ure of the intelligence is usually proportioned to the extent of the lesion; qualitative alterations of special faculties bear a more or less definite relation to the seat of the lesion. The dissociations of the speech faculty are numerous, giving rise to aphasia, amnesia, agraphia, alalia, and their combinations. Failure in memory, as in all chronic cerebral disease, is one of the earliest mental defects; the moral sense, dependent on the ethical mechanisms, the most delicate of all (Griesinger), is blurred.

Localized atrophies have been of great service in investigating the problems of localization in cerebral functions.

Prognosis of cerebral atrophy depends upon the extent of lesion, and upon its stationary or progressive character. When only a limited amount of brain tissue is destroyed, its function may be vicariously performed by another portion, and the loss thus repaired. This is impossible with extensive acquired lesions. In the famous cases of congenital atrophy of the cerebellum or corpus callosum, however, the functions of these parts must have been performed by other organs, for their defect could not be diagnosed during life.

A broad distinction exists between the prognosis of congenital and that of acquired atrophy, in that the former far more frequently coincides with an arrested morbid process, the latter with one that is either continuously progressive or liable to renewal after temporary arrest. The mental defects due to atrophy of any portion of the adult brain cannot be repaired; the utmost to be expected is their limitation; the probabilities are that they will steadily or intermittently increase. On the contrary, in a brain partially atrophied while still in process of development, a vigorous psychic education may often hope to develop faculties by exercise of the intact portions. The difficulty of doing so increases with any form of disturbance of the speech faculty. Congenital paresis tends spontaneously to diminish, and muscular power may be greatly increased by systematized gymnastics. The paresis of acquired atrophy, unlike the paralysis due to destructive lesions, tends to permanence or to increase; the latter case being the rule in the general paresis of the insane. Muscular rigidities, contractures, and the deformities of limbs caused by them, increase for a long time in congenital cases, first as a result of the extending spinal degeneration, then as a consequence of malposition and adaptive shortening. The latter cause may be greatly palliated by appropriate apparatus; and the prognosis in respect to deformity, and to the power of walking and other use of the limbs, is hopeful in direct proportion to the influence of malposition, and inversely to that of the lateral sclerosis. Corresponding to the lesser extent of the brain lesion, the deforming contractures of acquired atrophy are much more limited, and therefore of less importance; their degree of amenability to therapeutic palliation is about the same. General acquired atrophy is not followed by deformity, for all the muscles are equally affected. The duration of life is quite indefinite. Death is never the direct consequence of the atrophy, but results from asthenia due to the progressive impairment of brain nutrition; from œdema, as the walls of the blood-vessels become more altered; from convulsions, especially associated with extension of secondary irritation; or from renewal of the primary accidents (hemorrhage, thrombosis, meningo-encephalitis, etc.).

Treatment is palliative, and in the directions implied in the remarks on prognosis. The primary morbid process, if still going on, must be treated by appropriate measures; and an important point of the diagnosis is the decision whether this primary process is or is not arrested. In congenital atrophy, the mental faculties must be awakened as far as possible by psychological education, which, to attain the end, must be both persevering and profound. The muscular paresis must be combated by gymnastic exercises, the deformities by apparatus able gradually to stretch retracted muscles, to support limbs in proper position, and by means of springs and artificial muscles to facilitate attempts at voluntary movements. The proposal to relieve the deformities due to rigidity of the adductor femoris muscles by circumcision—an operation intended to relieve a hypothetical genital irritation—is most irrational. If relief ever follow this operation, it can be only in cases of entirely different character, a purely functional spasm, possibly associated with masturbation, and having nothing to do with lateral sclerosis of the cord. The convulsions which are so common in congenital atrophy require the usual treatment. For the eye symptoms (blindness, strabismus, nystagmus) nothing can be done.

In acquired atrophy treatment mainly consists in averting conditions which are likely to revive the primary accidents, and in treating these as they arise. The earlier in life the cerebral lesion occurs the more the conditions approach those of congenital atrophy; and when the accidents date from the first years of childhood the practical treatment is identical for the two classes of cases. Conversely, as a patient approaches old age, both the primary accidents and the atrophy are more liable to be progressive, and the rôle of the therapeutist becomes more purely passive. There can be no longer question of developing a brain, checked in its evolution, but only of shielding it from new injuries which would cause fresh deterioration of faculties; hence mental strain and excitement of all kinds are to be avoided. The keynote of the treatment is the necessity for repose for the nervous processes proper and for the cerebral circulation. Regulation of the social medium is the principal factor for the first; of exercise and climate for the second. Apparatus for deformity is less needed and less tolerated than in children. The contractures principally affect the upper extremities, instead of, as in children, the lower; and it is much more difficult to facilitate the functions of the arms than of the legs by prothetic apparatus. Still, with ingenuity, this may sometimes be accomplished.

