be confounded with granulations of the choroid or ependyma, with vegetations notably in the cavernous sinus (Hyrtl, 1862), nor with granular (aciniform) conditions of the meninges seen in inflammations. They are often clustered, become pedunculated, penetrate the opposed dura, and finally project into the overlying venous channels, and even excavate (in part repel) the bone. They are usually most abundant opposite the middle of the falcial sinus—more correctly opposite the largest parasinual spaces—but may occur on both sides, along nearly the whole length of this sinus, beside the transverse and tentorial sinuses, and even beneath the latter, also in the temporal fossa opposite the trunks of the medidural vessels and the frequently present temporal sinus. They occur not rarely 3 cm., and even farther, from the long sinus along the line of large incoming veins after the latter have left the pia, and also farther out at one point near the frontal border of the parietal bone, probably where a medidural artery bends to connect through the skull with temporal vessels. Hence, in general, they occur opposite intradural blood channels of some size, and especially those subject to continual variations in calibre (pulsation, ebb and flow). Their prevalent growth toward and into venous spaces has led to the assumption that the granulations themselves were venous structures, or opened into such (Key and Retzius, Kollmann, and others; it is not true that Trolard claimed this). But it is now generally thought that the injection of the spaces through the granulations must have been effected by the rupture of granulation vesicles.* From the intimate connection of the older granulations with the spaces, they become darker colored; or, on tearing the two structures apart, flecked with blood. But the younger free corpuscles are pale, whitish.

The parasinual spaces are said rather to diminish with age, while the granulations certainly increase. Up to the twentieth year they are rarely developed to any extent. In congenital defect of the heart, they appear early and excessive (case of Lambl, 1860; one of the writer). However, at all ages continuous or intermittent compression of the brain space, of whatever origin, is the most frequent cause of their over-development. Under this head fall intracranial tumors of any kind or seat. Repeated congestion, as in chronic alcoholism, is also an accepted factor, doubtless acting mechanically. Various systemic troubles, as nephritis and diabetes, favor the enlargement of these bodies, possibly by unusual variations in the encranial pressure.

In view of all the facts—viz., that while these corpuscles regularly occur opposite intradural (extra-arachnoidal) blood channels not necessarily venous, their growth is especially favored on the one hand by venous stasis, on the other by pressure from the side of the cranial space—it is evident that they result from the oft-repeated local oscillation of the arachnoid. As the cerebro-spinal fluid is subarachnoidal, it, especially when under pressure, forces that membrane at its weakest points into any depression, as that beside a dilated vessel —*the granulations always grow away from the cranial cavity, never toward it.* When, then, the favoring condition—be that even negative instead of positive—relaxes, the granulations, to the extent that they have formed, press against and penetrate superimposed structures, the continuous alternation of the conditions as continually favoring the process. This, as we believe, clear and simple explanation suffices for all the main features of these little growths.

Pathology, Clinical History, etc.—(*a*) Like the wrinkling of the skin, the turning gray and falling out of the hair, and many other processes, the granulations themselves should be considered pathological only when they become excessive or develop prematurely. (*b*) Calcification or ossification of these bodies is not unusual. They may contain deposits of so-called brain sand and even fat globules. (*c*) Where they penetrate into blood spaces and even a sinus, they so far interfere with the return current, and also favor thrombosis (only one case of the latter, Förster's, has been attributed to this cause). (*d*) Foveæ glandulares, sharp depressions or excavations in the inside of the skull (preceding *e* and *f*), quite analogous to that seen in aneurism of a dural artery, are sometimes found. From their more or less intimate relations with the sinus walls, Allen advises avoidance of the middle line in all operations upon the skull cap. However, the diploë itself is never opened by these growths as a new layer of bone always forms around the foveæ. (*e*) Small flat elevations of the external plate of the skull, opposite the foveæ, and hence near the superior median line, are mentioned by many. (*f*) Very rarely complete perforation of the cranium occurs.

1. Case of Weber-Ribes (1819; v. Pozzi); hole small, and covered by ligamentous membrane. 2. Luschka (p. 116); perforation of squamous portion of temporal bone. 3. Lecat (v. Heincke in Pitha-Billroth); pneumatocele capitis; skull at some points perforated by granulations of the dura. 4. Demme (1862; v. Mastin, "Venous Blood Tumors of the Cranium." Reprint, 1886). "On the left of the sagittal suture (posteriorly) was a sharp-edged opening the size of a cherry stone, through which protruded a Pacchionian granulation." 5. The writer's observation. Man of forty; had suffered over three years from a tumor of the brain; autopsy, August, 1887. At the favorite spot near the frontal border of the parietal, somewhat removed from the sagittal suture, there was a clean-cut, complete perforation of the skull by Pacchionian granulations. This was circular, fully 0.5 cm. across, surrounded even in the diploic portion by smooth continuous bone, and covered by periosteal membrane. At the border there was a very trifling over-projection of the outer bone plate.

It is recognized that such perforations—even when incomplete—may, from local injury, lead to the formation of epicranial blood cysts, inasmuch as the causative granulations usually traverse some blood channel.

