries (arteria cerebelli inferior posterior, arteria cerebelli inferior anterior, and arteria cerebelli superior) leave their principal branches at a right angle, and therefore an embolus will be carried away by the circulation through the wider basilar artery, and will be finally lodged in the arteria cerebri posterior.

Thromboses of cerebellar arteries are quite rare; occasionally thromboses of the vertebral arteries may lead to patches of softening in the cerebellar hemispheres.

Hemorrhages of cerebellar blood-vessels are also not very frequent.

The cerebellar arteries are very much smaller than the cerebral, and the only larger artery, the arteria corporis dentati, is accordingly the one that is most frequently ruptured.

Some cases of acquired cerebellar atrophies and scleroses are perhaps interpretable in a similar way on an arteriosclerotic basis, as are analogous conditions of the cord or brain.

Inflammations of the cerebellar tissue are occasional findings, but apparently most of the time they are secondary purulent infections.

The autochthonous and metastatic tumors of the cerebellum have here the same causes as in the other parts of the body.

Disease of the bones, meninges, and other contiguous tissues, and various external injuries, lead to a variety of secondary pathological states of the cerebellum.

Symptoms.—About twenty years ago, Nothnagel wrote in the classical monograph embodying the clinical thought of that time that there is more diversity of opinion and controversy concerning the symptomatology of diseases of the cerebellum than of any other part of the brain.

At present there are still some who have this opinion, and occasionally reports are heard of cases of disease of the cerebellum "without symptoms"; in other words, the cerebellum is still to be classed under the "silent areas." Increased clinical minuteness diminishes considerably, however, the number of cases of disease of the cerebellum "without symptoms."

At an earlier date, experimental methods were employed to clear up this question, and indeed greater unanimity was reached. The results obtained by experimental physiologists (Flourens, Magendie, Weir Mitchell, Luciani, Ferrier, and Turner), by their efforts to ascertain the symptoms of irritation and paralysis of this part of the nervous system, furnish a safe basis for the construction of the symptomatological picture. However, a great many points of the symptomatology are still under discussion, because focal and distant symptoms are not kept sufficiently asunder as yet.

The symptoms of cerebellar disease that may be termed focal evidently do not depend upon the nature of the causative lesions. Tumors, abscesses, softenings, degenerations, etc., will necessarily give symptoms more or less identical of irritation or paralysis of function.

Differences of degree only and not of kind are frequently observed. These are dependent upon the character of the lesion, whether slow or rapid, destructive or stationary.

Territorial variations of the lesions do not seem to exert any considerable influence.

The point particularly dwelt upon by Nothnagel, that only a lesion of the vermis leads to symptoms, while lesions of the hemispheres have no clinical manifestations, has not been corroborated as fully as it was originally claimed.

All of the foregoing will be better understood when a few cardinal points of the physiology and pathology of the cerebellum, about which all clinicians and experimental physiologists seem to agree, are borne in mind. These points are, first, the fact of the anatomical and physiological homogeneity of the cerebellum.

Microscopically, the structure of the cerebellar hemispheres is found to be the same all over, and on that basis, as well as on clinical and experimental evidence, physiological identity is conjectured. Thus it is evident that one part can easily take up the function of the other.

It is further established by clinical and experimental evidence that the functions of the cerebellum are taken up by other parts of the nervous system, particularly by the somæsthetic cortical areas (Luciani).

The above-mentioned dissensions are easily understood, when one considers that the experimental physiologists observe the animal throughout the whole time of the disease, see it, as it were, during the flourishing period of the symptoms, and in the period when, through establishment of direct or indirect compensation, a good part of the symptoms are disguised or obscured; and that, on the other hand, clinical observations most of the time extend over a limited period only, and by some observers cases are seen with fully established compensation, and by others cases more or less compensated or not at all.

