I was substituting for my teacher, v. Dumreicher, in Vienna, when I was informed that a child, urgently requiring tracheotomy, was about to be carried in. I immediately interrupted the lecture, and the child was introduced. Its breathing really indicated a high degree of stenosis of the air-passages. The most natural thing to think of was croup. But the child did not show the retraction of the head usually seen in croup. On the contrary, the head was bent forward and kept in this position without the slightest movement. This circumstance, combined with the pale looks of the child, caused me to suspect, at the first glance, that the dyspnœa might be due to caries of the cervical spine, with retropharyngeal abscess. Without delay I palpated the neck, and discovered a prominent spinous process. I now passed the fingers of my left hand into the child's mouth, and found an elastic bulging of the posterior wall of the pharynx. Introducing a knife, I opened an abscess, the whole incident occupying only a few seconds. The dyspnœa rapidly diminished, and I now had leisure to explain my actions to the students.

CHAPTER II

INJURIES TO THE SKULL AND BRAIN, AND THEIR PRIMARY SEQUELÆ

THAT the final outcome of injuries of the skull can never be predicted with any certainty has been known from the earliest times, and is a fact which still holds good. Severe penetrating wounds, complicated by crushing of large areas of brain substance, may heal without leaving any after-effects or any disturbance of the bodily functions. On the other hand, a blow on the head, which does not cause the victim to fall to the ground but which produces a small wound of the soft parts, may rapidly lead to erysipelas, meningitis, and death. In recent times antisepsis has proved a blessing in these cases, when applied at the right time, by preventing the appearance of suppuration. I have made these preliminary remarks in order to indicate in advance that in wounds of the skull—just as in wounds of the thorax and abdomen—antiseptic measures must precede all attempts at arriving at a more accurate diagnosis.

Injuries to the skull may be divided into:

1. Injuries of the soft parts.
2. Injuries of the bones of the cranium.
3. Injuries of the intracranial contents (brain, meninges, blood-vessels, nerve-trunks).

These injuries may be subcutaneous or open; the latter are known as wounds.

Accordingly, we distinguish wounds of the soft parts covering the skull, of the skull bones, and of the brain and its meninges. According to the weapon inflicting the injury, the wounds are divided into punctured, incised, tearing, lacerated, gunshot, etc. Such injuries may be accompanied by considerable loss of substance, the most striking example being the "lopping off" of a part known among the ancients as Αποσκεπαρνισμος. In cavalry encounters, a sharp sword, or a Damascus blade, occasionally lops off part of the skull; for instance, if the blow strikes the parietal eminence, a flap, consisting of soft parts, bone, and brain, may be entirely severed.

Subcutaneous injuries are represented by bruises of the soft parts, hæmatomata, fractures of the skull, bruises and rupture of the brain-matter, rupture of the internal blood-vessels (meningeal or cerebral hemorrhage), and of the nerve-trunks situated within the skull. To these we may add two peculiar varieties of injury—*compression* and *concussion* of the brain.

The whole attention of the physician who is treating an injury of the skull is first directed to determine whether the brain and its membranes have escaped. If the wound is compound, direct examination by sight, and eventually by the probe and finger, are undertaken as a matter of routine. If no wound is present, if the injury is subcutaneous, the involvement of the brain can be diagnosed only by means of certain functional disturbances.

It is best, therefore, to begin at once with one of

these difficult cases, and to discuss by what signs an injury to the brain can be recognised.

Bruises, tears, pulpification, or breaks in continuity are always *local* lesions, no matter whether simple, and then due to the splinters of a depressed fracture, etc., or compound, as the result of an open wound. They affect only a circumscribed portion of the organ, the cortex, as a rule, being the part involved.

In compression and concussion the whole brain takes part. These two forms represent *diffuse* lesions.

As the brain functions are localized in discrete, circumscribed parts of the whole brain, theoretically, at least, we may hope to recognise the destruction of any part by the disturbance of those functions which are regulated by it. Unfortunately, however, our knowledge of the separate centres is yet imperfect. Consequently, Local Lesions of the Brain can be diagnosed only in a limited number of cases by means of the resulting disturbances of function.

