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.

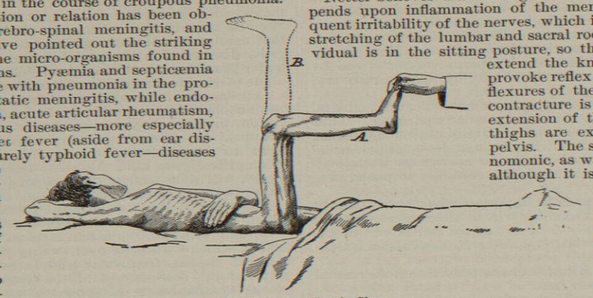

Fig. 993.—The Kernig Sign.

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.

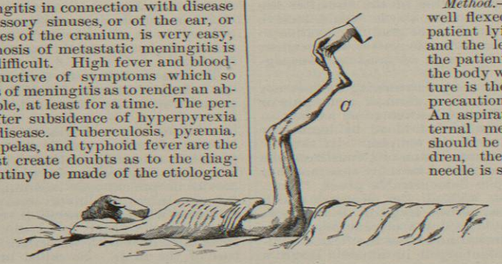

Fig. 994.—The Kernig Sign

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

this will be definitely the case if the embolism remains in place. When the vessel in question is a terminal artery, which does not exchange blood with other arteries, there will be immediate stoppage of cerebral function, followed by rapid changes of the brain elements, into fat and eventually into the liquescent state.

The anatomical characters and circumstances that preside at the evolution of cerebral softening have been fairly made out. Observations in this direction warrant the statement that the *arteria fossæ Sylvii sinistræ* is most exposed to embolic occlusion, which circumstance may be explained anatomically by the difference in the angles at which the left carotid and the innominate arteries are given off by the aorta. For this reason the left carotid is liable to catch an embolus coming from the heart, and the left Sylvian artery being the terminal artery in question, the regions provided by this vessel are consequently most in danger of being affected by the embolic process. These regions are the nucleus lenticularis, the terminal nuclei in part, the external capsule, and part of the internal capsule. The terminal branches of this vessel supply the second and third frontel convolutions, the island of Reil, and proximal surroundings, these being the portions of the brain most likely to suffer from the necrobiotic process. Further anatomical changes in the cerebral substance may result from the formation of an autochthonous coagulation, in consequence of degeneration of the intracranial vessels. Circumstances favoring the rapid formation of the thrombus are diminished motor force of the heart, roughening of the inner walls, narrowing and loss of force and elasticity of the vessels. Tumors of rapid growth, and inflammatory processes and their surroundings, which compress the vessels to such an extent as to lead to softening, are phenomena often observed in connection with softening. Occasionally the affected vessel is discovered entirely empty at the post-mortem examination, and for this reason it is supposed that the occluding mass has been reabsorbed.

The first fact of importance in connection with the pathological details of softening is the infrequency of anatomical changes in the cerebral substance when the seat of obstruction is on the cardiac side of the circle of Willis, which permits the prompt re-establishment of the circulation on account of its free anastomosis. If, however, the embolus is lodged in one of the terminal arteries of the basal arterial system, and a large region be thereby deprived of the necessary supply of fresh blood, there will be flowing back from the veins, and the tissues will become hyperæmic and œdematous, and filled with small extravasations known as hemorrhagic infarctions. In this simple necrobiotic change, the blood and coloring matter pass through the ordinary metamorphosis, the tissue of the brain swells and decays, leading to the rapid development of masses of granular cells, and finally to fatty emulsion. Later, the diseased focus may become reabsorbed and a cyst remain in the place of the softening, but this is rare; in fact, such a result may never occur except in the case of a small focus of inflammation. The process is more often followed by red sanguineous infiltration and yellow softening. The color, depending merely upon the amount of blood in the tissues, is, however, not an essential point, nor is sanguineous infiltration always present, and yellow softening is sometimes observed without its presence.

