found that the first rupture of the vessel did not prove fatal, and that a second rupture occurred after a variable period, sometimes extending over a couple of years.

The majority of writers assert that the rupture of the aneurism gives rise to meningeal hemorrhage; but this opinion should be revised in view of the fact that in all (eleven) of Coats' cases, and in a number reported by other observers, the hemorrhage took place into the substance of the brain. The extravasation is generally so extensive that it breaks through the brain tissue into the lateral ventricles, and often also into the other ventricles.

Treatment of this affection is altogether futile. The most that can be done is to relieve the pain (which is often even more excruciating than that caused by other forms of cerebral tumor) by the administration of morphine. Trial may be made of potassium iodide, as in aneurism in other parts of the body.

Leopold Putzel.

BRAIN, ATROPHY OF.—As applied to the brain, the term atrophy embraces the results of three different kinds of morbid processes. In the first two there is really atrophy in the ordinary meaning of the term—that is, loss of substance through previously existing disease. Thus: First, loss of substance, either from idiopathic disease inherent in the brain, progressive general paralysis; or associated with general disease, infectious fevers; or, from failure of nutrition, senescence, insanity. Second, loss of substance from destruction of tissue, or from a sclerosis secondary to localized destructive lesions. In the third class there is no proof that a tissue once existing has been destroyed, and the term atrophy can be used only by a certain license. But for practical convenience there are many reasons for ranking the cases in question with the atrophies. They are, third, cases of imperfect development of the brain, cerebral agenesia, partial or general (microcephalus).

This classification of cerebral atrophies is traversed by another division into congenital and acquired; since many cases of arrested development of the brain are clearly traceable to diseases identical with those which occur after birth. The agenesia is then secondary, and except from its intra-uterine origin, could be referred to the second class of cases.

Congenital, Primary, and General Atrophy of the Brain constitutes microcephalus, the rarest form of idiocy (Ireland). No destructive lesion is discernible, but the size of the brain as a whole is much below normal, and the internal proportion of its parts is disturbed. The dimensions of the cranium correspond to those of the brain, and thus during life the size of the latter can be estimated by measurements of the former. Normal mental faculties are impossible with an adult head of from eleven to thirteen inches in circumference and from eight to nine inches from the root of the nose to the posterior border of the occipital bone. A circumference of fourteen to seventeen inches and an antero-posterior diameter of eleven to twelve decimetres implies a brain too small for ordinary intelligence (Voisin). Ireland calls microcephalic all adult heads below seventeen inches in circumference.

The lightest human brain on record weighed six ounces, in a child five months old (Sanders). In this the circumference of the head was less than six inches. Several brains are described of seven ounces (normal weight for men, forty to fifty-two and one-half ounces; for women, thirty-five to forty-five ounces. In microcephalic brains, the frontal lobes taper to a point; the occipital lobes either taper, or are much shortened, in either case exposing more or less of the cerebellum. The parietal and temporo-sphenoidal lobes are the best developed. The corpus callosum is often thinned, or shortened at its posterior end. The island of Reil may be left uncovered. The convolutions are remarkably simple; few secondary folds are developed. The disposition of many sulci is changed, the fissure of Sylvius shaped like a **V**, instead of a **Y** (Vogt); the calcarine fissure is prolonged so as to separate the gyrus fornicatus from the gyrus hippocampi (Putnam-Jacobi), the bridging convolutions of the occipital lobe are indistinct or wanting (Betz). In a celebrated hypothesis, microcephalic brains have been considered to constitute anatomic reversion to the ape type (Vogt). There is, however, no constant resemblance between the two; the brain of the microcephale may be simpler than that of orang or chimpanzee, it is not similar (Gratiolet, Adriani).