After these remarks it is safe to state that the symptoms of disease of the cerebellum, no matter what lesion gives rise to them, are symptoms referable to disorder of the equilibration, regulation and adjustment of the voluntary movements, and are comprised in the name of cerebellar ataxia.

Luciani attempted to analyze this compound phenomenon and claims that it is composed of the following three elements: asthenia, atonia, and astasia. All these three elements give rise to a disorder of motor function that he calls motor dysmetry.

The point to which the compensation has progressed, necessarily influences the intensity of the ataxia. Not all agree, however, with Luciani, and particularly the asthenia and the atonia are denied by others.

In addition to this cardinal and direct or focal symptom of disease of the cerebellum, a number of symptoms are described in cases of cerebellar disease, the greatest part of which can manifestly not be taken as focal but must be designated as indirect or distant symptoms.

The principal focal symptom of cerebellar disease is the cerebellar ataxia. This ataxia manifests itself on the patient, first by disturbance of the general equilibrium in the form of swaying that becomes markedly increased when the eyes are closed and the feet put together. The gait designated as cerebellar gait, and according to the French (démarche d'ivresse) gait of drunkenness, is very much altered; the patient progresses in a zigzag line, the individual steps are irregular and unequal, the feet are lifted unduly high from the ground and brought down heel first with undue force.

These evidences of static and locomotor ataxia are differentiated from the same symptoms in cases of genuine tabes by the fact that patients with cerebellar disease show little or nothing of this ataxia when in bed.

This disorder of the muscular apparatus is in most cases evidenced exclusively or predominantly in the lower extremities.

A group of motor phenomena, called forced attitudes and forced movements, form the focal symptom next in importance. The head and trunk of the patient are either permanently deviated toward one side, or the patient performs queer rotatory movements around his own axis toward one side or the other—movements evidently not purposeful, and having clearly a character of forced propensity (manège movements).

The side toward which the permanent deviation or the forced movement is directed is differently reported by different observers. According to some, the head or the trunk deviates or the patient moves toward the side of the lesion; according to others, toward the opposite side; and according to still others, the passive deviation is toward the opposite side, while the active movements are toward the side of the lesion.

Another explanation of this diversity is based on the fact that lesions are either irritative or paralyzing; irritative lesions are thought to produce forced *movements* toward the same side, and paralyzing lesions forced *attitudes* toward the opposite side.

It is otherwise stated that forced movements and forced attitudes are dependent upon the situation of the lesion, and they are then interpreted as focal symptoms of the cerebellar peduncles, particularly of the middle ones.

The forced attitudes are clinically more frequently observed than the forced movements. A good illustration of a forced attitude is given in the accompanying picture of a patient in whom a tumor of the right cerebellar hemisphere was found, in which the deviation toward the same side is seen when the patient is in a re-

clining position, and more marked when she is standing (Fig. 817).

The two symptoms already named, *i.e.*, the cerebellar ataxia and the forced movements and attitudes, are the cardinal focal symptoms of cerebellar disease. But in

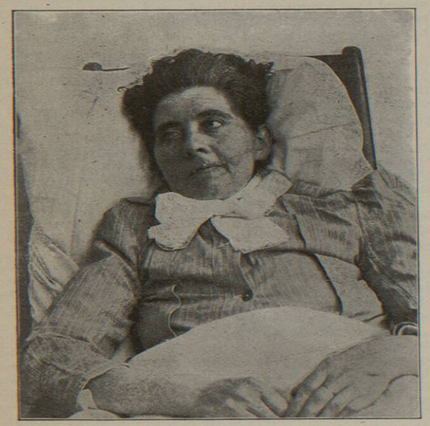


FIG. 816.—Case of Cerebellar Tumor, Showing Peripheral Facial Palsy.

almost every case there are additional symptoms recorded; some evidently not focal, others mooted as to their localizing significance, which are discussed seriatim below.

Motor Symptoms: The upper extremities do not always escape the characteristic motor disorder. Here the ataxia is not so marked, but is often replaced by atactiform tremors and unsteadiness, occasionally simulating the intentional variety. The head may show similar motor unsteadiness, although rarely.