Diagnosis.—In congenital or early cases it is principally necessary to distinguish between primary and secondary cerebral atrophy, and between the latter and the various lesions upon which it depends, as hydrocephalus, hemorrhage, meningo-encephalitis. Primary atrophy may be inferred from deficient or idiotic intelligence, without motor disturbances or lesions of the special senses, while hydrocephalus may be excluded from the size of the head. The extreme degrees of diffuse primary atrophy constitute microcephalic idiocy, and are recognizable usually from the minute proportions of the cranium. The diagnosis cannot, however, always be made, for, though rarely, large portions of the encephalon may be absent, without any more defect in the intelligence than is often observed without gross cerebral lesion at all. And, on the other hand, the destructive lesion causing a secondary localized atrophy may be so limited that the descending degeneration occasions no characteristic symptoms, yet the finer mechanisms of the brain may be so jarred that the evolution of the mind is permanently impaired. In any case in which the associating fibres or the superadded convolutions (Broadbent) are affected, while the motor fibres prolonged from the crus remain intact, imbecility with preservation of muscular function is possible. In secondary atrophy, if the primary morbid processes have been arrested before they have been discovered, or at birth, it is often difficult, in the latter case impossible, to distinguish between them. Congenital hydrocephalus is recognizable when the head is already enlarged at birth, and may be a cause of dystocia. When the head begins to enlarge within a few weeks after birth, it is probable that ventricular effusion has already begun before. The probability is greater if spina bifida coexist. In such cases it is sometimes the symptoms of the effusion, sometimes those of the atrophy and spinal sclerosis, that predominate. An extra-uterine cerebral hemorrhage or attack of meningo-encephalitis is indicated by the usual signs; but these are liable to be masked in infancy by convulsions and fever, common to both, and also to so many other infantile disorders. Persistence of an inflammatory process may be indicated by

irregular recurrence of fever, associated with pain, retraction of head, pupillary symptoms, and other characteristic signs of meningeal irritation. Still these may also be aroused by the irritative process of degeneration, especially when this reaches the motor tracts in the pons and medulla. It may be said that in secondary atrophy at any age, the diagnosis may be made by the coincidence of three symptoms: mental defect, muscular paresis, and muscular contracture; these coming on gradually or after a period of stormy cerebral accidents. The various other symptoms enumerated may or may not be present; among them lesions of the cranial nerves are of the same order as the rigidities of the limb muscles, and, like them, may be referred to the descending sclerosis accompanying the atrophy. Convulsions and fever may be due to this or to persistence or revival of the primary process.

Mary Putnam-Jacobi.

BRAIN: CEPHALOCELE.—(Synonyms: Encephalocele, Hydrocephalocele, Hydrencephalocele, Cephalhydrocele, Meningocele, Hydromeningocele, Craniocele, Hernia seu Fungus seu Ectopia Cerebri). Hernia of the brain or meninges is a name given to a protrusion of a portion of the contents of the cranial cavity through an opening in the skull beneath the scalp. There is need of greater simplicity in what is now a rather confusing terminology, and it would seem that the best designation for all of these tumors as a class is cephalocele. There is a correspondence in application between this word and enterocele because of the similarity of their constituents. There is a hernial canal or opening in the skull, a hernial sac consisting of the dura mater, the hernial contents composed of whatever substance or fluid may be forced out from the cranial cavity, and, finally, the various coverings of the hernia, such as the pericranium, fascia, and skin. It is necessary, in a true cephalocele, that the dura mater form the hernial sac. The tumors formed beneath the scalp in traumatic lesions of the skull and dura, by the pouring out of cerebro-spinal fluid, are not true cephaloceles, and are more properly termed *pseudo-meningoceles*. Protrusion of brain substance in compound fractures of the skull is not considered here, though sometimes improperly called a hernia cerebri; the correct designation is *prolapsus cerebri*.

The varieties of cephalocele depend upon the contents of the tumor. Hence we have a *meningocele* where the hernial contents are cerebro-spinal fluid alone pressing out the dura mater; a *hydrocephalocele*, where the tumor consists of an internal hydrocephalus expanding and forcing outward the ventricular walls; and, finally, an *encephalocele*, composed wholly of brain substance with more or less fluid usually surrounding it.

These tumors are mostly of congenital origin, but there are certain rare cases in which they are acquired after birth, through disease of the cranial bones or traumatism. It is usual, therefore, to distinguish two forms of cephalocele, the congenital and the acquired. In describing the latter it will be best to use the prefix *pseudo*.

