between the cerebellum, postoblongata, and occipital part of the cranium, with the spinal subarachnoid space.

E. The relation of the falcula (falx cerebelli) to the

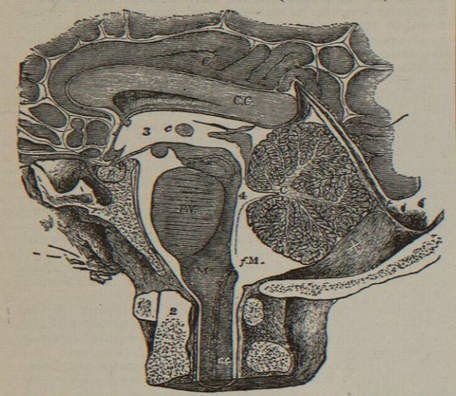

FIG. 805.—Medisection of the Cerebellum and Adjacent Parts. (From Key and Retzius, 1875, Taf. viii., vii., Fig. 1; after Shäfer: Quain, iii., Fig. 131, reduced and somewhat modified.) Compare Figs. 670, 800, and 801.

1, 1′, Atlas vertebra; 2, 2′, axis vertebra; 3, diacele (third ventricle); 4, epicele, the cephalic or cerebellar portion of the "fourth ventricle"; C, cerebellum; C.C., callosum; C′, callosal gyrus; M, postoblongata; P.V., pons; X, falcula (falx cerebelli); *c*, medicommissure; *c.c.*, just dorsad of (behind) the myelocele (central canal of the cord); *f.M.*, metapore ("foramen of Magendie"); *p*, hypophysis; *t*, torcular.

Preparation.—A blue mass was injected into the spinal subarachnoid space; the head was then frozen and mediseeted. The original includes the mesal aspect of the entire head, less the integument and mandible. The true encephalic cavities and the subarachnoid spaces are colored, so only the actual mesal parts appear.

Defects.—In the original there is no indication of the arachnoid, although the circumscription of the subarachnoid space was the very feature supposed to be illustrated. Should it be claimed that the arachnoid is sufficiently indicated by the ental boundary line of the dura, the answer would be that, although in places the two meninges may be in contact, they are not in all; furthermore, as distinctly shown upon Taf. vi. of the same work, in Figs. 801, 806, and 807, there is a point near the crest of the cerebellum (nearly opposite *b*) where the arachnoid (or its ectal layer) leaves the cerebellum and passes directly to the dura at the *foramen magnum*. There is no boundary between the metepicele (fourth ventricle) and the subarachnoid space; even if, as in other cases, the membranous roof of the metacele (metatela) adheres to the caudo-ventral surface of the cerebellum, the plexuses and the endyma constituting its ental surface must end somewhere.

Since the cavities are not colored, they appear as white areas without perspective, as if the preparation were a thin mesal slice. Most unfortunately, probably through some defect in execution, there is left a clear line between the epiphysis and the splenium, as if there were a passage from the diacele (third ventricle) to the irregular subarachnoid space between the splenium, epiphysis, pregeminum, and cerebellum. This is altogether misleading, for, as shown in Figs. 670, 687, 759, and 801, and stated in § 66, H, the diacele is completely circumscribed at that point by the endyma reflected from the velum upon the epiphysis. In the present copy this defect has been remedied so far as it could be by uniting the epiphysis and splenium so as at least to block the passage; but it should be remembered that it is closed not by nervous tissue but membranes. The editors of Quain have represented the missing metatela by the dotted line from near the number 4 to near the abbreviation *f.M.* A continuous line would have been more appropriate, and separated farther from the metacelian floor; that could not be changed in the present copy, but the interval representing the metapore (foramen of Magendie) has been enlarged; this, however, is conventional, and as if to correspond with the perhaps unusual condition shown in Fig. 690. Finally, the falcula (falx cerebelli), which was unmarked in Quain, is here designated by a cross (x).

mesal portion, vermis, of the cerebellum. In Fig. 707 this is obscured by the fact that part of the left lateral lobe remains.

§ 404. *The Cisternas.*—At several regions the ental layer of arachnoid is separated from the pia by considerable spaces, called cisternas by Key and Retzius, 1875, p. 93.* They are enumerated and described by Browning.

§ 405. *Fig. 806 illustrates:* A. The general appearance of this aspect of the cerebellum together with the oblongata and pons; in Fig. 697 these two parts were omitted.

B. The extent of the postcisterna (*cisterna magna* or *c. cerebello-medullaris*) upon about one-half the entire caudal aspect; there is, however, considerable variation in this respect.

C. The definite dorsal and lateral limitation of the postcisterna, although the boundary line is undulating and asymmetrical.

D. The lack of ventral boundary of the cisterna; the ectal layer of the arachnoid is attached to the dura so that this cisterna is continuous with the spinal subarachnoid space.

E. The union of the two vertebral arteries to form the basilar.

F. The origin of the postcerebellar arteries from the vertebrals near their junction.

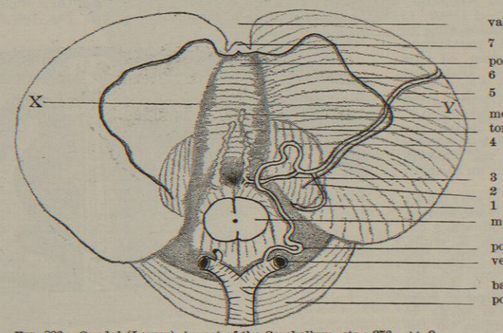

FIG. 806.—Caudal (Lower) Aspect of the Cerebellum, etc.; 376. × 2.