(*g*) In a few cases growths of this class have pressed on passing nerves, causing local neuralgic or paretic symptoms. Though it has been claimed that at times these growths induce headache, it is probable that they are then but co-effects of some other cause. (*h*) In animals generally these corpuscles are not present (brains of sheep, calf, rabbit, dog, and cat examined). Luschka found them only in the horse. *William Browning.*

* This is a matter of interest in connection with the theory of brain pressure (*Hirndruck*). Bergmann lays much stress on the continuous (or intermittent) discharge of cerebro-spinal fluid through the granulations into the venous spaces; while Adamkiewicz ignores or argues against the existence of such discharge.

Bibliography (Modern).

Luschka: Virchow's Archiv, xviii.
L. Meyer: Ibid., xix., 1860.
H. C. Bastian: Quarterly Journal of Microscopical Science, 1866.
Key and Retzius: Studien, etc., 1875, I. Hälfte, Stockholm.
Labbé: Archives de physiologie, 1879.
H. Lähr: Dissertation, Berlin, 1880.
Kollmann: Correspondenz-Blatt für Schweitzer Aerzte, 1880, No. 18.
Browning: American Journal of the Medical Sciences, October, 1882.

BRAIN: SIMPLE MENINGITIS.—(Pachymeningitis, πᾰχύς, thick; leptomeningitis, λεπτός, thin; simple, as distinguished from cerebro-spinal and tuberculous meningitis; meningitis of the convexity as distinguished from basilar meningitis.) Meningitis, in general, was first recognized as an affection separate from disease of the brain by Morgagni, 1760. Epidemic cerebro-spinal meningitis first attracted the attention of Vieusseux, of Geneva, 1805, and of Strong, North, Fish, Hale, Miner, and Williams, of our own country, 1806–1814, and was at that early period easily differentiated from affections limited to the membranes of the brain. Parent-Duchatelet and Martinet, 1821, first distinguished inflammation of the dura and pia mater, and Guérin and Guersant, 1827–1839, first distinctly recognized and set apart the tuberculous, granular, or basilar form of the disease. The first clear descriptions of the exclusively "simple" meningitis, from a pathological standpoint, are to be found in the works of Cruveilhier, 1830, and from a clinical standpoint, in those of Andral, 1834, and of Rilliet and Barthez, 1843. The recognition of the fact that simple meningitis is always a secondary affection is the result of the more accurate post-mortem observations of the last two decades, in the light of the recent investigations concerning infections, and the contributions from rhinology and otology.

That the various forms of meningitis are caused by specific micro-organisms was pointed out by the workers in bacteriology, especially by Koch, 1882, who demonstrated the tubercle bacillus in tuberculous meningitis; by Leyden, 1883, who found a diplococcus in the cerebro-spinal fluid and the pia, which Fraenkel and later Hauser showed to be identical with the pneumococcus; by Weichselbaum, 1887, who showed the presence of the diplococcus intracellularis meningitidis in epidemic cerebrospinal meningitis; and by Foà and by Bordoni-Uffreduzzi, who described a third and a fourth variety of the meningococcus.

Weichselbaum in Germany and Adenot in France, 1884, showed that meningitis occurring during or after an attack of pneumonia depended as a rule upon the same micro-organism that caused the pneumonia. Later observers have found meningitis frequently due to the pneumococcus in the absence of pneumonia. Thus the pneumococcus was found by Grasset in cases of meningitis occurring with inflammatory rheumatism; by Gabbi and Puritz in cases of meningitis associated with periarthritis and endocarditis; and by Ellehorst in a case of meningitis apparently due to fracture of the base of the skull.

The diplococcus intracellularis meningitidis is believed by some observers to be a variety of the pneumococcus, while others hold that it is quite a different organism. The latter view is held by Jaeger, who believes that the diplococcus intracellularis meningitidis is the cause of epidemic cases, while sporadic cases may be due to the pneumococcus. Jaeger proposes the name tetracoccus intracellularis for the micro-organism described by Weichselbaum as the diplococcus intracellularis, since this organism frequently appears in the form of tetrads.

In 25 cases of suppurative meningitis reported by Netter the pneumococcus was found in 18, the streptococcus pyogenes in 4, the diplococcus intracellularis in 2, and the typhoid bacillus in 1 case.

Next to the tubercle bacillus, the typhoid bacillus is most prone to cause purulent meningitis. Cases have been reported by Roux, Adenot, Kamen, Honl, Hintze, Fernet, Monsi and Carbone, Stühlen, Tietine, and other observers.

Next in the order of frequency, after the typhoid bacillus, is the bacillus coli communis, which has been found by Howard, Biggs, Sestre, Scherer, and others.

A rôle in etiology has been ascribed to the bacillus pyocyaneus by Kossel, Pesina, and Honl.

Among the pleomorphic bacteria that have been described in the exudates of meningitis are the cladothrix asteroides of Eppinger, and the actinomyces described by Moosbrugger, by Honl, and by Lenzine.