The intelligence of microcephalic individuals who survive is rudimentary, but at various degrees removed from imbecility, complete aphasic idiocy. There is no definite proportion between the degree of intelligence and the size of the brain. In those who attain adult age a certain amount of speech is often possible, and there is some power of attention, memory, and emotionality. Sometimes the amount of intelligence is remarkable, if we consider the dimensions of the cranium (case, Antonia Grandoni; circumference of head, at age of forty-one, thirteen inches). There is usually full and restless use of the limbs, and the impressions of the senses are lively. The absence of local or general paresis distinguishes cases of pure microcephalus from those in which the arrest of cerebral development is associated with destructive lesions. Sometimes, however, the muscles are notably feeble, the characteristic restlessness of microcephaly is exchanged for immobility, varied by rhythmic, swaying motions (Meynert). In these cases there is probably nutritive alteration of the brain tissue; or deficient elaboration of its minute structure (Jastrowitz). There may be persistence of the molecular substance which precedes the formation of the medullary sheaths to the nerve fibres, or of the large granular fat cells, which belong to the transitional stage, and have been sometimes taken as evidence of congenital inflammation (Virchow).

Congenital Primary (?) and Partial Atrophy.—In this class of cases, one or more portions of the encephalic masses are absent, and no traces remain of destructive lesions sufficient to account for their disappearance. Thus they have been explained by a primary aberration of development. The explanation is most plausible in regard to partial or total defect of the corpus callosum (Hitzig). This great commissure begins to develop toward the end of the fourth month of pregnancy, by buds which appear on the internal lateral surfaces of the hemispheres, and grow simultaneously inward and backward. In partial defect it is the posterior portion of the corpus which is wanting, showing an arrest of the partly completed process. The arrest sometimes involves the fornix trigonum and septum lucidum, sometimes these are intact. In all recorded cases, the anterior white commissure is preserved. The lateral ventricles are distended. This fact suggests that an undeveloped corpus has been destroyed by a ventricular effusion, the latter subsequently reabsorbed. The cerebellum has several times been found defective. In one celebrated case, unique according to Hitzig, the organ was completely wanting, being reduced to two small knobs, at the base of a serous cyst which occupied its place under the tentorium (Cruveilhier and Combetta, case Labrosse). Hitzig explains this case by an arrest of development; the author, much more plausibly, by a serous apoplexy which had occurred in early or in fetal life. The loss of the cerebellum was accompanied by total absence of the pons; the pyramids arising directly from the cerebral peduncles. In Otto's case, the cerebellum was very much reduced in size by filling up of the occipital fossæ, through inflammatory thickening of the bones. The atrophy was therefore again a secondary lesion. Atrophy of the cerebral hemispheres is usually evidently traceable to destructive lesions. Sometimes, however, the traces of these have disappeared, and the atrophy then seems primitive. Cruveilhier describes the reduction of one hemisphere to one-third the size of the other. The space beneath the dura mater was filled with serous fluid. Isolated groups of convolutions are often atrophied: thus, lower part of the central convolution, with supramarginal and superior temporo-sphenoidal (Beach); central convolutions on one side (Virchow's *Archiv*, July, 1882);

central convolutions on both hemispheres (McNutt); right ascending parietal convolution (Bastian); right cerebrum, right crus, pons, and pyramid, left lateral pyramidal tracts (Pick); paracentral lobule, superior extremity of left ascending parietal convolution (Varigny). In many of these cases a primitive arrest of development has been assumed; in none is it demonstrated, or even probable (Steffen, Brouardel, Hasse). The entire class of cases should therefore be consolidated with secondary atrophies.

Congenital Secondary Atrophy.—This is partial or general, according either to the extent of the destructive lesion, or of the interference with the general development of the brain which may have been exerted by a localized lesion. Nutritive processes throughout the brain may be so disturbed that nerve tissues cease to grow, nutrition is diverted to the cranial bones, premature ossification of the skull occurs, with some degree of microcephalus.

On the other hand, with a fetal hydrocephalus, all the brain above the medulla may be destroyed and converted into a serous cyst, while the cranial bones continue to grow, until after birth the head attains a circumference of two feet (Cruveilhier).