Preparation.—Through the kindness and skill of Dr. W. C. Krauss (a former student, now professor in the Medical Department of Niagara University, the brain was received fresh and in the dura. The cavities were injected with alcohol; the arteries first with alcohol and then with the starch mixture (see article *Brain: Methods*). The alcohol passed through the metapore into the postcisterna and thoroughly preserved all the parietes; it had access also about the myel, where the arachnoid was cut in removing the brain. The ectal layer of the arachnoid was cut away along the line of its attachment.

Defects.—The perspective of the postoblongata is defective. The metapore is vaguely indicated and few of the vessels are shown. Of the lobes only the tonsillas are outlined. The flocculi and nerve roots are omitted, also the rimulas (interfoliar crevices) on the left side. The most serious defect is the non-indication of the dorsal limit of the endyma which presumably accompanies the metaplexuses; see § 417.

1, 3, Branches of the postcerebellar artery, the former passing between the cerebellum and the oblongata, the latter apparently supplying the corresponding metaplexus; 2, 6, edge of the ectal layer of arachnoid bounding the area whence it had been cut away; 4, loop of postcerebellar artery, an example of its tortuous course; 5, main trunk of the artery near where it reaches the crest of the cerebellum; its branches are omitted; 7, mesal ridge formed by the vein which divides into a right and left branch upon the caudal surface; the arachnoid here forms a somewhat sharp angle.

G. The length and course of the postcerebellar artery, and the tortuous course of its central portion.

* Admitting that most of the cisternas do lie between the arachnoid and the pia, as commonly described, my later observations lead me to regard the postcisterna as between two layers of the ental arachnoid itself (see Figs. 805 and 807).

H. The passage of a branch of the postcerebellar artery mesad toward the metapore, apparently supplying the metaplexus.

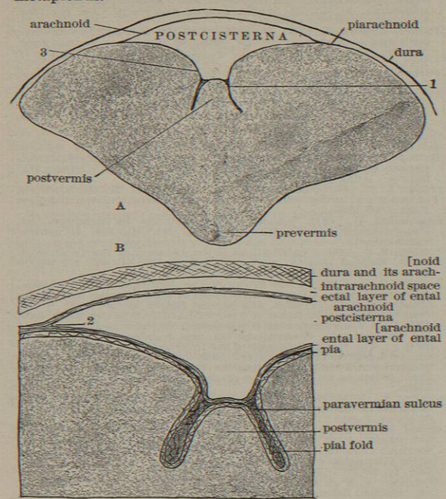

FIG. 807.—Sections of the Cerebellum and Postcisterna; semidiagrammatic.

A, Dorsal (cut) surface of the ventral portion of the cerebellum, together with the adjacent dura and the large "subarachnoid space," postcisterna, commonly called *cisterna magna* or *c. cerebello-medullaris*. At the meson appears the postvermis, separated by the paravermian sulci (1) from the large lateral lobes; 3 is the ental layer of the arachnoid. The meninges are here represented by lines only.

B, Enlargement of the meso-caudal region of A. The meninges are here represented by zones conventionally shaded; 2, the place of junction of the two layers of the ental arachnoid at the margin of the postcisterna.

Preparation.—An adult cerebellum (2,891) was divided at a plane corresponding with the line X—Y in Fig. 806, so as to separate the dorsal two-fifths; on Fig. 801 the plane of section would be indicated approximately by a line across the unshaded (cut) surface connecting the points where the dotted lines from the words *nodulus* and *epicele* intersect the margin of that surface; as seen in Fig. 806 it passes dorsad of the plexuses. The ectal layer of arachnoid is represented as the continuous caudal boundary of the postcisterna, while in Fig. 806 it is supposed to have been trimmed closely along the line of its depression from the ental layer 3.

Defects.—For readier comparison with Fig. 806 the figures should have been inverted so as to have the postcisterna nearer the reader. In B the postcisterna is enlarged two diameters, but the several zones representing the meninges are disproportionately widened, and their shading is conventional for discrimination only, and not for the indication of histological structure. The ectal or dural layer of arachnoid was inadvertently omitted, and there is no indication of the two layers of the dura itself. The numerous rimulas and intervening foliums that were divided in the section are not indicated, and the usual relations of the pia and arachnoid to each other and to narrow encephalic depressions generally are illustrated only at the paravermian sulci. According to the present view * that the metapore is the orifice of an evagination, the postcisterna may be lined, in part at least, by endyma; but it was not recognized in this preparation, and even in the embryo represented by Blake (1898, Fig. 26) it seems to have disappeared at a lower level.

I. The extension of the metaplexuses dorsad from the metapore upon the cerebellum.

§ 406. *Postcisterna.*—Notwithstanding the presumption that all the cisternas form a continuous series, my observations, up to the present time, induce me to regard the space in the angle between the cerebellum and the oblongata as presenting an important peculiarity, viz., as lying, not between the pia and the visceral arachnoid, but between two layers of the latter. The facts upon which this view is based cannot be detailed here. The view is indicated upon Fig. 807.

I am aware of the difficulties involved in its acceptance; without question, the postcisterna communicates on the one hand with the true encephalic cavities through the metapore, and on the other with the spinal subarachnoid space; its free communication with the other cisternas, although commonly accepted, seems to me not yet clearly demonstrated.