Etiology.—Although the etiology of the large group of clinical symptoms popularly known as brain softening touches one of the most delicate points of medicine, but little satisfactory is to be said regarding its remote causes. Among those which predispose more or less are old age (from fifty to eighty) or agedness, chronic alcoholism, syphilis, sexual excesses and fast life, Bright's disease, acute rheumatism, the dartrous diathesis, chorea, scarlet fever, insolation, intense cold, intense and long-continued intellectual exertion, severe and protracted emotional disturbance, misery, fright, overwork and responsibility, the abuse of opium, menstrual troubles, and, according to some authorities, the puerperal state. The cachexias and the inopectic diathesis (that is, a tendency to embolism, to thrombosis, and to coagulation of fibrin) are adjuvant causes, the importance of which should not be overlooked. Among negroes, the intertropical races, and the inferior races generally, softening and other forms of brain disease are infrequent, but the aptitude for such disease grows with the degree of perfection of the species. Women are less subject than men, principally for the reason that, being women, they do not undergo the strain and exhaustion of high brain energy and severe muscular labor, and are not so exposed as men to the poison and excitement of alcohol, syphilis, and tobacco.

Brain softening is the pathological sequence of many different conditions; but the more important causes that bring about the results in question may be stated as follows, when brought concisely together: endocarditis, through the production of movable products; myocarditis, through the formation of thrombosis in the heart; all processes in the lungs leading to coagulation or to the reception of septic material into the veins of the lungs; aneurism of the aortic arch; atheroma of the cerebral arteries; tumors of the brain, and even encephalitic foci; syphilis of the brain; the accumulation of pigment and pigmentous flakes in the blood, in connection with severe cases of malarial intermittent fever; and capillary occlusion through drops of fat. Other sources are injuries and inflammations of the bone, the occlusion brought about by pus cells or white corpuscles, and the blocking of the vessels by lime metastasis.

Of the phenomena of the occlusion of the intracranial vessels usually preceding the encephalomalacia, it is impossible to present a clinical picture in definite terms without going into the details of embolism, thrombosis, and hemorrhage, for full particulars of which the reader is referred to their respective headings. Although spoken of in the same connection, cerebral softening and occlusion of the cerebral vessels are not necessarily interdependent, since softening does not always follow occlusion, and for this reason there is a tendency among some to consider softening as a distinct pathological condition.

Symptoms.—Softening of embolic origin always begins by the symptoms peculiar to encephalic effusion of blood, or to those of apoplexy, the word being taken in its traditional sense. In fact, the symptoms are so similar to each other that their differentiation may be a matter of great difficulty to the most experienced. The symptoms that characterize the early period of *Embolism* are sudden. Without premonition the patient is seized with a sudden dizziness, or a momentary headache, and with an involuntary cry falls consciousless; motion and sensation appear to be extinguished, and unilateral paralysis, generally of the right side, follows. The only apparent difference between the symptoms occasioned by embolism of a cerebral artery and those of hemorrhage is the more transient state of the unconsciousness. For this reason part of the symptoms are often spoken of as apoplectic. In some cases, when the symptoms are less distinct, the unilateral paralysis forms a prominent feature. Other cases are prominently marked by more or less dizziness, by the absence of coma, by convulsions, and by unilateral symptoms. This seems chiefly the case when embolism occurs in a circumscribed smaller cortical region, or when the embolus is reabsorbed and the circulation restored. Vomiting sometimes attends the onset of the attack; at other times delirium of a transitory character occurs; and in many cases sudden aphasia results from the anæmia produced in the speech centre by the embolic occlusion of the artery supplying that region. There seems to be diversity of opinion among observers regarding the state of the pupils during the onset of the attack; it is probable, however, that they vary in different cases. At the fundus of the eye there may be found papillary or retinal œdema. It is only in chronic senile cases that papillary atrophy occurs.

