

about the extravasation is caused by a bloody infiltration of the tissues at the edge of the hæmatoma. Consequently the finger can make an impression in the ridge, in some cases even flatten it completely here and there. A feeling of very delicate crepitation, due to the friction of the blood coagula in the tissue spaces, may be obtained. No hesitation need be felt about making this examination, as the patient suffers no harm by it.

If a sabre wound extends along a markedly curved segment of the skull, and if the wound is long, we may generally suspect that at least the centre of the wound communicates with the cranial cavity. This assumption has more or less of a geometrical basis.

If, in an incised wound of the scalp, the edges of the wound do not gape, the injury has been confined to the skin; if the edges separate, the galea has been severed. Anatomical facts explain this, for the aponeurosis is drawn upon by its muscles, and thus separated. As the skin is closely connected with it, the wound gapes.

If a hæmatoma, or swelling, appears at a point opposite to the point of entrance of a shot, the projectile may be situated under this spot. Larrey found a bullet by cutting down upon the suggillation.

If the unharmed dura, exposed by some defect in the skull bones, shows no brain pulsation, one of several conditions may be present. The part of the brain situated beneath this point may be anæmic, or contused; or there may be an extravasation of blood under the dura. The anæmia may be caused by a splinter of bone wedged in between the dura and the skull. Contusion interferes with pulsation, because the contused parts have their circulation disturbed through destruction of the vessels. A blood extravasation situated beneath the dura will impart a bluish colour to the affected area.

### CHAPTER III

#### INFLAMMATORY COMPLICATIONS FOLLOWING INJURIES TO THE SKULL

IN many cases, carefully executed antisepsis will determine whether a penetrating injury to the skull will be followed by suppurative inflammation of the intracranial contents. Frequently, however, reliable antisepsis is out of the question. For instance, in fractures of the base, which involve the tympanum or nasopharynx, and consequently tear the mucous membrane, the pyogenic cocci find a point of entrance in spite of all possible precautions.

The most frequent complications to be considered are meningitis, brain abscess, and phlebitis of the sinuses. Other causes in surgical practice may, however, occasion these diseases. Thus, tuberculosis of the petrous bone or a suppurative otitis media may readily lead to an abscess in the neighbouring parts of the brain, or to inflammation of the meninges. A phosphorus necrosis may spread to the sphenoid bone and cause a meningitis. Erysipelas of the scalp may travel along the emissary veins and give rise to purulent meningitis, often with sinus thrombosis. Analogous complications may arise from a furuncle of the face. Following a resection of a cranial nerve infection has been known to travel along the nerve-sheath and enter the skull. I



even saw a case of double sinus thrombosis of the cavernous sinus with fatal meningitis, shown by autopsy to have been due to an infection which had as its starting-point a carious tooth.

*Abscess of the Brain* demands our greatest attention in the study of the three chief varieties of secondary inflammations found in the interior of the skull—meningitis, abscess, and sinus thrombosis—because opening the abscess is a life-saving operation. It is true that many false diagnoses are made, and many purposeless trephining have been done; but in the last few years successful cases have considerably increased in number because diagnosis has become more certain. Brain abscesses due to a foreign body have most frequently been opened, but here the foreign body served as a guide to the surgeon. If the operator discovers the foreign body which has become wedged in the skull bone, and, upon removing it, finds pus flowing out of the wound, he deserves credit only for finding and removing the foreign body, or at the utmost for enlarging the wound in the bone.

We intend to discuss chiefly the chronic brain abscess, which anatomically is characterized by an inclosing pyogenic membrane, and which is embedded in the brain substance just as a cyst would be. In recognition of the pyogenic membrane it is known as an encysted abscess of the brain. It is most frequently due to traumata which have caused some foreign body to penetrate and remain fixed in the brain. Next in frequency it is due to suppurative foci in the petrous bone. Third in frequency these abscesses are the result of metastases, and with greatest relative frequency of metastasis caused by endocarditis or by a

septic process in the lung. Operative interference is naturally confined to the first two varieties.