Pachymeningitis, inflammation of the dura mater, presents itself in two forms, external and internal, purulent and hemorrhagic, representing entirely different disease processes. Pachymeningitis externa, the hemorrhagic form, is really the result of a degeneration rather than of an inflammation; but in the absence of definite knowledge regarding the genesis of this disease, the two forms may be best studied together.

Pachymeningitis Externa.—Accidents or injuries which directly expose the dura, or effect its separation from the bones of the skull, with consequent extravasation of blood, whereby is implied, at least, a "hidden crevice" or some communication of the dura with the air, lead at once to inflammation of the outer lamella which may extend so as to involve all the rest of the membranes of the brain. Carious processes of the ear constitute an even more frequent cause of this condition. A mere microscopic breach in the thin wall of bone that forms the upper covering of the tympanic cavity will bring pus from the tympanum to the dura. So, also, caries of the ethmoid bone (ozæna) or other bones of the cranium (syphilis, carbuncle) may excite this form of meningitis; and even without caries, purulent inflammation of the mucosæ in the ethmoid and frontal sinuses may extend to the dura through the natural openings of communicating vessels. This complication has been noticed more especially in erysipelas after "mixed infection," whose nature it is to spread. As purulent pachymeningitis rarely remains confined to the dura, but extends, as a rule, to involve the pia mater, the symptoms, pathology, and treatment of this condition will be further discussed with leptomeningitis.

Pachymeningitis Interna (Consult Plate XV., Fig. 1).—The disease of the dura which merits most consideration, from its frequency, limitation, and recognizability in life, is that affection of the inner layer which is characterized by the extravasation of blood and subsequent development of an adventitious membrane, commonly known as hæmatoma duræ matris, and technically described as pachymeningitis interna hæmorrhagica. With these characteristics it is plain that internal pachymeningitis does not supply the requisite conditions, nor rise to the nosological dignity of an inflammation in the modern sense of the term. It develops oftenest independently of all infection, and should properly be discussed as a subvariety of cerebral hemorrhage.

The pathology of this affection remains as yet obscure. The early anatomists and clinicians were fain content with descriptions of the condition without venturing to express opinions concerning the nature of the disease. It was commonly held and taught that the disease consisted in the extravasation of blood, and the only question discussed regarded its situation. Thus Abercrombie and Andral, 1807, maintained that the blood was effused between the dura and the parietal layer of the arachnoid so called; while Houssard, 1817, located the extravasation in what was then, and for the sake of convenience is still, known as the cavity of the arachnoid. The hemorrhagic nature of the affection was nearly lost sight of when Bayle, 1843, considered the hæmatoma as an inflammatory product of the dura, but it was again restored by Durand-Fardel, 1854, who believed in the development and organization of a flat blood clot. Heschl, 1855, regarded the membrane as a highly vascular connective tissue, a view which Virchow, 1856, with his predilections for cellular pathology, elaborated into a hemorrhagic inflammation of the dura as the first process, and a subsequent infiltration of blood as the second. The authority of these pioneers carried their views with almost undisputed conviction up to our own times, when the studies concerning the nature and processes of inflammation and infection naturally diverted attention to the condition of the blood and blood-vessels as prime factors in the production of the disease.

That hemorrhagic pachymeningitis is not the expression of an ordinary inflammation is shown by the fact that no amount of irritation of the dura will produce it. Injections of ordinary irritants into and beneath the membranes of the brain of lower animals may be followed by purulent, but never by hemorrhagic, pachymeningitis. On the other hand, the injection of blood with all its constituents sufficed, in the experiments of Sperling, to produce the typical signs and lesions of the disease. The rôle of the fibrin in these cases is evidenced by the fact that a membrane was not developed after injections of defibrinated blood.

Internal pachymeningitis consists, then, in the extravasation of blood, the formation of a blood clot which, when the effusion is not too great or rapid, is flattened by pressure, to become subsequently organized into a membrane. In the first stage of the disease process, the thin layer of coagulated blood soon begins to show, in the separation of its fibrin, a meshwork which contains multitudinous blood corpuscles. At this time there is no apparent connection with the dura, whose epithelium remains intact. In the consolidation which continues,

the clot assumes the appearance and density of a membrane, which now in reality develops, from the transformation of white blood corpuscles into spindle-shaped connective-tissue cells, whence the synonym, P. fibrinosa. The red corpuscles now gradually lose their coloring matter, which collects in spots on the surface, and in the texture of the membrane (P. pigmentosa) lose their regular contours, and finally become transformed into masses of protoplasm. Young vessels now connect the dura with the membrane, which becomes gradually more dense, thick, and adherent. In the mean time new layers of blood may be effused into the membrane already in process of formation, which consists thus of superimposed lamellæ—Virchow has seen as many as twenty,—for a time separable from one another. The effusion takes place chiefly upon the convexity of the brain, limited, in fifty-four of sixty-five cases collected by Kremiansky, quite precisely to the region covered by the parietal bones. It is rather more frequently bilateral than unilateral, being confined to one hemisphere in but forty-four per cent. of cases.