§ 407. *Is there Direct Communication of the Subarachnoid Spaces with the Intrarachnoid (or Subdural) Space?*—Whatever view they adopt regarding the constitution of the arachnoid as a whole, most writers agree that the arachnoid covering the brain and myel is continuous, excepting for the capillary spaces about the nerve roots referred to in § 401. Hence, while the neurolymph may pass to and fro between the true encephalic cavities and the postcisterna through the metapore, and may thus enter the other cisternas (§ 406) and the spinal subarachnoid space, it is nevertheless confined thereto.

But Dr. Langdon (1891) holds that "at the base of the cranium there are two points where the visceral [ental] arachnoid is deficient, one on either side, in the 'bridge' of arachnoid which stretches across from the cerebellar lobes to the under [ventral] surface on the oblongata. These foramina measure about half an inch (12 mm.) in longitudinal diameter by one-fourth inch (6 mm.) transversely, and are crossed by three or four fibrous bands, the attachment of which to the edges of the openings produces a multiple crescentic appearance of their margins, which suggests the name '*lunulate foramina.*'"

It will be noted that the location of these alleged lunulate foramina in the arachnoid corresponds with that of the ventral ends of the lateral recesses. Hence, on the one hand, if both are natural, the transfer of the neurolymph from the true encephalic cavities to the arachnoid space is provided for; on the other, the relation of the nerve roots to both the pia and the arachnoid renders both liable to rupture during extraction or manipulation of the brain. Hess implies (1885, Fig. 10, *ar.*) that the arachnoid was cut and reflected at this point. On the whole subject, and on the metapore see the later observations of Blake, 1900.

§ 408. *Fig. 807 illustrates:* A. The usual relation of the meninges in these respects, viz., the independence of the dura; the adhesion of the pia to the brain substance; the dipping of the pia into the narrow depression at either side of the vermis as a vascular fold; the adhesion of the arachnoid to the pia over most of the cerebellum, so as to constitute a piarachnoid (Fig. 796).

B. On the caudal aspect of the cerebellum, the formation of a considerable space, the postcisterna, by the separation of an ectal layer of the ental or visceral layer of the arachnoid.

§ 409. The inadequacy of the foregoing account of the postcisterna and its relations with the metapore is fully conceded. It is no disparagement to the labors of Blake and others to add that no account known to me is altogether clear, consistent, correct, and complete. The difficulties involved can be fully appreciated only by those who have already attempted to elucidate the subject. The material must be specially prepared for the purpose and examined by improved methods, both anatomical and histological.

BURT G. WILDER.

§ 410. The following list includes treatises upon the gross anatomy of the brain, mostly recent, likewise a few special papers; other papers are named in the text. Other things being equal, preference is given to such as contain full bibliographies. The history of Neurology up to 1822 is given in Burdach. For current literature consult the *Journal of Comparative Neurology; Anatomischer Anzeiger; Index Medicus; Neurologisches Centralblatt; Brain; L'Encéphale; Neurâxe; Jahresberichte*

* At the time § 83 was made up into the page I was unaware that the German edition (1894) of Minot's "Embryology" has this passage, p. 698: "The foramen of Magendie (Wilder's metapore) and the openings of the lateral recesses, according to this view, would be not true perforations of the ependyma, but the outlets of evaginations."

für Anatomie, etc. See § 12. *A. A. A. Proc.* stands for *Proceedings of the Association of American Anatomists.*

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BRAIN, ABSCESS OF.—Cerebral abscess is always the result of the introduction of pus-producing germs into the tissues of the brain. The organisms which are found most frequently are streptococcus pyogenes and staphylococcus pyogenes aureus. Staphylococcus pyogenes albus and citreus are also found occasionally, but are always associated with the former ones. When the pus from the brain lesion has been compared with that from the primary wound or otorrhœa, the same organisms have frequently been found in both places. Bacillus pyogenes fœtidus has been found in one case in the abscess and in the discharge from the ear. Fraenkel's diplococcus pneumoniæ was obtained in a meningeal abscess which accompanied an abscess in the cerebellum. In one case the observer was unable to decide whether the germ was Eberth's bacillus entericus or Neumann's bacillus meningitidis purulentæ. In several cases pyogenic organisms were associated with Gessard's bacillus pyocyaneus. In two cases oïdium albicans was discovered; in one of these the brain contained small abscesses which were filled with the fungus.

Sabrazès reports a case of abscess of the centrum ovale, abscess of the apex of the right lung, anthracosis of the lungs and purulent softening of an infarction in the right kidney. The pus of the abscesses contained a micro-organism which was regarded as a streptothrix. It is probable that the lungs were the primary point of entrance of the germs. Ferré and Faguet also found a streptothrix in a cerebral abscess. Fraenkel obtained pure cultures of tubercle bacilli from the pus of a tuberculous abscess of the brain.

These various germs may be derived from any part of the body, but in the large majority of cases the primary lesion is located in the vicinity of the head and is due either to injury of the head or to ear disease.

When the disease is due to injury of the head the pyogenic organisms are sometimes introduced directly from the outer world. For example, in punctured wounds of the skull, the foreign body (splinter of wood, dagger, etc.) may be the direct carrier of the staphylococci and streptococci into the brain. Sometimes the skull itself is infected, then infection thrombi develop in the diploic vessels, and the bacilli pass along the perivascular sheaths to the brain. The infective injury to the skull may also be produced without fracture, if the outer table has been laid bare.

In the majority of cases due to injury the abscess is found in the vicinity of the injured part of the skull, but in exceptional instances it is found on the opposite side of the brain (contrecoup).

A similar pathological condition is presented by affections of the face and scalp, which may set up thrombosis of the veins and thus communicate with the brain.