The lesions leading to congenital cerebral atrophy are produced during fetal life, and do not differ essentially from those which may be occasioned during the process of parturition, or so soon after birth that the symptomatology becomes identical with that of strictly congenital lesions. They are, in addition to the hydrocephalic effusions just mentioned, hemorrhage, inflammation, and softening by steatosis.

Throughout fetal life the rapidly growing brain is exposed to a graded series of accidents, corresponding to varying tendencies to effusions from the cerebral blood-vessels—now serous, now sanguinolent. At a certain degree of intensity these dropsies or hemorrhages result in complete malformation of the brain (hydrencephalon, or anencephalon); more limited in extent, the effusion results in the formation of a cyst, whose contents may be clear serum, or serum stained with the coloring matter of blood. The cyst replaces whatever nerve tissue has been destroyed. Intra-uterine cerebral hemorrhage is much more liable to occur into the meninges than into the substance of the brain itself. A clot is then formed in the cavity of the arachnoid, and lying upon one or both cerebral hemispheres, which it is destined to compress during the process of its shrinkage. The subjacent tissues atrophy under the double influence of the pressure and of the sclerosis excited by its irritation.

Precisely such arachnoid hemorrhage, with consecutive atrophy, is a not uncommon accident of a difficult parturition; and is thus the efficient cause of many cases of idiocy and spastic contracture. Hemorrhage is also possible, but much less frequent, during the first months or year after birth. The cerebral atrophy, however, can be called congenital only when dating from accidents which occur previous to all distinct psychic developments, thus hardly after the first month.

Meningo-encephalitis is, during the period immediately succeeding birth, more frequent than hemorrhage, and also occurs before birth. An intense form, occurring at the third or fourth month of pregnancy, is probably the cause of the pseudencephalic monstrosity. Traces of a more tolerable, and even extensive, grade of inflammation are found in thickened brain membranes, enclosing shrunken masses of nerve tissue, or even empty of these. All but the most limited and localized cases of meningitis are liable to be complicated by hemorrhage from the tender blood-vessels. Localized basal meningitis may interfere with the development of the brain, and thus, if the paradox be permitted, cause a species of primary atrophy, with premature ossification of the skull. The most striking effect of such lesion, from its proximity to the optic chiasma, is a descending neuritis and atrophy of the optic nerves.

When a portion of the brain has been destroyed by either hemorrhage or inflammation, serum is poured out to fill the threatened vacuum. This non-encysted fluid is found both in the ventricles, in the cavity of the arachnoid, and in the subarachnoid space. It constitutes the so-called hydrocephalus *e vacuo*, which must always be interpreted as a secondary lesion, and not as the essential cause of the symptoms observed during life.

In certain cases a portion of the nerve tissue of the brain is found to have simply disappeared, sometimes throughout the entire depth of the hemisphere, thus leaving an opening which leads from the convexity pia to one of the ventricles (porencephalic defect, Kundrat). This lesion is not entirely peculiar to the new-born, for it has been found in adults after chronic brain disease (Cotard). In the new-born, however, it is due to a peculiar lesion, a steatosis of the nerve substance, which results in red or white softening, according as it is or is not complicated by vascular congestion and capillary hemorrhage (Parrot). The foci of steatosis contain a great number of granular corpuscles, due to the fatty infiltration of neuroglia cells; also free granulations and species of cylinders, resulting from deformation of the corpuscles of Gluge. The veins are occluded by thrombi. The nerve tissue is reduced to a milky pulp, and, if the process lasts long enough, is gradually reabsorbed. The lesion may be circumscribed to a mass the size of a filbert, or may extend nearly throughout a hemisphere. In the first case it is usually multiple; in the second, the corpus callosum is the most affected.