The source of the hemorrhage still remains a matter of dispute and doubt. Kremiansky thought it came from the middle meningeal artery, an origin which comports well with the situation of the clot; but Huguenin declares that he has never seen this vessel affected in any of his observations. This author is inclined to find the lesion in the veins which run from the cortex to the longitudinal sinus along the falx cerebri; and Pacchionian vessels have likewise been accused, but all alike without as yet satisfactory anatomical proof.

The chief danger of these effusions is pressure upon the brain, which shows itself in proportion to the amount of the extravasation. Huguenin has seen a hemisphere flattened by a large unilateral hæmatoma, which may be as large as a hen's egg—Eichhorst mentions effusions of 500 gm.,—and in some cases a lateral ventricle has been reduced by pressure to half its size. The great evil of pressure is obviated in many cases by the latitude allowed by atrophy of the brain substance, a condition rather, as a rule, coincident with hæmatoma of the dura. In fact, the greatest contingent of cases is found in connection with paralytic dementia, and cases independent of some degree of atrophy are comparatively rare.

When, from any cause, a real inflammation is engrafted upon this hemorrhagic degeneration, serum or pus may be found in connection with the blood which forms the hæmatoma. As curiosities in this direction, Virchow describes a hydrocephalus externus pachymeningiticus, and Weber saw, in a lamellated hæmatoma, blood in one cavity and yellow-green pus in another.

Various changes in the skull, membranes and brain have been observed in connection with pachymeningitis, but none so frequently as to belong to it of necessity. Thus the bones have been found thickened or thinned, with an agglutinated dura at times, the pia anæmic, hyperæmic and swollen, or cloudy and opaque, separable from or adherent to the dura, etc. The frequency with which general atheroma of the cerebral vessels is seen, with thromboses, softenings, apoplexies, scleroses, etc., of the brain, bespeaks the intimate relation of these processes to the development of the disease, in connection more especially with general paralysis, alcoholism, insanity, senile atrophy, etc.

Pachymeningitis is a much more frequent affection than is commonly believed. Savage records its presence in three per cent. of the autopsies made at the asylum at Bethlehem, and when it is remembered that there are more cases of dementia and insanity, not to mention alcoholism, out of than in asylums, it is seen that this percentage is far too low. It is safe to say that most of the cases remain undiagnosticated during life; and death, when it occurs, though perhaps caused by this affection, is ascribed to the disease in the course of which this accident develops. All authors agree in noting three-fourths of all the cases in the male sex, a proportion which corresponds to the relation of the sexes to the affections which produce the disease. For the same reason hemorrhagic pachymeningitis is a disease of advanced life. Exceptional cases at early periods of life—six months to eight years—have been recorded by Weber, Moses, Steffen, and others, mostly in connection with the venous stases from the strain of asthma, pertussis, etc., or the impoverished nutrition of blood-vessels from scurvy, leukæmia, and more especially pernicious anæmia; and cases have been more abundantly reported during adolescence and maturity in connection with tuberculosis, empyema, valvular lesions of the heart, the various forms of Bright's disease, the various infections (variola, scarlatina, acute articular rheumatism, and typhoid fever), and more especially local injuries of the dura (seventeen of seventy-four cases described by Schneider); but aside from these accidents, pachymeningitis remains a disease of age. The largest number of cases, twenty-two per cent., in the collection of Huguenin, occurred between the ages of seventy and eighty.

Symptomatology.—Internal pachymeningitis exists at times without a symptom to mark its presence. Moses reports such a case in a child, aged seven months, who died of catarrhal pneumonia. At the autopsy there was found a pachymeningitic cyst which covered the anterior half of the right hemisphere, though no sign of brain disease had ever been manifest in life. Slight extravasations often show no sign because of absence of pressure, or, if slowly effused, because of tolerance, which the brain acquires often in astonishing degree. In other cases the accident is overshadowed by symptoms pertaining to the original disease. These are, however, all exceptional cases. As a rule, the disease may be diagnosticated during life by signs which are not so valuable in themselves as in their etiological relations.

In the majority of cases the disease announces itself suddenly and violently. The patient is stricken with apoplexy. The hemorrhage may be so great as to cause death by compression of the brain within forty-eight hours. The nature of the disease, or more strictly, the localization of the hemorrhage, is, as a rule, in such cases impossible to determine. The first attack is not, however, usually fatal. In exceptional cases the patient may recover fully, but as a rule a train of symptoms ensues which more or less distinctly characterize the disease. These symptoms vary greatly in individual cases, vary according to the locality and extent of the effusion, as well as according to the nature of the original disease; but they do not differ in essential characters from the symptoms of meningitis from any cause. Headache, stupor, which may at any time deepen to coma, monoplegias, hemiplegias, or, in the irritant stage, unilateral twitchings and convulsions, limited at times to one extremity, or confined to the area of distribution of the facial nerve; aphasia, when the region of the language centre is compressed—these symptoms, together with an irregular or retarded pulse, vomiting, and more especially contracted or dilated pupils irresponsive to light, with little or no disturbance of general sensation, make up a group which as a rule distinguishes the disease.

But, as already intimated, it is not so much the symptomatology of the affection as its etiological relations which strictly define the disease. The general signs of meningitis refer especially to hæmatoma only when they occur in the course of general paralysis, chronic psychoses, alcoholism, chronic Bright's disease, pernicious anæmia, traumata, the affections mentioned in the discussion of the etiology of the disease.