Congenital Cephalocele.—These tumors almost always have their peduncles at or in the immediate neighborhood of some cranial suture. The great majority are in the antero-posterior median line, as a rule either in the frontal or in the occipital region. According to Giovanni Reale, who collected 68 cases of cephalocele, 10 were at the nasal root, 9 in the frontal suture, 5 at the posterior fontanelle, and 22 in the occipital bone, the rest arising from some of the lateral sutures. Larger tabulated 85 cases, 44 of which were occipital and 41 frontal or sincipital. The favorite location of the sincipital tumors is at the root of the nose, either at the inferior part of the frontal suture or at the junction of the ethmoid and frontal bones. The hernial canal or opening is generally bounded by the separated or more or less malformed ethmoid, frontal, nasal, or lachrymal bones, and sometimes even by the nasal process of the superior maxilla. Fenger (*Am. Jour. Med. Sciences*, 1895, cix., 1), in an article entitled "Basal Hernias of the Brain," describes such sincipital tumors as differ from the others in not protruding in the face. The least uncommon form is the spheno-pharyngeal cephalocele, protruding into the nasal or naso-pharyngeal cavity. These may be more common than is supposed. They may be mistaken for nasal polyps. The occipital cephaloceles are the most frequent of all. They are situated either in or near the posterior fontanelle, or lower down under the occipital protuberance, where the hernial opening may be conjoined with the foramen magnum. Hernial protrusions from other sutures are much more rare, although they do occur in the greater fontanelle, in the squamous suture, or between the ethmoid and sphenoid bones at the base of the skull.

Cephalocele is uncommon, Trélat finding but 3 in 12,000 births, and Vines 1 in 5,000. It is said to be more common in females than in males, though Z. Lawrence, quoted by Erichsen ("Surgery," vol. ii., p. 378), collected 39 cases, 21 of which were males.

The tumors vary in size from that of a pea to that of a child's head. The occipital are always the largest, and are usually hydrocephaloceles. The sincipital tumors are, as a rule, small and simple encephaloceles. Meningoceles may exist in either place, but are more common behind. Hydrocephaloceles are rarely sincipital, owing to the positions and conformations of the ventricles, which are more apt to dilate posteriorly.

The occipital *hydrocephaloceles* are usually constricted at their base, often pedunculated, almost globular in form, and seldom attached by broad bases. They generally contain the dropsical posterior horns of the lateral ventricles or their fetal analogues. Those in the lower occipital region enclose the cerebellum and the hydropic fourth ventricle. In some of the largest hydrocephaloceles have been found a great part of the cerebrum, the cerebellum, the fourth ventricle, and the quadrigeminal bodies. The protruding cephalic parts are commonly of inferior or defective development, often difficult to recognize as brain substance, owing to cystoid degeneration and sclerosis (Huebner). P. Berger (*Revue de Chir.*, 1890, x., 269) reports an extirpation of an encephalocele and a case by a colleague (Périer) in which both specimens were carefully examined by Ranvier and Suchard, showing features not hitherto described. In both were found histologically a mixture of nervous elements of both cerebrum and cerebellum without lines of demarcation, and Berger regarded these tumors as forming a variety of central neuromata such as have been described by Virchow (tumors formed in the ventricles of subjects afflicted with congenital hydrocephalus). Berger proposes the term *encephalome* for this species of encephalocele. Sometimes the constriction at the hernial opening is so great that there is marked stasis of blood in the pia, which may lead even to extravasations. Occasionally these tumors present longitudinal constrictions caused by the venous plexus or by the falces of the dura. The quantity of fluid contents varies, but may reach two quarts. It has been found to be rich in albumin, with a specific gravity of 1.010–1.012.

The *meningoceles* of the occipital region are quite as large as the hydrocephaloceles, and similar in shape, while those of the sincipital and lateral areas of the skull are much smaller. One very large meningocele held fluid which had a specific gravity of 1.004, and contained a small quantity of albumin, uric acid, chloride of sodium, and biliary coloring matters (Heineke).

The *encephaloceles* are usually small tumors with broad bases, having a diameter as a rule between 1 and 3 cm., generally occupying a position at the root of the nose, and containing often, besides cerebral substance, a small quantity of subdural fluid.

Pathology.—There are several theories advanced to account for the origin of these congenital tumors. One is, that there is a limited ventricular dropsy, which by pressure at some certain circumscribed portion of the skull expands and separates the cranial bones and thus protrudes. From a hydrocephalocele thus formed an encephalocele may be produced later, by reabsorption of the dropsical fluid; or a meningocele, by a recession of the cerebral substance into the cranial cavity. The

arguments against this hypothesis are, that there would scarcely be a circumscription of the dropsy as imagined, but rather a tendency to a general hydrocephalus; and, furthermore, Ackermann and Heineke have determined that there is a diminution, and not an increase, of intracranial pressure in cases of cephalocele.