Localized unilateral and generalized muscular spasms, with and without loss of consciousness, are frequently mentioned, and by most observers are interpreted as distant symptoms.

Motor paralysis sensu strictiori is occasionally observed. Hemiplegias, paralysis of one-half of the body, are frequently mentioned in older histories of cerebellar cases. Here, too, the side upon which the paralysis occurs is not uniformly named; at one time the paralysis was homo-, at another heterolateral to the cerebellar lesion.

In view of the fact that experimental evidence has proven the homolateral influence of the cerebellum, it is to be assumed that hemiplegic symptoms are dependent upon encroachment of the lesion upon the pyramidal tracts, and not upon lesion of the cerebellar tissue proper. This is well illustrated by the accompanying photograph (Plate XIV.), which represents a case of tumor of the cerebellum in which the observed left-sided hemiplegia, which was produced, as can be easily seen from the photograph, by pressure upon the left-sided pyramidal tract, gave rise to diagnostic difficulties.

Various disorders of the ocular musculature, nystagmus, strabismus, ptosis, paralysis of individual ocular muscles or muscle groups, are also frequently mentioned. The greatest part of these symptoms are evidently due to pressure upon the corpora quadrigemina or oculomotor nuclei; but some of them must necessarily be taken as focal symptoms.

Experimentally, deviations of one or the other eye toward the same or the opposite side of the lesion were frequently ascertained.

The facial musculature is also frequently reported to have suffered. Occasionally this disturbance is only slight and transient, and probably of a nature similar to the motor disturbance of other muscles—*i.e.*, a cerebellar motor dysmetry; but most of the time it is due to compression of the adjacent seventh nerve. It is then a paralysis of the peripheral type, involving all the three branches and showing more or less disturbance of the electrical reaction. This is well illustrated in the accompanying photograph (Fig. 816).

The motor portion of the fifth nerve is sometimes found defective, evidently through pressure upon the peripheral course of the nerve.

In the sensory sphere no symptoms directly referable to the cerebellum are observed.

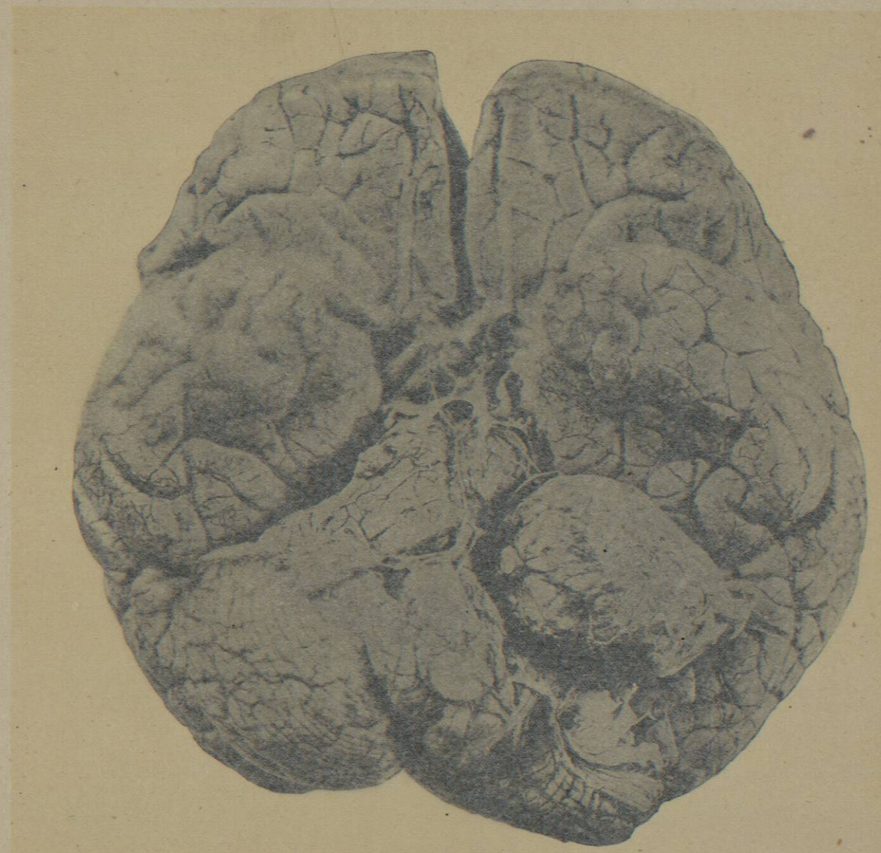
The sensibilities (tactile, algesic, thermal and kinæsthetic) are mostly unaltered. If sensory disturbances