Another distinguishing, but by no means so distinctive, feature to indicate the nature of the affection, is the recurrence of the symptoms. Total or partial recovery from all the general manifestations of meningitis is followed in pachymeningitis, as a rule, by repeated attacks, and though the special symptoms may show great variety in relapses or recurrent attacks, the general character of the new signs is definitely sustained.

The *Diagnosis* of pachymeningitis is based upon these two cardinal points: the existence of an underlying condition or causative disease, and the more or less rapid recurrence of the attacks. Cases are further characterized by suddenness of onset and rapidity of recovery. Dr. Whittaker had at one time under observation an individual affected with chronic alcoholism who was suddenly stricken with apoplexy on the streets. The patient was carried comatose to the hospital. The coma subsided in the course of a few hours, to leave a complete right-sided hemiplegia, which entirely disappeared in three days, leaving the individual in better physical and mental condition than for ten years. Many of the cases of so-called "serous" apoplexy, characterized by sudden onset, and more especially by speedy recovery, are really cases of pachymeningitis.

The predominance of symptoms indicating cortical lesion is another feature of diagnostic importance. Thus localized convulsions and contractions, monoplegias, contracted pupils, following an apoplectic attack in an individual predisposed to the disease by the factors already emphasized, point almost certainly to pachymeningitis.

Considerable diagnostic importance has been attached to rigidity of an extremity, which often develops as an expression of irritation of the motor centre of the extremity, to become later manifest by convulsion or paralysis.

Collins considers it rather characteristic that the cranial nerves always remain free.

A point of differentiation from other forms of intracranial hemorrhages is that the symptoms come on gradually and the stage of irritation is prolonged.

The age and sex of the patient must not be overlooked.

Basilar meningitis is differentiated by the youth of the patient, the family history, the presence of tuberculosis elsewhere, by its long prodromes, its insidious approach, its general and special hyperæsthesia, opisthotonos, boat-shaped abdomen, etc.

Cerebro-spinal meningitis prefers winter, soldiers, and children, occurs at times in endemic proportions, shows opisthotonos, herpes, and sometimes petechiæ, extreme hyperæsthesia, spinal lesions, and does not recur.

The *Prognosis* is always grave. Recovery without recurrence is possible, but not probable. The patient succumbs, as a rule, in a subsequent attack, if he does not fall a victim in the mean time to the original disease. The immediate prognosis is best established, as after any cerebral hemorrhage, by frequent observations of the temperature, whereby the degree of the rise after the initial depression incident to the shock would receive proper interpretation. A sudden or gradual elevation to a high grade (105° F.) at any time thereafter, independent of the original disease, is a sign of most ominous significance.

Therapy.—The treatment of pachymeningitis does not differ materially from that of any form of meningitis or cerebral hemorrhage. The application of an ice bag to the head, the local abstraction of blood by leeches or cups behind the ears or over the temples, "derivation" by purgatives (calomel, senna, croton oil), constitute the routine plan, which is sanctioned more by time and use than by benefit based upon demonstrable proof. Tranquillity of surroundings, with all the measures which make up a more or less perfect hygiene, is the most effective agent in prophylaxis in the chronic psychoses; while abstention from alcohol addresses the "causa indicationis" in cases dependent upon its abuse. Bright's disease, heart disease, pernicious anæmia, etc., in short, the underlying condition, calls for appropriate treatment, and paralyses, convulsive manifestations, persistent headaches, whatever symptoms may be left, are to be met with symptomatic treatment.

Pachymeningitis Cervicalis Hypertrophica is a peculiar subvariety of meningitis, produced by great thickening of the meninges in the cervical cord, and marked by severe pains in the back of the neck and both arms, with atrophy of the muscles of the neck and flexors of the hands, and final spastic paraparesis.

Leptomeningitis.—It is possible, as already stated, that a real inflammation may limit itself to the dura mater alone, but such a distinct circumscription is very rare. Inflammation of the dura extends, as a rule, so as to involve the pia mater. The same qualification applies to the pia mater, though a strict limitation to the pia mater is more frequently observed. The subsequent remarks apply more especially to inflammation of the pia mater, with which the dura is, or may be, secondarily affected in greater or less degree. It is taken for granted that cerebro-spinal meningitis and tuberculous meningitis, diseases due to special causes, are not included under the title leptomeningitis, which embraces all other kinds of simple meningitis of known or unknown cause.

Leptomeningitis is always a secondary affection. The cases considered idiopathic become, under closer observation, so much fewer every year that it is more safe to appeal to unknown primary affections than to subscribe to the possibility of a spontaneous or idiopathic meningitis of any kind. A thorough conviction in this regard will alone lead to the searching investigation necessary in many cases to discover the original disease.

Affections of the nose, accessory sinuses, and ear constitute by far the most fruitful causes of leptomeningitis.