Thrombosis of the cerebral vessels is usually attended by premonitory symptoms, as persistent headache, either diffused or localized, slight dizziness, a sense of general confusion, unilateral disturbances of sensation, and even

violent transient paresis. Further development of the trouble gives rise to excitement and active delirium, causing the patient to get out of bed at night and to commmit other unreasonable acts. Symptoms of depression may follow this period of excitement, the patient becoming apathetic and answering questions with difficulty. His movements are slow; there is tendency to somnolency; and a notable decrease occurs in the psychic functions, this decrease being characterized mainly by failures of the reasoning process, and by a more or less compromised memory, both of which are shown in verbal amnesia and other dysphasic affections. The humor is changing and emotional, and later the mental and other symptoms may become those of localized cerebral disease generally. As the patient goes into greater decline there may be bed-sores, and he may die from these; from an intercurrent malady, as a cardiac, renal, or splenic complication, or a pulmonary phlegmasia; or a new attack or some new lesion of the encephalon may carry him off. Generally the patient dies in a state of profound adynamia. The size and physiological importance of the occluded vessels determine the difference in the extent and duration of the symptoms. In fresh and vigorous subjects the occlusion of a small vessel may be followed by recovery, if the collateral circulation be established before the stage of necrobiosis begins. However, in most cases of autochthonous thrombosis that survive, the subsequent history is that of chronic localized brain disease, and of the motor, sensory, and intellectual disturbances that follow. The same may be said of embolic softening. Actual softening being fully established, the most prominent symptoms are permanent weakness, often persistent hemiplegia accompanied by athetoid spasms, and progressive mental weakness ending in paretic dementia. With the exception of the disturbances of vision, the affections of the special senses are the same as those that occur in connection with cerebral hemorrhage. The same is true of the sensory, trophic, and vaso-motor disturbances. Sudden amaurosis from occlusion of the arteria centralis retinæ has been observed to occur in some cases of embolism. Anæmia of the fundus has also been found; and certain observers attach much diagnostic importance to the arterial and venous hyperæmia of the retinal vessels and to congestion of the optic disc.

The paralyzed limbs are generally those of the right side, for the simple reason that the left Sylvian artery is oftener occluded than the right. Bilateral paralysis may, however, follow bilateral vascular occlusion. Since we are unable to offer any satisfactory explanation of either the presence or the absence of contractures of the paralyzed limbs, they can scarcely be regarded as of pathognomonic significance. Motor restlessness, though characteristic of the worst cases, is greatly influenced by heredity. A patient of bad nervous antecedents, with a spot of progressive softening in one of the corpora striata, may become noisy and restless and suffer from insomnia, while another, with no nervous heredity and under the same conditions, is quiet and manageable. As enfeeblement progresses, the motor symptoms are particularly noticeable in the paretic walk and aphasic speech, the latter resulting from a disturbance of the secondary coordination consequent upon a lesion of the basal motor ganglia. (See *Aphasia*.)

A brain affected by softening being on the verge of dissolution, the most prominent and troublesome symptom is the disturbance of the mental functions. The faculty most prone to fail is memory. The vesicular neurin not being susceptible to the impression of events, the patient is unable to recall recent experiences and impressions with distinctness. The destructive metamorphosis of the convolutional structure is further shown in the impairment of the reflective faculty or power of judgment; and as the cerebromalacia progresses the patient may become whimsical and peevish, or his affective power may be deadened, and the intellectual faculties may decline into childishness or gatism and finally become extinct.

The occurrence of these mental changes in connection with the situation of the brain lesion may be further studied under other headings (see *Brain Diseases: Diagnosis of Local Lesions*, and *Brain: Functions of the Cerebral Cortex*).