FIG. 817.—Case of Cerebellar Tumor, Showing Hemiplegia and Deviation.

are found, as in a case of cerebellar abscess lately observed by the writer, they are easily traced to the spreading of the disease into the sensory pathways.

Of the special senses sight and smell are frequently found impaired. Choked disc and optic atrophy are frequent and early accompaniments of compressing lesions

PLATE XIV



TUMOR OF THE CEREBELLUM

clining position, and more marked when she is standing (Fig. 817).

The two symptoms already named, *i.e.*, the cerebellar ataxia and the forced movements and attitudes, are the cardinal focal symptoms of cerebellar disease. But in

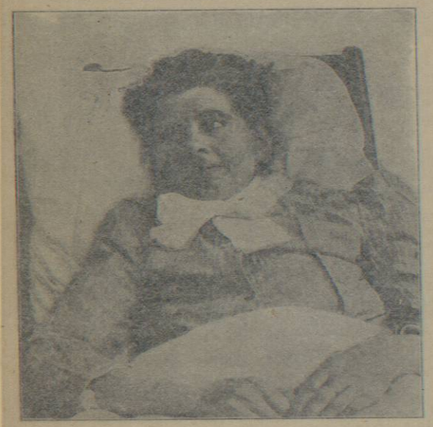


Fig. 816.—Case of Cerebellar Tumor, Showing Peripheral Facial Palsy.

almost every case there are additional symptoms recorded; some evidently not focal, others mooted as to their localizing significance, which are discussed seriatim below.

Motor Symptoms: The upper extremities do not always escape the characteristic motor disorder. Here the ataxia is not so marked, but is often replaced by atactiform tremors and unsteadiness, occasionally simulating the intentional variety. The head may show similar motor unsteadiness, although rarely.

Localized unilateral and generalized muscular spasms, with and without loss of consciousness, are frequently mentioned, and by most observers are interpreted as distant symptoms.

Motor paralysis sensu strictiori is occasionally observed. Hemiplegias, paralysis of one-half of the body, are frequently mentioned in older histories of cerebellar cases. Here, too, the side upon which the paralysis occurs is not uniformly named; at one time the paralysis was homo-, at another heterolateral to the cerebellar lesion.

In view of the fact that experimental evidence has proven the homolateral influence of the cerebellum, it is to be assumed that hemiplegic symptoms are dependent upon encroachment of the lesion upon the pyramidal tracts, and not upon lesion of the cerebellar tissue proper. This is well illustrated by the accompanying photograph (Plate XIV.), which represents a case of tumor of the cerebellum in which the observed left-sided hemiplegia, which was produced, as can be easily seen from the photograph, by pressure upon the left-sided pyramidal tract, gave rise to diagnostic difficulties.

Various disorders of the ocular musculature, nystagmus, strabismus, ptosis, paralysis of individual ocular muscles or muscle groups, are also frequently mentioned. The greatest part of these symptoms are evidently due to pressure upon the corpora quadrigemina or oculomotor nuclei; but some of them must necessarily be taken as focal symptoms.