Next in importance—perhaps even equal—to traumatic causes stand inflammations of the middle ear. The primary lesion is generally present from early childhood, as a sequel of one of the infectious diseases common to that period of life. It terminates in caries of the petrous portion of the temporal bone, attended usually with suppurative otitis media.

Caries in the tympanic cavity usually extends most markedly in certain directions. When it spreads through the antrum and involves the mastoid cells, some of which are adjacent to the sigmoid groove, the latter is apt to be involved and exposed. When the caries attacks the roof of the middle ear, perforation into the middle fossa of the skull frequently results. When the petrous portion is the site of extensive disease, suppurative leptomeningitis of the basal ganglia and cerebellum is apt to occur; even when the destruction is much less marked and the internal auditory meatus is exposed, the infective inflammation may pass along the sheaths of the facial and auditory nerves. In perforation of the tegmen, the abscess is situated in the temporo-sphenoidal lobe; in perforation into the sigmoid groove, the abscess is generally located in the cerebellum.

The mode of development of the abscess may vary considerably.

If the tegmen is involved the overlying dura mater becomes inflamed and adherent to the adjacent pia mater, and thus the veins and lymphatics of the latter are directly affected. The arterial vessels become thrombosed, and particles from the resulting emboli are carried with the pathogenic cocci to the interior of the temporo-sphenoidal lobe where they give rise to an abscess. As in cases of injury, the process may also creep along the perivascular sheaths.

When the perforation takes place into the sigmoid groove, the outer wall of the sinus undergoes inflammation; this extends through to the inner wall and thus induces thrombosis. The thrombus offers a nidus for the micrococci which pass along the cerebellar veins to the cerebellum. It must be remembered in this connection that the current of blood in the superficial veins may flow in either direction. The cerebellar arteries may also be thrombosed, and thus convey infected emboli to the interior of the cerebellum.

Cerebellar abscess may also be due to direct extension of the inflammation through the dura on either side of the sigmoid sinus.

In young children the infection often travels through the petroso-squamosal suture.

Cerebral abscess from ear disease is much rarer after acute suppurative otitis media than after the chronic form. In rare cases it follows tuberculous and syphilitic affections of the temporal bone.

Otitic abscesses occur generally in the temporo-sphenoidal lobe or the cerebellum on the same side as the aural lesion. In 119 cases collected by Koerner, 79 developed in the cerebrum and 40 in the cerebellum.

Infection from the nose and its appendages is much rarer. In a case reported by Dreyfuss, the infection, which started from suppuration of the antrum, was due to thrombophlebitis of the pterygopalatine and ophthalmic plexuses. In another case the process extended to the adjacent ethmoid cells and caused perforation of the lamina cribrosa. When the primary lesion affects the frontal sinuses, the posterior wall usually undergoes perforation or the infection takes place through the diploë. In very rare cases the primary infection is located in the orbit. In almost all of this group of cases the abscess is situated in the frontal lobes.

Embolic abscesses of the brain may be due to infection in any part of the body. They are observed particularly in association with pulmonary diseases (putrid bronchitis, empyema, gangrene, tuberculous cavities). They are less frequent after septic endocarditis, suppurative processes in the abdomen, joint and bone suppuration. They have also been seen after typhoid fever, scarlatina, diphtheria, glanders, and general pyæmia. Boettcher reports a case after pulmonary suppuration in which lung pigment was found in the brain. In two instances the abscess was due to the presence of oïdium albicans.

The category of so called idiopathic abscesses of the brain is being continually narrowed. Although the existence of such cases cannot be absolutely denied, still none should be pronounced idiopathic until careful search has been made unsuccessfully in all the organs for some possible source of infection.

PATHOLOGICAL ANATOMY.—An analysis of 458 cases collected by Le Fort and Lehmann showed that the abscess occurred in the cerebrum in 327 cases, in the cerebellum in 113 cases, in the cerebrum and cerebellum in 11 cases, in the pons Varolii in 5 cases, in the cerebral peduncle in 1 case, and in the fourth ventricle in 1 case.

According to Macewen 93 per cent. of traumatic abscesses and 87 per cent. of otitic abscesses are solitary. The majority of metastatic abscesses are multiple. Abscesses of the brain vary extremely in size. Some of the multiple abscesses occurring in the course of pyæmia or ulcerative endocarditis may be the size of a pea. Old chronic abscesses may occupy a large part of a cerebral hemisphere, or perhaps a considerable portion of the cerebellum.

Acute abscesses contain a thin pus, usually of a color varying from yellow to green, sometimes of a darker shade, from admixture with blood. Under the microscope this is found to contain pus corpuscles, drops of myelin, granular matter, and detritus of the nerve elements and the pathogenic micro-organisms. The walls

of the abscess are usually irregular and jagged, and tear readily from the contact of the fingers. The cavity is irregular in shape at first, but at a later period it tends to become more rounded. For a varying distance around the cavity the brain substance may have a reddish, speckled appearance from enlargement of the blood-vessels and the presence of capillary hemorrhages. Still more externally the brain tissue is stained slightly yellow and softened from inflammatory œdema. The latter phenomenon is observed sometimes over a large area, and often appears to be the immediate cause of death. Sometimes the abscess ruptures into one of the lateral ventricles, and in rare cases the pus makes its way through the foramen of Monro into the opposite ventricle. In such cases death almost always occurs before ependymitis supervenes. Softening may extend to the ventricles and produce ependymitis even though perforation has not occurred. In other cases the abscess ruptures through the cortex (almost always at the convexity), and in this event, likewise, death may supervene before the development of meningeal inflammation. But in a large number of such cases meningitis occurs at the site of rupture and rapidly spreads over the entire meninges. Quite a number of cases have been reported in which the pus made its way through the original site of injury to the skull, through the external auditory meatus, through the sphenoid bones into the nasal cavity, or through the orbital plate into the orbit.