Chronic suppurative inflammations of the tympanic cavity, which constitute over twenty per cent. of all diseases of the ear, frequently lead to meningitis through caries of the osseous roof of the tympanum. The roof of the tympanum is composed of an excessively thin plate of bone, which is indeed at times congenitally defective, so that the way lies open to invasion of the cranial contents.

A more or less open avenue is also offered in the course of, or along the sheaths of, the facial and auditory nerves, and the vessels which penetrate the petrosal fissure. Communication by caries may be also directly established between the cavity of the cranium and the mastoid cells; while indirect involvement of the meninges may follow phlebitis and thrombosis of the cavernous, transverse, and superior petrosal sinuses, as revealed by dilatation of the veins and local œdema in the region of the mastoid process. Tuberculosis plays a prominent rôle as a special cause in the production of all these processes, while syphilis furnishes a small contingent of cases through caries of the upper meatus of the nose.

Every meningitis whose cause is not obvious should excite suspicion of disease of the nose, accessory sinuses, or of the ear. So, also, diseases of the nose, especially those involving the accessory sinuses; and every case of otorrhea, which may sometimes reveal itself to the sense of smell in an offensive odor, before or in the absence of visible discharge, should excite the fear of possible meningitis.

Trauma or injury to the cranial bones constitutes a not infrequent cause of simple meningitis. When compound fracture has occurred, or direct penetration has been effected, the sequence is sufficiently simple. In other cases the meninges, though not directly exposed, become affected through phlebitis, thrombosis, or suppurations occurring in the patulous veins of the diploë, whereby is implied, as previously intimated, some hidden crevice or pre-existent communication with the air. A far more infrequent involvement of the meninges occurs at times when an abscess in the interior of the brain reaches its periphery, or bursts into a lateral ventricle to come in contact with inflections of the pia mater at the base of the brain. So-called brain "softenings," which consist simply of brain and tissue débris, and simple hyperæmias, the so-called "congestions" of the brain, including sunstroke, could not, with our present knowledge of the nature of infections, produce a leptomeningitis.

Next in frequency to the direct invasion of the meninges from disease of the nose, accessory sinuses, and ear are the metastatic processes from distant depôts of infection. Any one of the acute infectious diseases may be thus attended or followed by meningitis, which is justly regarded as the most serious complication which can occur—which, indeed, imparts a sudden gravity to an otherwise mild case of disease. Of all the acute infections, pneumonia is the disease in which this complica-

tion most frequently occurs. The intimate relations of tuberculosis of the lung and brain in the frequent sequence of basilar meningitis upon tuberculosis pulmonum, prepare us in a measure for the frequent supervention of meningitis in the course of croupous pneumonia. The same connection or relation has been observed also in cerebro-spinal meningitis, and bacteriologists have pointed out the striking resemblance of the micro-organisms found in these two affections. Pyæmia and septicæmia may be said to vie with pneumonia in the production of metastatic meningitis, while endocarditis, empyema, acute articular rheumatism, the exanthematous diseases—more especially variola and scarlet fever (aside from ear disease), and very rarely typhoid fever—diseases mentioned in the order of frequency, furnish exceptional cases. Hinsdale reports a fatal case of purulent meningitis in a newborn child, due to a bacillus belonging to the colon group, which had apparently gained entrance to the body through the umbilicus. As curiosities equally illustrative, however, of the nature of the process may be mentioned the cases of meningitis which have followed such trivial infections as vaccinia and mumps.

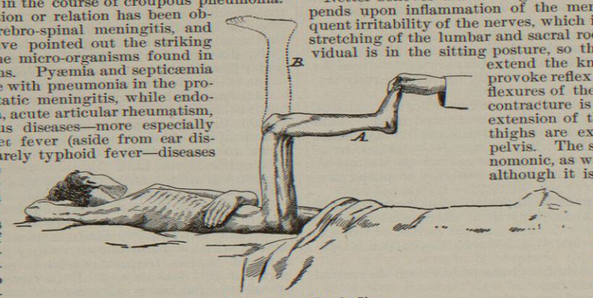

Fig. 993.—The Kernig Sign.

In a few reported cases, a paradoxical pupil reaction has been noted, the pupils being contracted to the size of a pinhead in the dark, and widely dilated in the light. The condition is rare and no satisfactory explanation of it has been given.

The morbid anatomy and symptomatology of leptomeningitis do not differ—aside from the fact that the convexity is more often involved than the base, when the inflammation is due to metastatic and traumatic causes—from the morbid anatomy and symptomatology of cerebro-spinal meningitis (to be described farther on, in this volume).

The *Diagnosis* of meningitis in connection with disease of the nose, of the accessory sinuses, or of the ear, or with a trauma of the bones of the cranium, is very easy, as a rule, but the diagnosis of metastatic meningitis is oftentimes exceedingly difficult. High fever and blood-poisoning may be productive of symptoms which so closely simulate the signs of meningitis as to render an absolute diagnosis impossible, at least for a time. The persistence of these signs after subsidence of hyperpyrexia sometimes declares the disease. Tuberculosis, pyæmia, scarlatina, variola, erysipelas, and typhoid fever are the affections which oftenest create doubts as to the diagnosis. But if close scrutiny be made of the etiological factors, and close attention be paid to the course of the disease, the diagnosis, as a general rule, soon becomes clear. In distinction from tuberculosis and typhoid fever, it may be said that meningitis develops quickly, almost suddenly, with violent pain in the head, active delirium, and often with stiffness of the muscles of the neck, or retraction of the head.