What are the symptoms of a brain abscess? By *a priori* reasoning we would, in the first place, expect the destruction of a part of the brain to be followed by circumscribed paralysis, just as is the case in the destruction caused by apoplexy, and this is quite true. Without the symptoms of some diseased focus in the brain (paralysis with or without convulsions) a positive diagnosis of brain abscess is impossible. But, unfortunately, softening of the brain following a contusion of the brain substance gives the same symptoms. Experience has also shown that abscesses of considerable size may exist for months, or even years, without the patients suffering the least disturbance of function. Only at the very last, when the abscess, which has been embedded in the brain substance, bursts into the ventricle or through the pia, terminal symptoms rapidly develop. In the second place we would anticipate *a priori* that suppuration in the brain would cause pressure symptoms through the tension existing in the focus; but this is, again, not the case.

The preceding remarks, showing, as they do, that patients with brain abscess may seem perfectly well, disprove it. It is true that a slight increase in pressure (hyperæmia) may cause pressure symptoms to appear without warning. These manifest themselves as dizziness, a tendency to vomiting, and eventually produce stupor, which may again disappear as the pressure declines.

A chronic or encysted abscess may be suspected by its course. An injury to the head, accompanied by slight primary cerebral symptoms, is succeeded by a



period of undisturbed health which may last for months or years. This is then followed by intermittent or continuous periods of headache, which constitute a particularly valuable symptom if they originate at the site of the injury. The patient suffers from dizziness and occasional attacks of vomiting, especially upon making some sudden movement of the head. Convulsions occur periodically, and evening attacks of fever are the rule. The abscess may be diagnosed, even at this time, if pus discharges from a small opening in the skull close to where a foreign body entered. Most likely a foreign body or a necrotic splinter of bone can be removed, the discharge of pus follows, and the probe passes down into the depths. On enlarging the opening in the bone we come upon a small slit in the dura, which, when dilated, gives vent to a large quantity of pus.

Or severe complications arise: severe attacks of fever and convulsions of the most diverse kinds; the headache becomes terrible; the patient grows delirious, and paralyses are noticed. Especially when hemiplegia on the side opposite to the injury occurs, the probability of a brain abscess is great if the general course of the disease has followed the type above described. If, relying upon the diagnosis, the abscess is opened by trephining, all these symptoms may promptly disappear; but if the disease is allowed to run its own course, high fever and convulsions are followed by stupor, œdema of the lungs, and death.

An especially doubtful picture is presented by the so-called *pachymeningitis suppuratoria externa circumscripta*. Heinecke relates a case in which no extension of the inflammation to the sinuses and to the pia took place. A blow upon the head was followed by fever and headache. A swelling, which pointed and discharged pus, formed.

A small opening in the bone admitted into the interior of the skull. On enlarging the opening with a chisel an abscess cavity between dura and bone was reached. There is no doubt that this was a case of osteomyelitis and necrosis of the skull bone, which had been bathed by pus on both its surfaces. But if any symptoms of meningitis had existed at the time of opening, we would have expected to find an abscess of the brain; only the fact that the dura was uninjured made this diagnosis improbable. Occasionally an idiopathic infectious osteomyelitis of the bones of the skull occurs and runs an analogous course.

Brain abscesses, secondary to suppurative otitis media, result either from direct extension from the bone to the meninges, or through metastatic progression of the cocci into the deeper parts, with an apparently healthy layer of brain substance between the ear and the abscess. Such abscesses, following disease of the middle ear, are legitimate objects for surgical interference, and in more recent years many successful trephinings have been accomplished.

Clinical experience has shown that suppuration starting at the tegmen tympani or in the anterior cells of the mastoid process, causes abscess in the temporal lobe, while suppuration of the posterior wall of the middle ear, or of the posterior mastoid cells, causes abscess of the cerebellum.

*Sinus Phlebitis* is much more readily diagnosed than brain abscess. Even if the general picture resembles meningitis in some of its details, we have three means of differentiation:

In the first place the etiological factor is of assistance. If, for instance, we are able to see phlebitis of a superficial vein during the course of a suppuration in the face or scalp, and this is succeeded by symptoms of meningitis, we are justified in concluding that the in-



flammation has travelled along the natural channels until it has reached the sinuses. In the second place, certain anatomical relations between the sinuses, nerves, and veins produce definite symptoms which point to a particular sinus. In the third place, in sinus phlebitis certain symptoms not present in meningitis occur; such are atypical recurring chills, jaundice, metastatic pneumonia or pleurisy, suppuration in the joints, etc., all of which are symptoms of pyæmia. They are due to direct infection of the blood by way of the sinus.