FIG. 808.—Abscess Cavity in the Brain. (Specimen in the collection of Prof. M. Allen Starr, M.D.: photograph taken by Dr. Edward Leaming.)

In the majority of cases the amount of healthy tissue between the cortex and abscess does not measure more than a few millimetres in thickness. In rare cases the abscess may be situated an inch or even more from the cortex. The abscess may be connected by a narrow fistulous tract with the meninges.

Chronic abscesses are usually round or ovoid in shape, and are provided generally with a connective-tissue membrane of variable thickness which lines the cavity. Rudolph Meyer states that about seven weeks usually elapse before the formation of a well-defined membrane. Lallemand found an abscess surrounded by a soft vascular membrane in a case which proved fatal thirteen days after the onset of the first symptoms. Huguenin found no membrane in an abscess which had lasted thirty-two days. Equally varying statements are made by other writers.

The membrane rarely insures a complete standstill of the process, inasmuch as it undergoes suppuration in places and this attacks the surrounding brain tissue. The new focus may again be surrounded by a fresh membrane.

The contents of the cavity are usually somewhat viscid, and consist of broken-down pus globules, drops of fat, cholesterin crystals, and granular matter. In a very few instances the contents have been found condensed to a thick, cheesy consistence, and in one case it is stated that earthy matter was present (calcification). When the abscess is of large size, fluctuation can usually be detected upon the convexity; the convolutions are flattened upon the side of the lesion, and sometimes the falx cerebri is pushed toward the opposite side of the brain.

When the abscess is situated in such a position (particularly the cerebellum) that the escape of fluid from the ventricles is interfered with, internal hydrocephalus may be the result.

The complications of cerebral abscess include suppurative meningitis, extradural suppuration, and thrombosis of the sinuses. All these conditions may also precede the development of the cerebral abscess.

According to Hessler, among 106 abscesses of the cerebrum 67 were uncomplicated, in 13 there was thrombosis of the sinuses, in 26 meningitis. Among 59 cerebellar abscesses 43 were uncomplicated, thrombosis of the sinuses was present in 10, and meningitis in 6 cases.

Macewen has reported a remarkable case of suppurative meningitis in the posterior fossa, while simple serous meningitis was found in other parts.

Widespread œdema of the brain is found not infrequently in cases of cerebral abscess, even in those provided with a thick membrane, and which do not appear to have given rise to much pressure. General cerebral anæmia is also observed quite often under such circumstances. The development of these sequelæ is still unexplained.

CLINICAL HISTORY.—The symptoms of an acute abscess of the brain after injury vary to a remarkable extent. Perhaps the immediate symptoms of the traumatism have been very slight; the scalp has been wounded, but the bones have escaped injury, and the patient is not supposed to be seriously injured. Or the patient has presented for a few days the signs of cerebral concussion,

and then apparently recovers. For a variable length of time (from a week to two or three months) he continues in apparently good health, but then begins to complain of headache, dulness, and irritability. The headache is one of the most constant and earliest of all the general symptoms. It is rarely absent but varies greatly in intensity. It is especially severe during the growth of the abscess, and usually slight during the period of latency. It is sometimes felt over the entire head, but is generally more severe on the side of the abscess. In Koerner's twenty-one cases of cerebellar abscess, the pain was located in the occipital region only eight times. Pain in the back of the head is sometimes observed in frontal abscesses. Headache is often accompanied by rigidity of the back of the neck, particularly in abscesses of the posterior fossa.

In the majority of cases, according to Macewen, the temperature is normal or even subnormal during the entire course. When the symptoms begin suddenly fever may be present, but it is probable that the febrile movement is partly due, in many cases, to the primary disease or to complications.

As a rule, the pulse is slow at the height of the disease. Toynbee and Wreden described cases in which it fell to 10–16 per minute. The slowness of the pulse may persist during an increase of the bodily temperature.

After a while the headache increases in severity, and the patient may be confined to bed on account of the general malaise. The irritability and excitement likewise increase, delirium supervenes, and epileptiform convulsions may make their appearance.

Then the delirium changes into hebetude, somnolence, and coma; the pulse becomes rapid and irregular. Death either ensues rapidly or recovery slowly occurs, and the patient is restored apparently to health, with the exception, perhaps, of a monoplegia or hemiplegia, or even without any local symptoms. He then enters upon the so-called latent stage. The symptoms described are very like those of acute meningitis, and, indeed, this lesion may be associated with the encephalitis from the beginning, or it may develop secondarily.

Sometimes the symptoms begin with great suddenness, and at once assume an alarming aspect.

In another series of cases the symptoms of concussion or compression of the brain, due to the original injury, continue without intermission or improvement, and are rapidly merged into those due to the developing encephalitis and abscess formation, so that it is impossible to tell when the one process ends and the other begins. Such cases usually run a more rapid course than the former variety.

Finally, not a few cases have been reported in which the abscess has remained entirely latent for a long period, without giving the least indication of its presence. In fact, the autopsy alone may reveal the existence of a hitherto unsuspected chronic abscess. In a case observed by Nauwerk, the period of latency lasted twenty-eight years.

Sometimes the sole symptom of the so-called latent stage has been the occurrence of epileptiform convulsions, so that the case has been regarded as one of ordinary epilepsy. In other cases, chills, followed by a hot stage and sweating, occur at irregular, or, in rare cases, perfectly regular intervals, so that a diagnosis of intermittent fever has been made. An instance of this kind has come under my own observation. Usually, however, such patients also suffer from violent headache, frequently localized in the vicinity of the abscess.