A more probable theory as to the etiology of these tumors is, that they originate in the early embryological

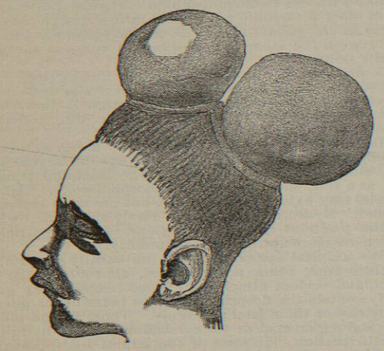

FIG. 809.—Double Cephalocele, in Which the Anterior Tumor is Covered by the Skin, the Posterior by the Dura Mater. (After Taruffi.)

periods, when the cerebral vesicle and its fluid contents are in process of growth, and their osseous capsule is not yet developed. There is then an arrested or defective evolution of the cranial vault, as a result of which complete closure of the skull does not take place, and a circumscribed portion of the fetal brain vesicle protrudes. According to this hypothesis, hydrocephalocele would belong to an earlier stage of development, and encephalocele to a later, when the protruding portion of brain no longer exhibits the configuration of the fetal brain vesicle as it does in the former. In like manner, meningocele may originate either by the mere extrusion of the dura mater through pressure of the subdural fluid, or by the bursting of a hydrocephalocele, or it may be the remnant of a disorganized encephalocele. Meningocele might also spring from the cerebral vesicle in the earliest stages of development, as a mere membranous cyst without brain substance. Yet, if the defect in the skull were the only factor in the formation of these tumors, it would be difficult to understand why they are not present in every child in the open cranial spaces, the fontaneiles. We are compelled, therefore, to recognize, as another factor in their causation, a general or partial hydrocephalus, either internal or external.

This conception of the embryological origin of cephaloceles is corroborated by the fact that most of them are found in front of and behind the head, in the median line, which is the most marked direction of growth of the cerebral vesicle; and that other defects and malformations of the skull, face, spine, feet, etc., are often observed in connection with these cases. Thus, striking departures from the normal shape of the skull are particularly noticeable in hydrocephalocele and the large meningoceles, where there is often asymmetry, thickening of the sutures, microcephalus, flattening from before backward, and sometimes hydrocephalus; while in the simpler tumors the skull presents, as a rule, a wholly normal configuration.

What may be the ultimate causes of these defects can only be surmised. Premature union of the meninges with the amnion, and injuries to the fetal head *in utero*, may sometimes be the occasion of their development. Benger (*loc. cit.*) reviews all the theories in detail (intracranial pressure from limited dropsy, arrest of fetal cranial development, fetal craniotabes) and believes himself that an encephalocele is a primitive protrusion of part of the brain previous to the formation of the cranial vault. He agrees with Fleischmann and Niemeyer that the cause of a simple encephalocele may lie in a hyperplasia of brain substance.

Lindfors occupies a whole number of the *Klin. Vorträge* (Leipsic, 1898, Nos. 222, 223) with a discussion of the history, etiology, and surgical treatment of congenital cerebral hernias, bringing the whole subject well up to date (April, 1898), and citing three full pages of literature. He believes there are many different forms and a variety of causes, that even within one class there may be special individual modifying causes. The etiology of hydrencephalocele is probably the same as that of hydrocephalus, but acting in a limited area. In other varieties, hydrocephalus, new growth, hygroma of meninges (Virchow), amniotic adhesions, rachitic condition of bones, may all have a place in the etiology of special cases.

Clinical Features.—The cutaneous surfaces of the cephaloceles are usually smooth, more or less distended, and sometimes very vascular. Occasionally they are excoriated, or present old cicatrices. Angiomata and lipomata have been described as forming upon cephaloceles (Larger, Ried). Hydrocephalocele and meningocele exhibit a distinct sensation of fluctuation, while the encephaloceles have more of a sensation of softness and elasticity, but also show fluctuation if surrounded by a sufficient quantity of fluid. When the coverings are thin, from great tension by fluid contents, these tumors may be translucent. They frequently pulsate synchronously with the pulse and respiratory movements; and their tension is often variable, as there may be a sinking of the tumor in sleep, and marked bulging out in the acts of crying or coughing. They may sometimes be diminished in size by pressure, which if moderate does no harm, but if great not infrequently produces symptoms of brain compression, such as vomiting, retardation of the pulse, unconsciousness, and convulsions. Pulsation is, as a rule, absent in meningocele and hydrocephalocele, but this naturally depends directly upon the size of the hernial opening in the skull, and the intercommunicability of the contents of the sac with those of