One of the most important anatomical consequences of any of the above-described lesions of the brain is a degeneration of the motor tracts which lead from the seat of the lesion to the lateral and innermost fasciculus of the anterior columns of the spinal cord (crossed and pyramidal tract). This degeneration involves two elements: atrophy of the myeline sheaths of the nerve tubes, and proliferation of the neuroglia tissue between them. These elements are combined in various proportions, and when the atrophic process predominates, it is difficult to say that a simple agenesia of the spinal cord has not complicated an arrest of development of the brain. The fibres of the pyramidal tracts assume their myeline sheaths much later than the other segments of the cord, beginning at the fifth month; but they should be completed at birth in the cord (Flechsig), though still incomplete in the cerebral peduncles (Parrot). Simple defect of these sheaths, therefore, without sclerosis, might, at birth or a little before it, result from a check to the developmental process, dating from some epoch of the sixth, seventh, or eighth month of intra-uterine life. A lesion occurring during the last month, at birth or shortly afterward, can determine only atrophy of the myeline sheaths through propagation of a nutritive irritation, which is further revealed in a co-existing proliferation of neuroglia. This, therefore, serves to distinguish between a primary and a secondary atrophy of the brain, the latter, except in steatosis, always involving irritation (see case McNutt). This degeneration can be traced by a column of grayish color, contrasting with the normal white, in the cerebral peduncle, half of pons and pyramid, on the same side as the lesion; and in the cord, through the posterior portion of the lateral column on the opposite side (Türck). A similar but much narrower gray tract occupies the inner part of the anterior column on the same side as the cerebral lesion. When the primary lesion is bilateral, the second is so as well. Under the microscope, the proliferated connective tissue is readily traced by its brilliant coloration with carmine. Descending degeneration may be observed after the steatosis of the brain, but much more rarely and less completely than at other ages or after other lesions; a fact which Parrot attributes to the incomplete development of the cerebral portion of the pyramidal tracts. The same condition should tend to avert sclerosis after any lesions which occur early in fetal life. Conversely, in the cases in which the symptoms of "spastic paralysis" indicate an extensive amount of secondary sclerosis, we may date the primary lesion back to near the moment of parturition.

According to Charcot, no cerebral lesions occasion sec-

ondary degeneration unless they involve the fibres of the anterior two-thirds of the internal capsule. These contain the superior fibres of the pyramidal tract. Atrophic lesions of the cortex are followed by diminution in the size of the corpus striatum on the same side, and, what is more singular, of the thalamus as well. The hemisphere of the cerebellum opposite to the lesion shares in the atrophy. When the lesion is localized primitively in one side of the cerebellum, the upper motor tracts are unaffected, but the olivary body on the opposite side wastes. Atrophic lesions of the basal ganglia are similarly followed by atrophy of the tracts below them, while those above remain unaffected. Lesions in the white substance of the hemispheres occasion no primary atrophy but what is due to direct loss of substance, and no secondary atrophy, unless they involve the fibres of the pyramidal tract (see *ut supra*).

Symptoms of Congenital Cerebral Atrophy.—These are of two kinds: 1, Visible lesions; 2, disturbance of functions.

1. Hemiatrophia facialis has been observed at birth, and then, plausibly, referred to a cerebral hemiagenesia (Emminghaus). No autopsies exist to confirm or annul this hypothesis. The hemiatrophia of the brain, if existing, could be a coincidence only of the lesion of the trigeminus, to which this singular affection is usually referred. The majority of reported cases are acquired after birth.

Unilateral or even monoplegic atrophy of the limbs is, however, frequently in cerebral hemiatrophia associated with motor paralysis and different forms of rigidity of the same limbs. Of the three symptoms, the paralysis is alone congenital; the atrophy of a limb or a segment of a limb, as a hand, is associated with diminished size of Ferrier's cortical motor centre for the part. It is questionable whether the cerebral atrophy is then the cause or the consequence of the peripheric lesion. Similar wasting of the cortical motor centre has been seen in an individual in whom the leg had been amputated many years before death. The cerebral atrophy was there evidently secondary. Many cases of congenital loss of the segment of a limb are due to intra-uterine amputation through ligature by amniotic bands. The accident then essentially resembles that of extra-uterine life.