Abscesses of the frontal lobe are more apt than others to be unattended with symptoms during the stage of latency. In a case of this kind, observed by me in the person of a lunatic, the mental condition seemed to be somewhat improved during the period of latency (one month). The night before her death, she had what were supposed to be hysterical convulsions (irregular twisting movements without loss of consciousness), as the patient had exhibited other hysterical symptoms for a year (the injury which gave rise to the abscess was received a month before the onset of the convulsive seizures). On the following morning the patient was found dead in bed. The autopsy showed an abscess of the frontal lobe, which had burst (evidently shortly before death) through the convexity.

The clinical history of otitic abscess of the brain is modified by the symptomatology of the primary disease of the ear. The initial symptoms usually consist of pain in the ear, vomiting, vertigo, tinnitus, and rise of bodily temperature. Inasmuch as all these symptoms may be the result of simple retention of pus in the diseased ear, it may be impossible to determine for some time whether the brain has really become involved. The condition is still further obscured by the fact that in these cases we observe frequent complications with purulent meningitis, pachymeningitis, and thrombosis of the sinuses.

In the majority of cases the abscess runs an acute or subacute course.

Mental obtuseness generally becomes noticeable soon after the onset of the disease. The patient answers questions correctly but slowly, and, when the eyes are open, stares into vacancy. The somnolence may deepen quite rapidly into coma.

When the disease runs a chronic course, the history is essentially the same as that of the traumatic variety.

On account of their embolic origin metastatic abscesses usually develop suddenly. In the majority of cases the emboli enter the middle cerebral artery and are carried to terminal branches, so that we generally find evidences of an affection of the motor region of the brain. Pure Jacksonian epilepsy is not an uncommon feature of these cases. Paralytic symptoms are much more frequent. Various aphasic disturbances have also been observed. In a considerable proportion of cases the abscesses are multiple and the focal symptoms are thus obscured.

The brain symptoms are often associated with evidences of general infection, viz., fever, chills and sweats, and rapidly developing cachexia.

It is unnecessary to say that, if the abscess is situated in such a position that it either involves the cortex in some part which presides over a special function, or destroys the white matter in such a manner as to cut across the fibres leading to such portions of the cortex, the corresponding symptoms will be produced. Thus, hemianopsia, aphasia, word-deafness, paralysis of individual nerves (very rarely), monoplegia, or hemiplegia may be produced in this manner. As occurs likewise in the clinical history of tumors of the brain, hemiplegia results usually from a succession of monoplegias, due evidently to a gradual spread of the destructive process from one set of fibres to another. This fact may be of invaluable service in making a regional diagnosis.

But, unlike tumors of the brain, cerebral abscesses rarely give rise to paralysis of cerebral nerves. Choked disc is also much less frequent and less marked than in tumors. According to Oppenheim, choked disc is much less frequent than optic neuritis. The affection of the optic nerve is sometimes confined to one eye. In operative cases the optic disturbances may disappear gradually after the operation.

The frequent absence of choked disc may be accounted for, perhaps, by the fact that the encephalitis spreads by causing adjacent parts of the brain to become directly involved in the inflammatory process, and in this way less pressure is produced upon adjacent parts.

In a certain proportion of cases all "head symptoms" are entirely wanting during the so-called latent stage, and we are sometimes astounded at the autopsy at discovering the great apparent disproportion between the amount of brain tissue destroyed and the slight character of the symptoms which had been produced. This is especially striking if we compare the symptoms with those which usually result from cerebral hemorrhages, even when much smaller in size. It must be remembered, however, that the latter lesion is usually situated in such a position (internal capsule and surrounding parts) that

comparatively slight destruction of tissue interferes materially with the conduction of nervous impulses.

Vomiting is a not infrequent symptom, and may occur when the stomach is either full or empty.

In certain rare cases, the sole symptoms observed during the period of latency are those of mental derangement, and the patient is supposed to suffer from insanity as the result of injury to the head. In a case recently under our observation, such an error in diagnosis was made by a very eminent neurologist, and the true nature of the case was revealed only at the autopsy, as the psychical symptoms persisted, uncomplicated with any other manifestations, until the terminal meningitis set in.

As a general thing, the latent period is brought to a close by a sudden irruption of symptoms, beginning either with an epileptiform convulsion or a series of convulsions, with great exacerbation of the headache, mental irritability and delirium, or with sudden coma. The symptoms which follow are very similar in character to those which we have described above as occurring in acute abscess. After a short period (varying usually from a few hours to several days), the symptoms either terminate fatally (this is the rule) or they gradually clear up. But sooner or later, after another period of latency, in which the symptoms are more violent than in the first period, a second outburst occurs, which terminates fatally.

From the reports of a few isolated cases it seems probable that chronic abscesses may undergo spontaneous recovery, inasmuch as the pus becomes cheesy or even calcareous. But such an event, if it happens at all, must be extremely rare.

Diagnosis.—In making a diagnosis of abscess of the brain great importance attaches to etiological considerations. We are rarely justified in making the diagnosis unless we obtain a history of a previous injury to the head, of a purulent disease of the ear, nose, or other adjacent parts, or of some source of infectious emboli in distant parts of the body, notably in the lungs.

Injury to the head is followed, in rare cases, by tumor of the brain. But in such cases it will usually be found that the primary wound was not infectious in character, and, moreover, the symptoms follow one another in a steadily progressive manner. Furthermore, choked disc is much more constant and pronounced in tumor cerebri, while the subnormal temperature, or the fever and chills of abscess are wanting.