Diagnosis.—The diagnosis of cerebral softening should be based upon the history of the case and the proving of such fundamental conditions as may produce hemorrhage and embolism or thrombosis. Hemorrhage cannot be distinguished from thrombosis by any absolute means, notwithstanding the various diagnostic signs that have been proposed at various times; but the condition of softening may be established with a probability bordering on certainty when the associated symptoms are taken into account. When the premonitory symptoms have continued for a long time, the so-called apoplectic accidents point to cerebral hemorrhage rather than to embolism, but the symptoms in all probability may be owing to thrombosis. Gradual march of the paralysis indicates thrombosis rather than cerebral hemorrhage. The rapid appearance or disappearance of the attack, and the situation of a centre of softening in the left hemisphere, point rather to embolism than to thrombosis. It can also be affirmed with almost entire certainty that the encephalic foyer is of embolic origin when the presence of the symptoms permits us to suspect the formation of splenic or of renal infarction. Such symptoms would be likely to occur after a sudden attack in a young subject with an active *bruit de souffle* and enlargement of the spleen, pains in the lumbar region, and the presence of blood in the urine. Cerebral softening may be confounded with hemorrhage, encephalitis, hæmatoma of the dura, and with tumors. In tumors the speech and intellect are generally unaffected, and there is pain with convulsions, vomiting, double optic neuritis, and choked disc; in hæmatoma the history of the case is the main reliance in making out a correct diagnosis; while in encephalitis there is a considerable rise in temperature and the evidence of tissue action in the respective region of the brain, which is notably absent in softening. The lowering of temperature, almost constant at the outset of cerebral hemorrhage, is absent in softening; aphasia is more frequent; paralysis of a mobile character increases by abrupt stages, and death may occur during the initial coma. The signs of arterial atheroma are of no value.

Prognosis.—Softening of the brain, more or less grave according to the extent and intensity of the functional troubles, is a disease that ends in death after a certain time, varying from a few days to several months or years. More die of the acute than of the chronic form. Some think the malady curable, but it leaves ineffaceable marks in the most favorable cases, and the reported recoveries are to be looked upon with a great deal of doubt. They are said to have occurred in young and vigorous subjects; and when it is taken into consideration that the symptoms may have been incorrectly observed, or that they may have been those following various depraved states of the nervous system, the statement becomes more problematical. When not carried off by the initial symptoms, the patient is left with an incurable infirmity, in one of the most unfavorable conditions known. With the gradual enfeeblement of the intellect he is constantly threatened with new symptoms, and the reproduction of the original causes that produced the centre of softening. In fact, these causes greatly influence any forecast that it is possible to make concerning the march and duration of the malady. As a rule, it may be said that the indications are the more favorable in simple circumscribed embolism, and most unfavorable in cases of autochthonous thrombosis. The prognosis is worse when there is slight impairment of the intellect, sensibility, and motility taken together than it is when any one of these singly is profoundly impaired. A case may be regarded as hopeless as long as the underlying cause of the attack remains, and extreme gravity is to be attached to such symptoms as rise of temperature and bed-sores.

TREATMENT.—The causes that produce softening are difficult to remove and the therapeutical treatment of their effects is generally barren in results, notwithstanding correctness of diagnosis and the most judicious efforts to meet symptoms as they occur. Once established, senile softening is not amenable to treatment. Preventive measures being out of the question, except when the premonitory symptoms have continued for a long time, a consideration of the causes becomes fundamental matter, and the state of the heart and its action the main question. A declivous position of the head and perfect rest in a uniform temperature are advisable during an acute attack, while the body should be kept warm by artificial heat, warm clothing, and the cautious administration of stimulants. The caution in regard to stimulation is the more to be observed if there be the least suspicion of hemorrhage. In such a contingency it is deemed wise to act as if the case were one of cerebral hemorrhage, since hemorrhage is more likely to occur than occlusion, and the harm following stimulation in such a case seems to justify the diagnostic doubt. Symptoms pointing to a severe collateral hyperæmia may be treated with large doses of the bromides, sinapisms, dry-cupping, and mild purgation. The actual cautery and bleeding are to be avoided; but when there is general vascular irritation, leeches may be applied to the anus and behind the ears, in connection with intestinal revulsives and cold applications to the head. Digitalis, or strophanthus with glonoin, and amyl nitrite, are indicated, if the arterial tension be weak. Their use is, however, inadvisable in old persons. Recourse may be had to nervine tonics and to mild forms of slow derivation after the attack has passed. The diet should be strictly regulated, all intellectual effort should be interdicted, the integrity of the nutritive functions should be maintained as much as possible, and the methods of treatment applicable to the chronic symptoms of circumscribed cerebral disease should be generally observed.