There remain to be mentioned only those peripheral symptoms which permit the localization of a phlebitis to some particular sinus. As the oculomotor, the trochlear, and the first division of the fifth lie on the side of, and the abducens traverses, the cavernous sinus, inflammation of this blood channel causes the following symptoms referable to these nerves: Pain in the frontal and supra-orbital region, ptosis of the upper lid, strabismus, cloudy or sometimes softened cornea. As the veins of the eye pour their blood into the cavernous sinus, œdema of the lids and of the subcutaneous tissue results from the arrest of circulation; also exophthalmos and immobility of the eyeball. If these symptoms appear in the opposite eye, the process has extended from the sinus of the one side to that of the other. If the phlebitis occurs in the domain of the transverse sinus the inflammation may spread directly into the upper bulb of the jugular vein. The nerves which pass through the jugular foramen are then pressed upon and paralyzed by the exudate and the swelling of the surrounding parts. This may cause a marked increase in the rapidity of the pulse, or fluctuations of the pulse-rate, aphonia, inability to swallow or cough—in short, to marked

phenomena in the distribution of the vagus, the spinal accessory and glosso-pharyngeal nerves. An additional manifestation of utmost importance is the occurrence of convulsions. If the inflammation spreads farther along the course of the internal jugular, the side of the neck becomes swollen, the external jugular prominent, and the swollen parts grow extremely tender and painful. Inflammation of the superior longitudinal sinus produces epistaxis and general epileptiform convulsions, with headache at the vertex. In some acute cases the progress of the disease from one sinus to another can be followed by means of these landmarks.

*Sinus Thrombosis* is distinctly different from sinus phlebitis. As, however, most cases of sinus thrombosis terminate in a sinus phlebitis as a result of the disintegration of the thrombus, it becomes a question of diagnostic interest whether the thrombosis can be recognised before the secondary inflammation has started. In isolated cases it can be suspected when the external symptoms of an arrest of the intracranial circulation occur, such as increased prominence of the eyeball with œdema of the lids and injection of the conjunctiva, the presence of unequally distended external jugular veins, etc. We may also suspect a thrombosis if cerebral symptoms are noticed—vomiting, headache, and delirium—which develop rapidly and then diminish, though they may recur with equal rapidity. Finally, in disease of non-inflammatory origin, such as marasmus (marantic thrombosis) or tumour, these symptoms will lead us to believe that the sinus has been compressed or its lumen encroached upon.

The symptoms of *Meningitis* due to trauma or to the extension of inflammation from neighbouring parts dif-



fer in no way from those of meningitis due to other causes.

For surgical reasons meningitis of the hemispheres is distinguished from that of the base, so that we speak of *meningitis of the convexity* and *basilar meningitis*. Even if the points of differentiation known to us are not absolutely trustworthy, their discussion is very suggestive, and may lead to further increase in our knowledge. The cases which come under the observation of the surgeon are clear, at least in one respect, as the local point of origin of the process is usually known (scalp wound, caries, or necrosis of the skull). This shows us, at least approximately, how the anatomical progress of the disease is marked by corresponding symptoms.

In all cases of meningitis we distinguish a stage of irritation and a stage of depression.

*The stage of irritation* is marked by psychical excitement, increase in pulse-rate and temperature, convulsions, contractions, hyperæsthesia, and headache.

In the *stage of depression* excitement is succeeded by unconsciousness; among the motor phenomena paralyses become the prominent symptom; even the reflexes are abolished. Micturition and defecation are involuntary; the fever persists, but the pulse is slowed, while respiration grows stertorous. Only toward the very end respiration again becomes rapid and the pulse more and more frequent.