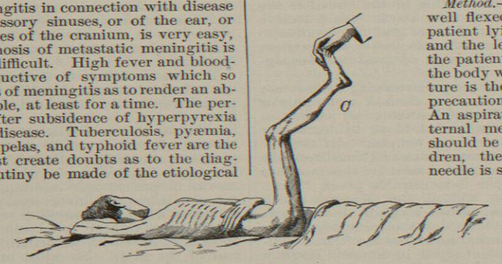

Fig. 994.—The Kernig Sign

The Kernig Sign.—In meningitis, according to Kernig, of St. Petersburg, if the hip be flexed so that the thigh is at a right angle to the body, the knee may not, without undue force, be extended in some cases beyond 90° (Fig. 993, *A*), never to the point of full extension (Fig. 993, *B*), and not farther than about 135° or 140° in any case (Fig. 994, *C*). With the hip extended, the knee may be readily straightened (Fig. 994, *D*). Thus, the ability readily to extend the knee, when the hip is flexed at a right angle (Fig. 993; *B*), would speak strongly against the presence of meningitis.

Netter believes that the sign described by Kernig depends upon inflammation of the meninges and consequent irritability of the nerves, which is increased by the stretching of the lumbar and sacral roots when the individual is in the sitting posture, so that the attempt to extend the knee is sufficient to provoke reflex contracture of the flexures of the leg. Such reflex contracture is not produced by extension of the knee when the thighs are extended upon the pelvis. The sign is not pathognomonic, as was at first claimed, although it is of great value in diagnosis. Netter has observed it in forty-five out of fifty cases of meningitis. Cipollina was unable to find it in some severe cases of meningitis, and did find it in other affections without the symptoms or lesions of meningitis. Packard has reported three cases of meningitis in infants, with the diagnosis confirmed by autopsy, in which the Kernig sign had been persistently absent. The absence of the sign was ascribed by Packard to a diminution of normal muscular hypertonia in infants. In another case the sign was present but no anatomical cause for death was found.

Lumbar Puncture (Quincke).—Puncture of the vertebral canal, which was originally used as a therapeutic measure, has proven of great value in diagnosis. At first the method was used in the study of diseases of the vertebral canal; but with the knowledge of the communication between the subarachnoid spaces of the brain and those of the spinal cord, the method has come into much more general use in the diagnosis of diseases of the interior of the skull.

Method.—The trunk should be well flexed, preferably with the patient lying upon the right side and the legs drawn up, or with the patient sitting up and bending the body well forward. The puncture is then made, under aseptic precautions, with a clean needle. An aspirating needle with an internal measurement of 1 mm. should be used in adults; in children, the ordinary hypodermic needle is sufficiently large. With the object of reaching the subarachnoid space at the beginning of the cauda equina, the needle is introduced between the second and third or third and fourth lumbar vertebræ. The puncture is made in the median line in children; in adults it is better to make the puncture close to the spinous process a little to one side of the middle line, in order to avoid the strong supraspinous ligament. The needle is introduced inward and slightly upward, to a maximum depth of from 2 cm. for children to 6 cm. for adults. When the needle enters the subarachnoid space, a varying amount of cerebro-spinal fluid will usually flow out, either in drops or in a stream. Occasionally the fluid will not escape or the flow will suddenly cease, probably through the occlusion of the needle by a flake of fibrin or in consequence of an obstructing filament of the cauda equina. To avoid injury to the spinal cord, it is better to detach the aspirator from the needle, and thus withdraw the fluid without force. Any fluid that is withdrawn should be examined macroscopically, microscopically, and bacteriologically. Thus may be determined, first, whether the fluid be clear, turbid, purulent, or bloody; and second, the micro-organisms that may be present either as the primary cause of the meningitis or as a secondary infection.

Osler confirmed the diagnosis in this way in thirteen out of seventeen cases. (See also p. 248 of the present volume.)

Tuberculosis and typhoid fever show typical temperature curves, with lung symptoms in tuberculosis, and abdominal symptoms in typhoid fever. In scarlatina, variola, and erysipelas it is rather a question of detecting a complication, as each disease shows characteristic eruptions upon the surface. Here, too, the persistence of cerebral signs after subsidence of the high temperature is of value. Septic and pyæmic diseases follow wounds, are attended with chills, and show joint affections and internal metastases. Ulcerative endocarditis, a septic process, has the same history. Uræmia is recognized by the dropsy, the condition of the urine, and, so far as the nervous symptoms are concerned, by the predominance of convulsions.