Irving C. Rosse.

BRAIN, SURGERY OF THE.—HISTORY.—Although in 1871 Broca located a cerebral abscess in the speech centre, and greatly relieved the patient by trephining, modern brain surgery begins properly with a modest report of a case by Macewen in the Glasgow *Medical Journal* for 1879, xii., 210, and a later more elaborate paper, by the same author, in *The Lancet* for 1881, ii., p. 541. In these papers he narrates three cases, occurring in 1876 and 1879, in which cerebral disease was located by focal symptoms. The first was a case of convulsions of the face, arm, and leg, in the order named, following a fall on the right side of the head. A trephine opening of the dura evacuated two ounces of blood, and the boy recovered without any febrile movement.

In the second case the symptoms pointed to a lesion of the frontal lobe, and, after trephining, a tumor of the dura mater was dissected out. The patient died eight years subsequently from Bright's disease.

The third was a case of cerebral abscess, existing not at the site of a prior injury marked by a distinct cicatrix, but correctly diagnosticated in a totally distinct position, that is, in Broca's convolution, by the focal symptoms. The parents declined an operation, and the child died. After death an operation was done precisely as it would have been done during life, and an abscess was found, the size of a pigeon's egg, at the spot indicated by the localizing symptoms.

In spite, however, of this remarkable paper, the surgical world seemed blind to its opportunity. But in *The Lancet* for December 20, 1884, Dr. Hughes Bennett and Mr. Godlee narrated a case of subcortical tumor of the brain, diagnosticated by the localizing symptoms alone and before operation. When the dura was opened no tumor was visible: but so certain were they that a tumor existed that an incision was made in the apparently healthy brain tissue, and a morbid growth the size of a walnut was found one-fourth of an inch below the surface. This case, though ultimately unsuccessful because of suppurative meningitis, instantly arrested the attention of the surgical world by the precision of the diagnosis, the success of the operative technique, and the evidence it afforded that we could successfully cope with heretofore hopeless cases. Its very failure, like the failure of the first Atlantic cable, but pointed the way to success.

The first American paper on cerebral tumor, by Hirschfelder and Morse, of San Francisco, appeared in the *Pacific Medical and Surgical Journal* for April, 1886. The case was that of the successful localization, but unsuccessful removal, of a brain tumor. Two most remarkable papers on brain surgery, however, were published soon afterward by Mr. Victor Horsley, in the *British Medical Journal* for October 9, 1886, and April 23, 1887. In these papers ten cases were related, all of which were correctly localized; only one died, and the remainder were either benefited or cured. These were in part cases of removal of brain tumor and of portions of diseased brain tissue the cause of epilepsy, and in part cases of trephining for relief of intense headache, etc.

In this country, besides many excellent publications that I have not space or time to enumerate, the most noteworthy early papers published have been those by John B. Roberts, read before the American Surgical Association in 1885; two by R. W. Amidon, in the *Medical News* for January 21, 1884, and the *Annals of Surgery*, vol. i., 1885, both of these authors making strong pleas for early and more heroic surgical interference in affections of the brain; and one by Seguin and Weir, in the *American Journal of the Medical Sciences* for July, August, and September, 1888. I have also published several papers to which I may allude; the earliest two appeared in the *American Journal of the Medical Sciences* for October and