Atrophy, in congenitally paralyzed limbs, appears after a time to be much more extensive than that of acquired cerebral paralysis. This is because the diminution of size depends principally upon interference with development. There is not loss of substance once acquired, but the affected limb remains behind its fellow in growth. Structural defects of the eye and ear are not unusual; thus, coloboma of the iris, eyelids, or retina, deformities of the external ear. Associated with the latter, as coincidently developed from the superior branchial arch, may be malformations of the mouth, harelip, cleft palate, etc. It is difficult to say, however, in what proportion the probability of such defects is increased by the existence of a cerebral atrophy compatible with life. Cerebral malformations of non-viable subjects are usually associated with other malformations of the body.

2. Disturbance of function. There are three in the motor sphere: Paralysis, rigidity, and convulsion; and to these must be added defect of intelligence. The sensibility is obtuse but not specifically affected. The paralysis is rarely complete, and by no means always localized. A general paresis of all the muscles of the body is the most frequent and earliest form of motor disturbance, even in well-defined unilateral cerebral lesions. The infant is able to move all its limbs, but cannot grasp with the hands, stand on the feet, sit up, or even hold its head up. Often after one, two, three, or four years the child acquires a certain amount of power in this respect. The motor paresis tends gradually to disappear, as in animals upon whom lesions of the cortical motor centres have been inflicted. When this happens, the cerebral lesion in children may also be referred to the cortex, and the basal ganglia be presumed to be intact. It is not infrequent, even, that congenital cortical lesions which render children incurably idiotic are unattended by any paralysis whatever.

The symptoms of rigidity are especially interesting. The muscles of a single limb, an arm, may be affected with transient attacks of rigidity, but they rarely assume the permanently rigid contraction seen as a late symptom in acquired hemiplegia. The most characteristic form is the spastic paraplegia of the lower extremities. This is by no means exclusively due to cerebral agenesia, for it exists without cerebral symptoms, as a symptom of primary cord disease. But when it is congenital, associated with defective intelligence or disorders in the range of the cranial nerves, it may certainly be referred to atrophia cerebralis. Many entirely superfluous hypotheses have been made in regard to this disorder. There are two forms. In one the muscles of the lower limbs are permanently rigid, and principally the abductors of the thighs, the hamstring muscles, and the gastrocnemii. From retraction of the rigid muscles the thighs are abducted, the legs partly flexed on the thighs, the feet on the legs. In the other form the muscles are supple while the child is recumbent, but the attempt to stand or walk is immediately followed by spasm in the muscles indicated, or only in the abductors, so that the limbs are approximated or even crossed.

The bilateral character of spastic paraplegia does not always imply a double lesion of the cerebrum, though this was demonstrated in one remarkable case (McNutt) and inferred in another (Hutchinson). The immediate antecedent of all the spastic symptoms observed is, as in the late rigidity of acquired hemiplegia, secondary sclerosis of the pyramidal tracts in the lateral columns of the cord. Ninety-one to ninety-seven per cent. of the fibres of these tracts are normally crossed, the remainder direct (Charcot). The probabilities are, therefore, that the sclerosis and rigidity remain limited to the side opposite the cerebral lesion. But in children bilateral spasm may occur, either because the relatively minute extent of sclerosis on the same side as the lesion in the direct pyramidal tract exercises a greater functional effect, or because the proportion of decussating fibres has varied, leaving a much larger number in the direct portion of the tract, or because there has been a second decussation of motor fibres in the cord below the pyramids (Charcot).

When spastic symptoms have developed, paresis of the rigid limbs is liable to be masked by them.