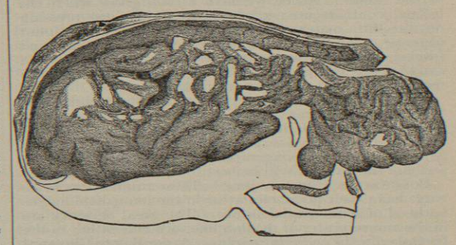

FIG. 810.—Section of Cranium in a Case of Sincipital Cephalocele. (After Vannoni.)

the cranium; these tumors also show generally little variation during sleep, coughing, or crying, or upon the application of pressure, unless the hernial foramen is large. Symptoms depend to a great extent, too, upon the physiological value of parts protruded; and in cephaloceles such parts are often very different from their analogues in the normally developed brain. In the light of the recent discoveries in cerebral localization these cases might often afford very interesting results by careful study.

Prognosis.—Many children affected with meningocele or hydrocephalocele die before or during birth. Sometimes the tumor bursts during parturition. Those born living rarely last many years, partly on account of the imperfect conditions of their brains, and partly because the tumor often rapidly increases to such size, by the superaddition of cerebro-spinal fluid, that it bursts spontaneously, causing death either speedily, in coma or convulsions, or slowly, from a consecutive meningitis. Re-

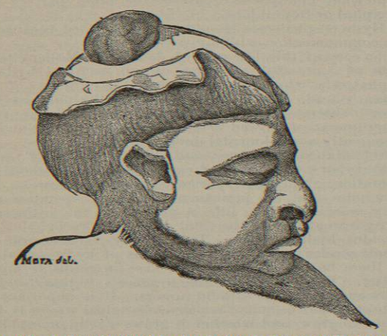

FIG. 811.—A Case Observed by Dr. Irving C. Rosse, of Washington, D. C.*

covery after such evacuation of the tumor takes place but rarely. Death occurs usually in the first two or three years of life, but a few children with meningocele have attained the age of twelve, and even seventeen years.

The encephaloceles being smaller and simpler than the varieties just mentioned, still-birth and rupture are less common in cases affected with them, although they do occur. As a rule, this class either grows slowly or remains stationary. Whatever disturbance is excited is due to disorder of brain function, but patients suffering from the frontal variety live longer and with fewer cerebral symptoms than those afflicted with the occipital variety. Children with occipital encephaloceles are generally idiots, whereas with the frontal or sincipital tumors they are more commonly of quite normal intelligence. Many have reached adult age.

Spontaneous recovery from encephalocele and meningocele has occurred, according to the observations of Wernher, Textor, von Bruns, and Held, by closure of the cranial defect and the complete isolation of the tumor as a cyst on the outside of the skull. Operative procedures have proved successful in some instances.

Diagnosis.—Every tumor of congenital origin situated along the median line of the head, in the region of the root of the nose or in the glabellar or occipital regions, should excite suspicion of a cephalocele; and defects or malformations in other parts of the skull or body would be corroborative evidence. The fact of its being congenital must first of all be determined; then its relation to the various cranial sutures. The diagnosis is easy when the tumor is in one of the situations that have been described, when there are pulsation and a sensation of fluctuation, when there is a change in tension during sleep or in crying, and when there is a recession of its contents into the skull upon pressure. If, at the same time with the recession of the brain substance or cerebro-spinal fluid, symptoms of irritation or compression of the brain develop, if there is an increase in the pulsatory or respiratory brain movements, and particularly if the edges of the hernial opening in the skull can be felt, a cephalocele is certainly present. But sometimes most of these pathognomonic symptoms are absent, and the diagnosis can be made only from the seat and history of the tumor. Although seldom necessary, a fine aseptic hypodermic needle may be introduced to determine the character of the contents and the presence or absence of an opening in the skull.

The recognition of these hernias in the occipital region is easiest, because other congenital tumors of similar form are of extreme rarity in this position; but dermoids, angiomata, and sarcomata of prenatal origin may have their seat at the nasal root, in the frontal region, or at the inner canthus of the eye, and thus lead to confusion. In the case of these latter, the mode of development must be carefully studied, and note taken of variations in tension, compressibility, and pulsation, and of the appearance of cerebral symptoms upon pressure. Angiomata exhibit pulsation and tension changes, and may be often completely compressed, but without brain symptoms or the discovery of an opening in the skull. Pulsation and compressibility are lacking in dermoid tumors. Cranial sarcomata which are congenital, or which appear shortly after birth, may present brain symptoms on pressure; there may also be an opening in the skull which pulsates; and finally these growths may have a similarity of feel, but they grow much more rapidly than cephaloceles, and usually are wanting in the more characteristic symptoms of the latter. The differentiation of the varieties of cephalocele can be made from the peculiarities that have already been described. In nasopharyngeal varieties it is well to remember their occasionally close resemblance to nasal polypi.