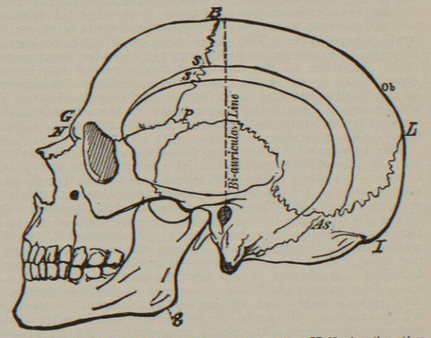

FIG. 995.—Skull Showing Points Named by Broca. *N*, Nasion (junction of the nasal and frontal sutures); *G*, ophryon (on a level with the superior border of the eyebrows, and corresponding nearly to the glabella, the smooth swelling between the eyebrows); *B*, bregma (junction of the sagittal and coronal sutures); *Ob*, obelion (the sagittal suture between the parietal foramina); *L*, lambda (junction of the sagittal and lambdoidal sutures); *I*, inion (external occipital protuberance); the basion is the middle of anterior wall of foramen magnum; *As*, asterion (junction of the occipital, parietal, and temporal bones). *g*, gonion (angle of the lower jaw); *S*, stephanion (or, better, the superior stephanion, intersection of ridge for temporal fascia and coronal suture); *S'*, inferior stephanion (intersection of ridge for temporal muscle and coronal suture); *P*, pterion (point of junction of great wing of sphenoid and the frontal, parietal, and squamous bones). This may be H-shaped, or K-shaped, or "retourné," in which the frontal and temporal just touch.

November, 1888, and in the *Medical News* for December 1, 1888. In Germany perhaps the most elaborate and important publication is von Bergmann's *Hirnchirurgie*.

In addition to these, Mr. Horsley published, in the *British Medical Journal* for June 16, 1888, an account of the removal of a tumor from the spinal cord, and Dr. Macewen (*British Medical Journal*, August 11, 1888) published six cases in which the posterior arches of the vertebra had been removed for tumor and compression of the cord, and for an abscess in the posterior mediastinum, two of which (paraplegia from Pott's disease and fractured spine) had been published as early as 1886 (*Glasgow Medical Journal*, xxv., 210.) (See article on *Spine, Surgery of the.*) I shall refer in the course of this paper to a number of other publications, but I have thought it right to sketch thus briefly the *early* historical development of the subject.

Two things have made such brain surgery possible. First, the accurate localization of the functions (especially the motor centres) by Ferrier, Horsley, Fritsch, Hitzig, and others, by means of which we can with fair accuracy determine the site of a tumor, abscess, cyst, etc., by the focal symptoms. Secondly, the impunity with which we can trephine and open the dura mater, and interfere with the brain tissue itself, due almost entirely to the introduction of antiseptic surgery. To Horsley more than to any one else we owe the formulation of rules for successful brain surgery—rules which will be given at length hereafter.

The Danger of Trephining.—For the technique of trephining I refer the reader to the paper under that head in a later volume. But it is important further to consider the question of the danger involved in this operation. In St. Bartholomew's Hospital Reports for 1882, Dr. Walsham published a paper entitled "Is Trephining of the Skull a Dangerous Operation *per se?*" In this article he analyzes 686 cases, of which 417 survived, the mortality, therefore, being 39.3 per cent. Dividing these large numbers into classes: first, those in which preventive trephining was used (nearly all for fracture), there being no cerebral symptoms, the mortality was 21.9 per cent.; second, trephining in which severe cerebral symptoms existed, the mortality was 48.4 per cent.; third, trephining in which moderate cerebral symptoms were present, the mortality was 27 per cent.; fourth, late trephining, mostly after inflammation had set in, the mortality was 58.5 per cent.; fifth, secondary trephining showed a mortality of 22 per cent.

Walsham showed that in 122 cases of late trephining, in which there was no condition endangering life, only 10.6 per cent. died. Amidon, in the paper referred to, analyzes 100 cases of trephining reported since 1879, in most of which antiseptics were employed. Of these 100 cases 26 died, but of these 23 presented at the time of operation symptoms already endangering life. He therefore concluded (and most later writers practically concur in his conclusions) that the mortality of trephining *per se* is but 3 per cent., a conclusion which would seem to be confirmed by Prunières and Robert Fletcher, by their investigations, which show the frequency of recovery in cases of prehistoric trephining. Seydel ("Antiseptie und Trepanation") even estimates the mortality as only 1.6 per cent.