The third motor symptom, convulsion, is irregular in the time and frequency of its occurrence, and variable in its intensity. Convulsions coming on within the first week or two after birth generally indicate that the cerebral lesion upon which the atrophy depends has been caused during parturition. In cerebral atrophy of intra-uterine origin convulsions are delayed for several months after birth. They often coincide with the first symptoms of rigidity in the limbs, or precede these a little, and seem, therefore, to mark the development of the irritative lesion of degeneration. Convulsions are never due directly to the atrophy itself, but only to the secondary lesions, as they descend through the pons and medulla. The convulsion may consist in a momentary stiffening of the body, followed by clonic agitation of one or more limbs, or it may be a complete epileptiform paroxysm. The slighter forms are often frequently repeated, sometimes even in a single day; the complete attacks occur at longer intervals, of weeks or even months. The close approximation of severe convulsions is dangerous, and a not infrequent cause of death. The intelligence is invariably impaired when the atrophy affects either cerebral hemisphere, or both, and is often deficient even in atrophy of those portions of the encephalon whose share in mental processes is not well understood, as the corpus callosum or cerebellum. It is probable in these cases that the finer structure of the cerebral cortex is imperfectly elaborated, even when no gross lesion is visible.

The degree of mental defect varies from simple flightiness and moral obtuseness (cases of atrophy of the cerebellum) to complete idiocy, and all intermediate stages

of imperfection of the intelligence are observed. The speech is sometimes disproportionately affected. There may be complete aphasia, yet the expression of the face and the actions indicate considerable intelligence. It is possible, then, as Gerhardt suggests, that the lesion is localized in the speech centre. In other cases there is no true aphasia, or inability to frame verbal conceptions, but articulation is interfered with by spasms of the facial muscles, analogous to those excited in the limbs by the attempt to use them.

Complete idiocy can be recognized in babies of three or four months old by the vacant expression of face, the inability of the child to recognize its mother or nurse, its inattention to all the usual objects of baby interest, the absence of the smiles and cooings of a healthy child. The slighter the degree of mental defect the later is its recognition possible, as it will become apparent only as the conditions of existence become complete.

The cranial nerves are often involved in either the primary lesions which have caused the atrophy or in the lesions secondary to them. Optic neuritis with secondary nerve atrophy may occur independently of any conditions of increased intracranial pressure. It is then attributable to a descending cerebritis. Early blindness in an infant renders detection of mental defect difficult or impossible. With or without blindness, irritative lesions of the sixth or third nerve may occasion strabismus or nystagmus. Coloboma of the iris, as well as congenital defects of other parts of the body, is not infrequently present.

Acquired Cerebral Atrophy.—This is always secondary, but may be general or partial.

General atrophy of the brain is found to a certain extent in all prolonged wasting diseases, especially phthisis. It is conspicuous in senile decay, but is not associated necessarily with any particular age. The brain is heaviest at thirty, but while some brains begin to degenerate at sixty, others resist decay at eighty or even older. The brain is always wasted in alcoholism, and also in chronic insanity, usually as a whole, sometimes more markedly in one hemisphere, or in certain lobes, especially the frontal. The most conspicuous general atrophy is seen in diffuse meningo-encephalitis (general paresis of the insane). The total weight of the brain may sink in this disease below 1,000 gm. (minimum normal weight for man, 1,130 gm.), and abundant serum may accumulate in the sulci. The frontal lobes are the most shrunken; after them the central convolutions; the cerebellum is always intact (Hitzig).

In all cases of chronic insanity, and most markedly in those of general paresis, the ganglionic cells of the cortex degenerate, become filled with fatty and pigment granules, and lose their protoplasm, nucleus, and axis cylinders.

In general cerebral atrophy both hemispheres are found sunken, retreating from the cranial bones, and the convolutions are thin and small, separated by wide furrows. In these enlarged sulci, as also in the dilated lateral and third ventricles, a serous effusion replaces the brain substance, as in congenital atrophy. The cortical cover of the ventricles may be so atrophied that the arachnoid and pia come in contact with the ependyma entirely, or in limited spaces, constituting an acquired porencephaly.

Generalized atrophy may also result from localized lesions of the cortex, whose ganglionic cells seem to have an influence on the general nutrition of the brain, analogous to that of the ganglionic cells of the cord on the peripheric nerves. Finally, mental inactivity predisposes the brain, as other unused organs, to atrophy. Thus probably is explained the frequency of premature senile atrophy among peasants and other uneducated people.