Treatment.—The most difficult class to treat are the hydrocephaloceles, but measures can at least be taken to prevent sudden death by spontaneous rupture of the sac. This is still more important in meningocele and cephalocele if there be a rapid increase of fluid and tension, as a cure is possible in many of these cases. Equable pressure should be made by means of cotton batting and a

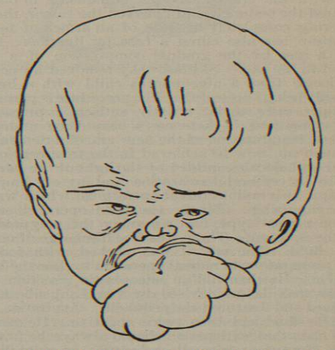

FIG. 812.—An Irregular Tumor Proceeding from the Cranial Cavity by a Large Opening Situated Immediately in Front of the Sphenoid, and Behind the Still Cartilaginous Ethmoid. Naso-pharyngeal cephalocele. (Virchow.)

bandage, care being taken not to produce brain symptoms by too strong compression. If the distention increases puncture should be made with a hypodermic needle, with antiseptic precautions, and the fluid slowly aspirated—all of it if the tumor be small, but only a portion in the case of large sacs. Then cover with aseptic cotton and continue the compression. Puncture may be repeated at appropri-

* The case shown here is that of an illegitimate male child of eight months. The tumor, about the size of a walnut, protruded from an opening in the right parietal bone just above and behind the protuberance. The child was club-footed, ectrodactylic, and had double harelip. Being unable to feed, it died after three days from inanition. The mesenteric glands were found to be enlarged.—I. C. R.

ate intervals. If rupture has already taken place, the antiseptic dressing and pressure bandage should nevertheless be applied. By this procedure rupture may be prevented, the tumor may be gradually diminished in size, and occasionally even dissipated. Youths or adults who are occasionally observed with stabile encephaloceles should wear protective plates properly curved, with bandages,

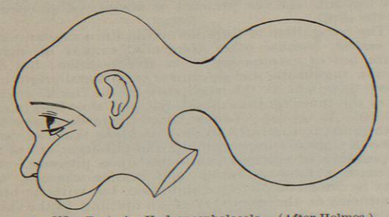

FIG. 813.—Posterior Hydrencephalocele. (After Holmes.)

and sometimes compression may be made use of, if it be deemed expedient to do anything, but great care is needed to avoid the production of brain symptoms. Puncture in simple encephaloceles is permissible only in case of continual augmentation in size—no other operative procedure is advisable. This is about all that can be done without risk. In many cases in which incisions have been made into these tumors, through a mistaken diagnosis, the result has been death from meningitis. Puncture and the injection of iodine have been fatal.

Meningoceles are more amenable to treatment, as a rule, than the other forms of cephalocele. Occasional punctures, together with compression, have met frequently with good results. Little is to be expected from iodine injection, although Paget was successful with it in one instance. The stabile meningoceles should, as a rule, be let alone, because they communicate almost always with the cranial cavity. Annandale in one case ligatured the peduncle and removed the tumor, the child recovering completely in spite of an attack of measles. Thompson has also cured a case by ligation. Schatz cured a case by the gradual compression of a clamp applied to the sac. Sklifasowsky removed an occipital cephalocele from a nine-months child with a ligature, the result being successful. The procedure is permissible in proper cases under strictly antiseptic precautions. Many operations in these cases have been undertaken since the inauguration of the new era of brain surgery, and much can now be achieved in the treatment of congenital cephaloceles, guided by the light of the latest discoveries in cerebral localization and by our improved technique in cranio-cerebral operations, which was formerly impossible. Fenger (*loc. cit.*) operated successfully on a nasal type, first removing the polypoid mass with the écraseur. There was free discharge of cerebro-spinal fluid. The nostril was packed with iodoform gauze preparatory to radical operation to close the hollow peduncle. He then did an osteoplastic resection of the superior maxilla (as devised by von Langenbeck), and transfixed and ligatured the peduncle. Aspiration of the tumor for fluid had been twice negative. He considers in this class that extirpation should always be practised even if the tumor is small and causes no more disturbance than an ordinary polyp, as there is always danger of meningitis from accidental injury or surface inflammation. Preparatory operations are necessary, and these vary with the different situations in the nose.