Injury to the head may also give rise to other symptoms which simulate abscess of the brain but which are in reality due to hemorrhagic encephalitis. In the latter affection the symptoms begin suddenly. They may terminate in complete recovery or rapid death, or the majority of the symptoms may disappear and leave permanent focal symptoms.

In considering suppurative disease of the middle ear as a factor in the diagnosis of cerebral abscess, it should not be forgotten that severe cerebral symptoms may be produced by the mere retention of pus in otitis media independently of the existence of cerebral lesions. A number of cases have also been reported in which optic neuritis or choked disc was associated with simple otitis media. This peculiar combination has been explained in various ways, viz., as the result of thrombosis of the sinuses, of infection of the carotid canal and extension along its lymph channels to the sheath of the optic nerve, and of serous meningitis.

Extra-dural abscess often cannot be distinguished from cerebral abscess; indeed, in many cases the two lesions are combined. According to Jansen, extra-dural abscesses exhibit the following characteristic features: (*a*) thickening of the bone, subperiosteal abscess or œdematous swelling behind the mastoid process; (*b*) pain on pressure and percussion in this region; (*c*) impaired mobility of the head, particularly around the sagittal axis; (*d*) nystagmus of both eyes, on voluntary motion, chiefly toward the side of the healthy ear.

Otitic thrombosis of the sinuses is also difficult of differentiation. In the majority of cases there is considerable intermittent fever, and the pulse is irregular and rapid. Many cases are attended with chills, sweats, and profuse diarrhœa. In some cases all these symptoms may be wanting. In thrombosis of the cavernous sinus we find œdema of the eyelids and surrounding parts, and protrusion of the eyeball. In thrombosis of the transverse sinus, certain symptoms may be due to implication of the upper part of the jugular vein. This vein is tender on pressure and may be felt occasionally as a hard cord. Small abscesses may develop in surrounding parts. Œdema behind the mastoid process is very frequent.

Focal symptoms are usually wanting in thrombosis of the sinuses.

When abscess and thrombosis are combined the condition becomes still more difficult of diagnosis.

It is also difficult in many cases to differentiate meningitis from abscess. The former disease usually runs a more rapid course than abscess, is attended with considerable fever and acceleration of the pulse, general convulsions are frequent, and there is also hyperæsthesia of the entire skin in many cases. Rigidity of the neck is a common symptom, together with retraction of the abdomen and increase of the tendon reflexes. In children all these symptoms are sometimes produced by retention of pus in the middle ear and may be relieved by evacuation of the pus.

The regional diagnosis must be made according to the principles laid down in the articles on *Diagnosis of Local Lesions* and on *Functions of the Cerebral Cortex* (both in the present series of brain articles).

Prognosis.—The prognosis of this affection has been modified very materially in the last fifteen years by the progress in antiseptic surgery. Spontaneous recovery by caseation or calcification of the abscess or by rupture externally is extremely rare. But operative interference now promises a good measure of success. Oppenheim found that in 53 cases of operation upon traumatic cerebral abscess 36 terminated in recovery. Koerner collated 92 operative cases of otitic cerebral abscesses; 51 cases recovered and death occurred in 41 cases. Macewen's results were remarkably favorable. Among his 25 cases of cerebral abscess he operated upon 19, and of these 18 recovered.

The poorest chances of successful surgical interference are presented by metastatic abscesses in which there is general pyæmic infection. Complication with thrombosis of the sinuses or meningitis also makes the prognosis more unfavorable.

Treatment.—Medical treatment of this affection, apart from meeting the individual symptoms as they arise, is utterly useless, as we possess no remedy which will cause the absorption of the pus after it has once formed.

Prophylactic measures are useful, perhaps, in preventing encephalitis after injuries to the head. The chief measures are absolute mental and bodily rest (the patient kept on his back, not allowed to sit up, to read, or to talk with those around him), which should be continued, if the injury has been violent enough to produce unconsciousness, for at least a week or two after the subsidence of the cerebral symptoms. In addition, the bowels should be kept thoroughly open by some mild saline, or by small doses of calomel.

Prophylaxis may also prove of benefit in diseases of the ear and nose. Free vent to the pus should always be secured, and paracentesis performed as soon as retention of pus develops.

As soon as the diagnosis is assured, operative interference is indicated. In some cases this has proved successful, even when the patient was in a moribund condition or a complication with purulent meningitis had developed. In some instances repeated operations have been necessary, either because the abscess refilled or because fresh abscesses developed in the immediate vicinity of the primary one.

Leopold Putzel.

BRAIN, ANÆMIA OF.—Anæmia is a condition and not a disease in the proper sense of the term. Moreover, it is a condition which rarely or never exists uncomplicated. The causes which produce it are very apt to produce independently irritation and inflammation, or the same conditions which at first bring about an anæmia of the brain may, if their action be longer continued, excite in its place a cerebral hyperæmia. In the present state of our knowledge it is, moreover, often impossible for us to prove whether certain symptoms are really produced by an anæmia or by a hyperæmia of the brain, and in such cases we are forced to come to a decision on very imperfect grounds.