It is necessary to bear in mind that symptoms of irritation (like convulsions) may be combined with symptoms of depression (such as paralysis), and further, that those groups of muscles which were at first irritated are not necessarily the ones which later grow paralyzed. The affair is not quite as schematic as this. Contraction,

tures, convulsions, and paralyses may occur together; for instance, ptosis of one lid, strabismus, and at the same time clonic spasms of an extremity. Groups of muscles which previously were not affected by convulsive twitchings may be paralyzed. The division into two stages is founded upon the general picture presented by the patient. In the first stage he is excited and delirious; in the second, comatose and stuporous.

*Meningitis of the convexity* is distinguished by the occurrence of hemiplegia, or at least of hemiparesis. This is explained by the assumption that the inflammation of the meninges renders the immediately subjacent motor cortex incapable of performing its functions. Bergmann has employed the following analogy: He believes that the process is similar to that seen in peritonitis, where an inflammation of the visceral peritoneum is followed by a paralysis of the underlying muscular coats. Basal meningitis runs its course without hemiplegia.

All in all, we must remember that meningitis is a disease which shows itself in a great variety of forms. As it may appear primarily (in the first few days following a trauma), or also secondarily (in the second or third week), after an injury to the skull, we must be on the watch for it, and give a very guarded prognosis.

If it is noticed that the injured person evinces a marked restlessness, that he tears the bandages from his head, grinds his teeth, spits, and, on being questioned, complains of headache, these symptoms must not be regarded lightly. If the temperature now rises more rapidly, our anxiety is increased. If vomiting sets in, the patient on the next day will show ptosis or facial paralysis, or the attendants report that the patient has



had convulsions. This shows that meningitis is already present.

In certain cases we may be deceived at the beginning of the period of irritation. For instance, following a case of fracture at the base, with severe concussion, the excitement of the patient may indicate the beginning of a primary meningitis, or perhaps no more than the period of excitement following upon a severe concussion. In the latter case, suitable treatment (rest, cold, purgation, or, if the pulse is hard and slow, venesection) will soon cause the disappearance of the symptoms.

The following case was seen by me at the beginning of my surgical career: During my period of service under Lorinser there was a woman about fifty years of age in the ward. She was suffering from tuberculosis and caries of the calcaneum. Suddenly, in the course of one night she became hemiplegic. Lorinser, making his rounds, saw her only in passing on three successive days, and merely stopped to feel her pulse and to have her show her tongue. On the third day the patient died. When asked what cause of death we should put on the death certificate, he answered, "Meningitis." We were surprised when, on autopsy, purulent meningitis was really found; and were eager to discover how Lorinser was able to diagnose the case with such superficial observation. The answer which we received has always remained fixed in my mind: "As the patient was not suffering from any circulatory disease, but from caries and tuberculosis, apoplexy was unlikely. The sudden onset of hemiplegia was indeed striking, but this continually increased in severity. For on the first day the patient's tongue was protruded to one side; on the second, much more so, and on the third, even more. This pointed to a slowly progressing process, in other words, to an inflammatory affection. On the third day the patient's pulse grew slower, in spite of the increase in the paralysis, and in spite of her having suffered for weeks with fever. If you had watched the patient more carefully you surely would have discovered other symptoms of meningitis." We really discovered, upon questioning the patient in the adjoining bed, that the deceased had suffered from convulsions during the night, which the nurse had failed to report.

## CHAPTER IV

### TUMOURS OF THE SKULL

A SERIES of very interesting tumours are found on the skull.

The foremost in importance is *Hernia Cerebri*. Just as in all other herniæ, we distinguish hernial orifice, hernial sac, and hernial contents.

The point of exit is formed by the non-approximation of two or more skull bones. At this spot a gap exists, and the name given to each hernia depends upon the bones which have failed to come together and have thus given rise to the hernial orifice, such as a naso-ethmoidal or sphenoidal hernia, etc.

The *hernial sac* is formed by the dura mater.

The *hernial contents* varies in character.

(a) It may consist of a saclike protrusion of the arachnoid filled with cerebro-spinal fluid derived from the subarachnoid space—*Meningocele*.

(b) Of a part of the hemisphere, into which, however, the ventricle is prolonged—*Cenencephaloccele*.

(c) A more involved hernia, in which, owing to disappearance of the brain substance, the ventricle opens directly into the hernial sac.

The coverings of a hernia cerebri are composed of the meninges. In herniæ of small size their aspect may be normal; in larger ones they are, as a rule,