Möller (*Deutsch. Zeitschr. f. Chir.*, 1898, xlviii., 23) has an article on the surgical treatment of hydrencephalocele, in which he says that the old rule of Heineke against operation has now been overthrown, since the day when von Bergmann openly advised radical operation wherever possible (1888). Möller cites de Ruyter's (von Bergmann's assistant) rules, as follows:

Meningocele should certainly be operated, because of possible ulcerations, because the tumor presents a point of lessened resistance in which a meningitis may originate, and because the patient is greatly burdened by the presence of the tumor.

Encephalocele should be operated unless it contains too large an amount of brain tissue. It usually holds a somewhat unimportant part of the brain, part of the occipital or frontal lobe.

Hydrencephalocele should not be operated because of its communication with a ventricle.

To combat this last rule of de Ruyter, Möller cites a case of successful operation on an occipital hydrencephalocele, where the tumor was nearly the size of the infant's head. The peduncle was solid, the lumen having been obliterated. Examination of the child one year after operation showed it to be in good condition and of apparently normal mental development.

Schmitz (*St. Petersburg. med. Woch.*, 1898, N. F., xv., 193) reviews the forms of cephalocele and methods of operation, giving his experience of seventeen cases. He concludes that the permanent results of operation are very unsatisfactory. Life is prolonged, but in the case of hydrocephalocele, the child dies of hydrocephalus, or remains hydrocephalic. This he says is also true of meningoceles which show any signs of hydrocephalus. If there be no hydrocephalus, the prognosis is much better. Schmitz thinks the prognosis is best for the pure encephaloceles or solid tumors. It is important to note whether the tumor remains of original size or is disposed to grow, and whether it grows slowly or rapidly. In the latter case we must assume increasing intracranial pressure, and we have therefore a hydrocephalocele, or such a type is developing from the original encephalocele. Another question concerning the advisability of operation relates to the contents of the tumor. If sincipital, the middle frontal convolution is involved, and we may feel justified in removing a small portion. No motor and little mental disturbance will result. In the occipital class there is usually cerebellar tissue (hemisphere) and less frequently parts of the occipital lobe, and no symptoms are likely to follow removal of small peripheral portions of the cerebellum. In case of large prolapsus of brain substance, excision should not be practised.

Berger (*loc. cit.*) believes radical operation to be justified in cases in which the tumor is growing and in which the prognosis is bad without interference.

Tilmann (*Berlin. klin. Woch.*, 1895, xxxii., 1055) says that the old methods of compression, puncture, iodine injections, and use of ligature are not now considered in the

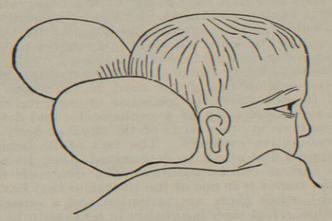

FIG. 814.—Tumor Divided in Two. (From Reall.)

treatment of meningocele. Even if infection is avoided there is always danger of meningitis. He cites two cases of cephalocele operated at Bardeleben's clinic. The first was an occipital meningocele, the size of an apple, removed a few weeks after birth. The child developed normally. The tumor had a very small lumen and thick walls of œdematous fibrous tissue. The second case presented a congenital occipital meningocele the size of

the child's head. Operation was performed one day after birth because of insistence of the father. One litre of fluid was aspirated and the tumor sac then removed. There was improvement at first, but the child died on the twenty-third day with paralysis and convulsions. The case was one of meningocele with chronic internal hydrocephalus. He discusses fully methods of operation.

ACQUIRED CEPHALOCELE OR PSEUDO-CEPHALOCELE.—Among the cephaloceles which originate subsequently to birth, through disease of the cranial bones or subcutaneous fractures, the most common is the pseudo-meningocele. This is observed almost exclusively in infancy or early youth, but, in very rare cases, sometimes in adults after healing of the superficial skull coverings in fractures. Conner* has written the best article upon pseudo-meningocele that is to be found in American literature. Under the name traumatic cephalhydrocele he describes 2 cases occurring in his own practice, and makes an analysis of 22 others collected from general literature; 19 of these were due to subcutaneous fractures of the skull, 2 to old gunshot wounds of the skull and membranes where the scalp had reunited, and 1 was the result of trephining; 17 of the cases were under three years of age. G. Vivien has collected 8 cases, and Thomas Smith (St. Barth. Hospital Reports, vol. xx.) reported 2, and described 20 analogous cases. All of Smith's were under sixteen years of age.