Partial atrophies occur at the seat of a destructive lesion, and also in regions functionally correlated with this. Of such lesions, hemorrhage is relatively less prominent than in fetal life: embolism and thrombosis from atheroma of blood-vessels are the most frequent. Both are attended by red or white softening, or even by true localized encephalitis; the brain cortex may be infiltrated with leucocytes following the track of blood-vessels, and evidently exuded from them; and also with the granular corpuscles of Gluge, resulting from fatty infiltration of neuroglia cells. To these processes are due the characteristic yellow patches so frequently found on the cortex of the brain, varying from an entirely insignificant size to the entire surface of a hemisphere, and sometimes penetrating its entire thickness (Cotard). Cysts and sclerosis may be found in adult life as in congenital agenesia, and the waste of nerve tissue may result, in either case, in simple defect; but the yellow patches are peculiar to acquired atrophy. The cysts are surrounded by an ochre-colored zone, where the nerve tissue has been stained by hemorrhagic effusion. Localized meningitis, or the outward-bearing pressure of a ventricular effusion, may also determine atrophies, either partial, or, in the latter case, symmetrical and generalized. Tumors of the brain necessarily determine atrophy of the nerve tissue they replace; but on their periphery there is an irritative hypertrophy, due, however, to proliferation of the neuroglia cells (Adamkiewicz). A band of this same sclerosis surrounds the focus of any destructive brain lesion. In senile atrophy occurs the vacuolation of the brain, long known as "l'état criblé" (Durand-Fardel), due to the enlargement of the lymphatic spaces surrounding the blood-vessels. This dilatation results from prolonged and intense hyperæmia. Hasse observes that in partial atrophy the primary destructive lesions affect all elements of the brain substance; in secondary atrophy the nerve elements alone. But in general diffuse atrophy, as from diffuse meningitis, an irritative process is first initiated in the neuroglia, and the nerve elements gradually waste. A linear cicatrix never forms in the brain, whose tissues are incapable of retracting; but there is always a space to be filled by a liquid, or by débris, or by newly formed connective tissue.

The *symptoms* of acquired cerebral atrophy are essentially the same as those of cerebral agenesia, but differently combined, and existing in different proportions. When the lesion occurs in adult life, it can cause no arrest of development in the limbs, such as is so common in agenesia. Partial paresis or paralysis may be produced when the lesion is situated in the motor tracts. But superficial patches of atrophy of the central convolutions may be found in persons who had completely recovered from the paralysis caused by the initial lesion. As in agenesia, the rigid contractions of paretic or paralyzed muscles develop together with the secondary degenerations; the "late rigidity" of hemiplegics is not the immediate consequence of the primitive lesion. This rigidity occupies by preference the superior extremity, and is often confined to that, even when the lower extremity is also paralyzed. It acts in the sense of flexion, so that the arm is drawn to the side, the forearm partially flexed, the two last phalanges of the fingers flexed, while, from atrophy of the interosseous muscles, the first phalanx is allowed to be extended. Hence results the "claw-hand" as characteristic of hemiplegia. Tremors, localized clonic cramps, and general epileptiform convulsions occur. The latter may precede the cerebral atrophy and indicate the progress of a localized meningitis, of which this atrophy is to be the result; or they may indicate the progress of a descending sclerosis toward the pons; or they may announce the termination of the whole morbid process. Convulsions are most frequent with lesions of the cerebellum, the cerebral cortex, and the ventricles; but these belong to the primary process, not to the atrophy itself. In the former case other symptoms coincide—febrile attacks, temporary strabismus, irregularities of the pupils, headache. In the latter any mental deficiency previously existing deepens, even to dementia, and as the end draws near the patient becomes comatose. In the atrophy of general paresis, the loss of muscular power is an early and prominent symptom. There is no rigidity. The mental defects in acquired cerebral atrophy are even more varying in nature and degree than the motor symptoms. Sometimes for many years these may be absolutely nil; then, if the atrophy progresses, the mental powers become gradually impaired. Simple fail-

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