Not only have antiseptics thus diminished the danger of simply opening the cranium, but the numerous cases which have been reported of opening the dura, even when followed by removal of tumor or of some brain substance, clearly show that only moderate danger is added in any case by such surgical procedures. "Heretofore," says Amidon, "the reluctance of the surgeon to open the cranium seems avidity as compared with his hesitation in piercing the dura mater. The cerebral cortex seems to be a 'dead line' inside the prison walls of conservative surgery, across which even the most daring are tempted or the most unwilling are dragged only by sure indications or desperate chances."

The future danger seems to be, however, that temerity may take the place of timidity, and that many patients will either die or go about with multilated brains that ought never to have been touched. This word of caution, therefore, at the outset may not be out of place.

TOPOGRAPHY OF THE BRAIN IN ITS SURGICAL RELATIONS.—The relation of the chief fissures and convolutions of the brain to the surface of the skull is of the greatest possible importance. It is essential that we shall be able, from fixed landmarks on the skull, to locate the various fissures and convolutions, and by them the various cortical centres. The subject has been studied by Reid, Horsley and Hare, Krönlein and others, each of whose methods of research has its own merit.

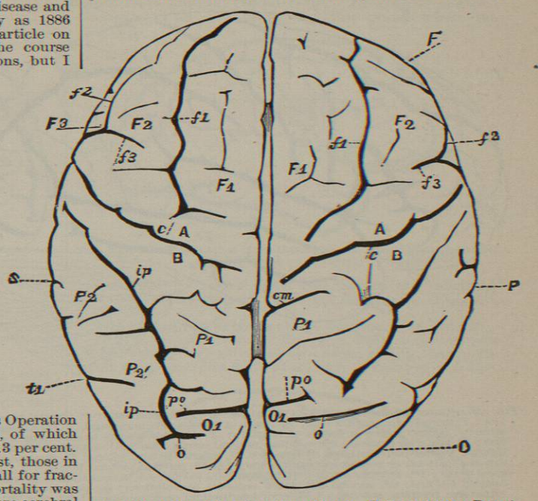

FIG. 996.—View of the Brain from Above. *F*, Frontal lobe; *P*, parietal lobe; *O*, occipital lobe; *S*, end of the horizontal branch of the fissure of Sylvius; *c*, central fissure or fissure of Rolando; *A*, anterior central or ascending frontal convolution; *B*, posterior central or ascending parietal convolution; F_1, upper, F_2, middle, F_3, lower frontal convolution; f_1, superior frontal sulcus; f_2, inferior frontal sulcus; f_3, vertical fissure (sulcus præcentralis); P_1, upper or postero-parietal lobule; P_2, lower parietal lobule, constituted by P_3, supramarginal gyrus; P_2', angular gyrus; *ip*, intraparietal sulcus; *cm*, calloso-marginal sulcus; *po*, parieto-occipital fissure; t_1, upper temporal sulcus; O_1, first occipital convolution; *o*, transverse occipital sulcus. (Ecker.)

I shall give a brief outline of the four methods, and in doing so I must take it for granted that the reader is fairly well acquainted with the chief outlines of the cerebral cortex. In order, however, to facilitate the study of the external topography of the brain, I have introduced cuts.

First: Fig. 995, from Broca, giving the points named upon the skull.

Secondly: Figs. 996–998, from Ecker, giving the names of the principal sulci and convolutions.

I have also appended two figures from Ferrier's "Functions of the Brain" (Figs. 999 and 1000) in order to fix as nearly as possible the relations of the various centres, so far as known at present. The figures in these two cuts are placed as follows:

1, On the postero-parietal lobule (precuneus), the centres for movements of the opposite leg and foot in locomotion. 2, 3, 4, At the upper end of the fissure of Rolando, and hinder part of the first frontal convolution, the centres for various complex movements of the