The history is usually that a child falls upon its head, and that, several hours or days after, a fluctuating swelling develops under the scalp, which may at first be diffuse, but later, as a rule, assumes the form of a sharply bounded cystic tumor. It may reach the size of the fist or be larger. It is over the injured spot, fluctuates, and often pulsates. There is sometimes a hardened border to the tumor. Occasionally, by firm pressure, the fluid may be made to recede more or less completely into the skull cavity, when the fractured bone may be felt. When this is not possible, the break may often be felt after puncture and evacuation of the tumor. In many of these cases puncture has shown the contents to be cerebro-spinal fluid. In post-mortem examinations which have been made, the fluid has been found to lie under the pericranium, lifting it with its outer coverings like a cystic sac; a cleft is observed in the bone, and one of the same size or smaller in the dura mater, through which the sac and subdural cavity communicate. Occasionally the cortex has been injured, and in recent cases there would be signs of softening and of laceration of the other membranes. In a case of Lucas, the pseudo-meningocele communicated with the open inferior horn of the left ventricle. Two or three cases may be mentioned in illustration of this traumatic form. Southam (*British Medical Journal*, May 12th, 1888) gives the details in the case of a male infant, aged six months, that fell downstairs and sustained a subcutaneous fracture in the right parieto-occipital region. Fourteen days later there was a swelling over the fracture, oval, soft, fluctuating, and pulsating. Moderate pressure was applied with a pad of lint and a bandage. In a month the swelling had disappeared and the child recovered. There was still a marked depression in the bone. Conner's first case was a boy twelve and a half years of age, who fell from a wagon, a wheel passing over his head. He was not unconscious, and walked into a house near by. There was a simple depressed fracture of the right half of the occipital bone, and a non-pulsating swelling over the fractured area, which he supposed to be due to extravasated blood. A week later, serum began to ooze from the nose, and there was puffiness of the right eyebrow. He punctured and drew off four ounces of a pale straw-colored fluid. The sac rapidly refilled, and four weeks later five ounces were removed. There were four subsequent tappings, the fluid withdrawn aggregating nineteen ounces. The specific gravity was 1.007; no albumin; sodium chloride, 6.6 parts per 1.000. The boy was never unconscious or delirious, but there were some insomnia and mental irritability, and the pulse ranged from 100 to 120. Conner's second case was a hydrocephalic and rachitic colored child about two years of age that fell a distance of five feet upon its head. There was no scalp wound, but a swelling appeared very quickly over the right parietal bone. In a few days it measured four and a half by three and a half inches, and exhibited pulsation and fluctuation. A radiating fracture could be felt through the tumor. No treatment was undertaken, and nine months after the accident the swelling had disappeared.

There are several facts which explain the almost exclusive prevalence of traumatic pseudo-meningocele in children. Possibly the flexibility of the skull gives a peculiar character to the fracture. The intracranial pressure is certainly relatively greater in children than in adults, and a more active secretion of cerebro-spinal fluid is probably another factor. The increase in subdural fluid may be due in part to the injury to the head, or in part to nutritive disturbance, rachitis, or some such thing, which does not give rise necessarily to a general hydrocephalus. The pouring out of the fluid, its augmented secretion, and the expansive power of the growing brain, all aid in preventing closure of the osseous

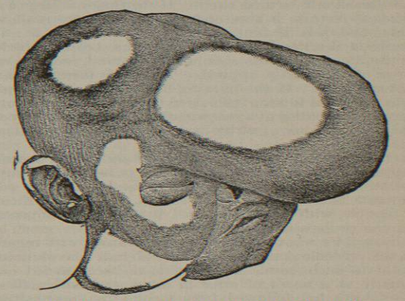

FIG. 815.—Sincipital Cephalocele. (After Vannoni.)

fissure, and, indeed, may actually cause its enlargement, and even eversion of its edges. But pseudo-meningocele is not a constant result of subcutaneous fracture in childhood, and it is likely that various causes and conditions conduce to its formation.

There are not many observations as to the further course of traumatic pseudo-meningocele in children. Conner found that fifty per cent. of the cases collected by him died of meningo-encephalitis. In some patients the tumor remains unchanged in spite of frequent puncture, and in others it is cured by this means. Spontaneous retrogression may take place. The cleft in the skull generally remains open, and hence the prognosis is always relatively unfavorable. These cases are often of considerable forensic interest. Bayerthal (*Deutsch. med. Woch.*, 1898, xxiv., 37–58) relates the case of a man aged eighty-one years, with a pseudo-meningocele still persisting, due to a brick falling upon his head when he was nine months old. The man was always normal, except that he did not learn to walk until four years of age, and that he developed epilepsy at the age of seventy-nine years. This is the only recorded case of such long persistence of a pseudo-meningocele.

Pseudo-meningocele in adults has been observed several times after gunshot wounds of the skull, when the scalp wound has healed, and once after recovery with defect of bone from trephining. Vivien mentions three such cases. As a result of subcutaneous fracture in adults it has apparently not been observed, and, if it occurs, must be extraordinarily rare.

* Phineas S. Conner: American Journal of the Medical Sciences, vol. lxxxviii., 1884, page 103.

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-