Cerebro-spinal meningitis is differentiated by the more prominent disturbances of sensation, by herpes, and by the occurrence of other cases. Basilar meningitis occurs more especially in children affected with tuberculosis elsewhere, or who come of tuberculous stock. It has long prodromes, and a longer duration. Its symptoms are less acute and intense. It more frequently implicates the membranes of the spinal cord. Pachymeningitis is a disease of age. It occurs in drunkards, and in cases of dementia paralytica, chronic insanity, etc. It shows a more fluctuating course. It must be repeated again and again that the various forms of meningitis are to be separated and recognized more by the etiological relations of the disease than by any difference in symptomatology.

The *Prognosis* is far more grave than that of cerebro-spinal, but not so absolutely fatal as that of basilar meningitis. The great majority of cases terminate fatally, in coma or convulsions, in the course of from two to ten days.

The *Treatment* of leptomeningitis does not differ in any way from that of any other form of meningitis; what little may be accomplished for the relief of symptoms will be mentioned under *Cerebro-Spinal Meningitis, Epidemic.*

The physician who is thoroughly indoctrinated as to the dangers of disease of the nose, of the accessory sinuses, and of the ear, and who is thoroughly familiar with the researches regarding the nature of infection, will prevent many cases of meningitis by timely treatment of the nose and ear, and by maintaining scrupulous asepsis in the management of all wounds of the skull.

James T. Whittaker.
George E. Malsbary.

BRAIN, SOFTENING OF.—The brain is said to be softened when its consistency is less than that taken as a normal standard. Diminution of consistence, however, shows but a part of the morbid work of an affection in which this phenomenon is only a circumstance quite accessory to the more important cerebromalacia, and the term in its literal acceptation could apply only to a diminution of the cohesion of the brain tissues, while their other properties remained intact. Softening, being a symptomatic word with a pathological meaning, like the word apoplexy, is rather a survival of a former belief, and its use is perhaps for this reason undesirable; but the name having been retained amid the fluctuations of opinions of writers on the subject, and in spite of its incongruity and the fact that it is often applied to cases in which there is really no softening, it is now employed to designate a necrobiotic process that occurs in the elements of the brain tissues and that is consequent upon nutritive changes ordinarily attended by sensory, motor, and mental disturbances, which may vary according as the lesion is circumscribed or general in character.

Pathological Anatomy—Descriptions of the pathological changes accompanying occlusion of the cerebral arteries and the so-called encephalomalacia are comparatively new. Their discussion is limited to contemporary authors, who have definitely settled certain points involved in the development of a new subject, notwithstanding diversity of opinion touching the nature and character of softening.

In determining the question whether the diminished consistence or diffluence of cerebral substance be of pathological origin, two sources of deception should be guarded against; for this condition may be the result either of cadaveric decomposition or of accident happening during the extraction of the brain from the skull. The genuine morbid appearances are then to be distinguished from the artefacta produced by the investigator, and from the spontaneous changes occurring after death. Nor should the condition be confounded with diminished density or specific gravity. Normal white matter gives a specific gravity of 1.040, but when softened it is from six to eight degrees of the hydrometer scale lower than the normal condition; and the consistency of the brain is the same in individuals of all ages excepting the newborn and very young children. Season, temperature, and the disease to which the patient has succumbed affect this consistence. An epileptic, dying during a paroxysm, in the month of June, by asphyxia from the penetration of food into the respiratory ways, has been known to present at the necropsy, twenty-four hours afterward, a completely diffluent encephalon. In a second instance, forty hours after death, with the same temperature, and in marasmus and an organic affection of the liver without cerebral symptoms, the brain was found to be in such a softened state that when it was placed on the hand the fingers penetrated by reason of its weight alone; while that of another cadaver, examined seventy hours after death and twenty-four hours after the necropsy, was far from this state of softening.

Though most frequent in old age, softening spares no period of life, its occurrence having been noted in the new-born and even in the fœtus. That form peculiar to the senile condition appears to have served as a type in all the descriptions given by the best writers, who seem to have overlooked the pathological conformity shown in the failure of the nutrition as the primary and common cause of cerebral softening at the two extremes of life. In the new-born the profound disturbance of nutrition which, through the intermediary of the blood, disintegrates an organ whose softness has not yet been effaced by age, is essentially the same as that which in the senile state prepares for arterial and cardiac lesions, the formation of embolism or thrombosis, and the consequent incomplete irrigation of the cerebral substance by the blood. This starvation of the brain consequent upon diminished supply of arterial blood is now spoken of as necrobiotic and non-inflammatory in its nature. Such terms, however, as cerebral infarction and necrobiosis, though establishing facts, do not explain the phenomena of cerebral disintegration.

Certain observers recognize in brain softening inflammation of an absolute kind; some regard it as a morbid state analogous to senile gangrene; others see a vital lesion affecting the nutritive integrity of the brain; while still others consider it as a lesion of nutrition owing to local ischæmia, provoked by arrest or diminution of circulation either in one of the cerebral arteries or in the capillaries of the brain.

This profound trouble of nutrition, the essence of which is unknown, may occur as a primitive necrobiosis of the brain substance, or this necrobiosis may be preceded by a vascular lesion that causes a want of supply of blood effected through embolism, the local formation of a thrombus, or by compression of the vessels. If a cerebral vessel be stopped, that part of the brain supplied by the vessel is suddenly deprived of nourishment, and