Of the fact that the anæmia in itself acts as an irritant on the nerve centres there can be no doubt, but its chief effect on them is produced through the deprivation of nutrition which it causes. In considering anæmia of the brain, it is not possible for us to differentiate the symptoms caused by deprivation of nutrition to the cerebral tissues through loss of blood and those caused by deprivation of nutrition to the same parts on account of the poverty of the blood in nutritive material suited to their needs. Hence the term cerebral anæmia has come to denote not only that condition in which there is an absolute diminution of the amount of blood in the brain, but also all those, so long as no toxic element is involved, in which the blood supplied to the brain is, from its composition, incapable of affording due and sufficient nourishment to the tissues. As a change in the condition of the blood is in most cases concurrent with a diminution in its amount, we may fairly say that cerebral anæmia, when universal, is usually "a complex condition, depending not merely upon a deficiency of the quantity of blood supplied to the brain, but also upon a change in its quality, and upon a diminution in the intracranial pressure" (Ross, ii., 611).

Anæmia of the brain may be either universal or partial, according as the whole or a portion only of the organ is involved. Partial anæmia, except when caused by occlusion of a blood-vessel through pressure, thrombosis, or embolism, is rare, and its symptoms are often not recognizable during life.

We shall hence consider here only universal cerebral anæmia. This may be either acute or chronic, and it may be due to a condition confined to the brain alone or may form part of a general anæmia.

Acute universal anæmia of the brain is always the result of the sudden withdrawal of a large quantity of blood from that organ. It occurs typically in ligature of the large arteries in the neck, the innominate, the carotids, or the vertebrals. These operations are performed usually either on account of injury, or for some tumor or aneurism. The vertebrals were formerly sometimes tied as a cure for epilepsy.

1. The most common cause of acute cerebral anæmia is, however, undoubtedly hemorrhage. This may occur from any part of the body, provided only that sufficient blood be lost with sufficient rapidity. Putting aside hemorrhages due to injuries, the more common forms are metrorrhagia, especially post partum, epistaxis, hæmoptysis, and hemorrhages from the stomach and intestines; occasionally also the rupture of aneurisms.

In cases of hemorrhage from injury the influence of shock must always be taken into account, as there are few cases of severe injury in which it does not exist to a greater or less degree. The pathological condition existent in shock is as yet unknown. One hypothesis is that maintained by Gröningen, that the complex of symptoms known by this name is due to an exhaustion of the medulla oblongata and of the spinal cord, produced by violent and severe drains upon their strength. Other writers consider shock as due to sudden changes in the calibre of the blood-vessels. Thus it is defined by Fischer as a reflex paralysis of the vaso-motor nerves, especially of the splanchnic, produced through a traumatic concussion. However this may be, cerebral anæmia is certainly produced by shock, and, as Travers says of shock and fainting: "They differ in degree and duration more than in kind."

2. Acute cerebral anæmia may be produced by any sudden change in the distribution of the blood in the body at large. Any cause which suddenly attracts a large quantity of blood to one portion of the body will naturally reduce the amount which can go to the other parts, and hence will induce an anæmia in them. This sudden change in the general distribution of blood in the body is said to occur after violent labor, when, the uterine vessels being suddenly released from pressure, large quantities of blood enter them freely and are thus withdrawn from the rest of the body. To this cause are probably in part also due the serious symptoms which sometimes occur after the withdrawal of large quantities of fluid from the pleural or peritoneal cavities. Another example of this form of disturbance is given in the action of Junod's boot, which, if carelessly used, may provoke dangerous symptoms.

3. A third cause of this form of anæmia is want of energy in the action of the heart. This is a common cause of chronic anæmia, but the acute form may readily be produced by any sudden demand for increased exertion on the already weakened heart. This is readily seen in anæmic persons and those suffering from insufficient action of the heart, in their liability to faint on any slight exertion, or even on rapid change of position. More especially is this the case in convalescents from acute febrile diseases, and it is particularly apt to occur after acute pneumonia. This frequently takes place when the patient rises suddenly from the horizontal position, the change in the distribution of the blood, which under normal circumstances would not be perceived, making itself felt.

In organic diseases of the heart cerebral anæmia in the acute form frequently occurs, more especially in aortic regurgitation, in which death from syncope is not uncommon. Persons suffering from myocarditis or from fatty degeneration of the heart are likewise peculiarly liable to attacks of fainting.

Anæmia of the brain may in like manner be produced by irritation of the vagus nerve through its action on the heart. Brown-Séquard and others have found that crushing of the right semilunar ganglion causes stoppage of the heart, and the deaths which sometimes occur from syncope in nervous and delicate persons, who are suffering from hepatic colic, may perhaps be referred to some similar cause. We may also mention here, though without any special reference to the method of their causation, those cases of syncope and collapse which occur in perforation of the stomach or intestines, and in which death is not infrequent. Hill believes that "the cerebral circulation is controlled by the vaso-motor centre acting on the splanchnic area."

That intestinal disturbances even of a light character are especially liable to produce syncope is well known. Even a transient abdominal pain or a slight attack of intestinal colic frequently causes the symptoms of faintness or even actual syncope. Syncopal cardialgia and a tendency to faint are said to be especially common in those suffering from the dyspepsia of gout.

4. Again, a diminution of the amount of blood in the brain sufficient to produce the symptoms of cerebral anæmia might be caused through the spasmodic contraction of the cerebral arteries. This is supposed to occur, when, from emotion or mental excitement, there is pallor of the countenance, and even loss of consciousness, without any failure of the action of the heart. The not uncommon occurrence of fainting at the sight of surgical operations, or at the sight of blood, would come under this head. Syncope, and even sudden death, may be caused by the sudden advent of any strong emotion—surprise, terror, grief, or joy. How far shock or the irritation of the vagus comes into play in these cases is yet undecided.

Nothnagel considers it possible that in attacks of epilepsy we have to deal with a spasmodic contraction of the cerebral blood-vessels, due to the irritation of a cere-