Experimentally, deviations of one or the other eye toward the same or the opposite side of the lesion were frequently ascertained.

The facial musculature is also frequently reported to have suffered. Occasionally this disturbance is only slight and transient, and probably of a nature similar to the motor disturbance of other muscles—*i.e.*, a cerebellar motor dysmetry; but most of the time it is due to compression of the adjacent seventh nerve. It is then a paralysis of the peripheral type, involving all the three branches and showing more or less disturbance of the electrical reaction. This is well illustrated in the accompanying photograph (Fig. 816).

The motor portion of the fifth nerve is sometimes found defective, evidently through pressure upon the peripheral course of the nerve.

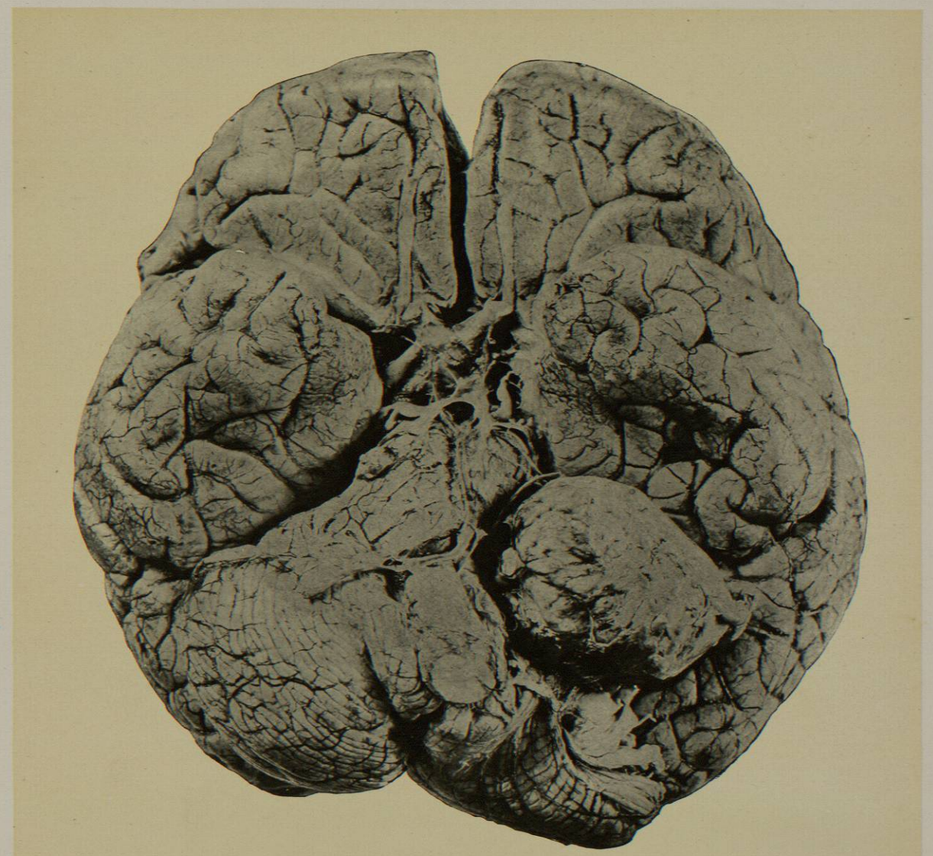
In the sensory sphere no symptoms directly referable to the cerebellum are observed.

The sensibilities (tactile, algesic, thermal and kinæsthetic) are mostly unaltered. If sensory disturbances

Fig. 817.—Case of Cerebellar Tumor, Showing Hemiplegia and Deviation.

are found, as in a case of cerebellar abscess lately observed by the writer, they are easily traced to the spreading of the disease into the sensory pathways.

Of the special senses sight and smell are frequently found impaired. Choked disc and optic atrophy are frequent and early accompaniments of compressing lesions



TUMOR OF THE CEREBELLUM

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