A few instances will not be out of place. If a soldier receives a bullet wound in the side of his head, and then grows hemiplegic on the opposite side of the body, a destruction of the cortical motor areas may be diagnosed. If the patient, after trauma, suffers from aphasia, an injury to the left hemisphere, embracing the part known as Broca's speech centre, may be assumed to exist. In paralysis of the facial nerve, its centre has been injured, unless the position of the wound indicates that the trunk, and not the centre, has suffered.

Paralysis, however, is not the only evidence of a local lesion, though it shows that a part of the central nervous system has been destroyed, or has been rendered inactive. Some portions of the central nervous

system can be injured or acted upon in such a fashion that they respond to this stimulus by irritative symptoms. A splinter of bone can penetrate and destroy one centre, but at the same time merely irritate an adjoining centre. The reaction following an injury can also produce symptoms of irritation in the neighbouring parts.

The deductions drawn from these facts are, that circumscribed paralysis, or circumscribed symptoms of irritation, but especially a combination of the two, point to a *local* lesion.

Circumscribed paralysis—i. e., paralysis limited to one group of muscles—is known as *Monoplegia*; circumscribed spasms are called *Monospasms*. If the spasms spread from the primarily affected group to other groups of muscles, the spasm which originated the phenomenon is known as the *Protospasm*.

The following combinations may be taken as types representing various local brain lesions:

Aphasia, with facial paralysis and paralysis of the upper extremity; proto- and monospasms of the facial; hemiplegia; hemiplegia, with spasm of the affected limbs; aphasia, with protospasm of the upper extremity followed by hemiplegia.

These various combinations can be explained by bearing in mind that the centres governing the extremities are contiguous, and that the centre for the facial and the centre for speech are situated in close proximity.

Spasms are the result of irritation, and this irritation soon leads to exhaustion of the centre; or a secondary inflammation may destroy the centre, which at first was merely irritated. This explains why paralysis

of the affected group of muscles may follow protospasms.

Of the diffuse brain disturbances, CONCUSSION is by far the most frequent. As the name indicates, concussion represents the rapid molecular displacement of the brain substance. The causes are a blow or fall on the head.

If we examine a case of concussion of medium severity, the following striking combination of symptoms are noticed:

1. Immediately after the trauma the patient falls to the ground and lies *unconscious* for a period varying from five to fifteen minutes. He has no recollection of the accident (amnesia).

2. Immediately, or on recovering consciousness, *vomiting* sets in, and may continue at intervals for several hours.

3. The pulse is *slowed*, the rate per minute being 60, 56, or 48 beats, or thereabouts.

In these cases respiration also is somewhat slower, the surface temperature subnormal. When the patient wakes up he is but partly conscious, and remains somnolent, torpid, and without speech or motion.

Minor degrees of concussion evince themselves by a *dazed* or *stunned* condition following the injury. Hearing, sight, and cerebration are inhibited for the time being. The patient falls to the ground, but awakes in a short time from his unconscious condition. For several hours slight dizziness, tinnitus aurium, muscular weakness, and headache remain. Recovery takes place within one or two days without treatment.

High degrees of concussion of the brain present a very grave picture. Unconsciousness may last for

hours, the patient lying as if dead. The physician finds him still comatose, with muscles wholly paralyzed and not responding to stimuli; or the condition may be one of stupor, with but momentary returns to consciousness. The body is pale and cool, respiration weak and superficial, the pulse small, irregular, and usually slow. Reaction, as a rule, follows, the body becoming warm, respiration deeper, pulse fuller, and consciousness and ability to move return. Instead of the pallor noticed in the first stage, the patient now appears flushed. One symptom may remain prominent for several days. This is the *slowing of the pulse-rate*, which may drop to 40 a minute, and yet the pulse continue to be full and of high tension. When the pulse returns to the normal rate the remaining symptoms—especially the headache, tinnitus, drowsiness, and slowness of speech—disappear.

The characteristic symptoms of concussion of the brain are evidently a *transient condition of depression*. If irritative symptoms arise, or the depression grows more profound, the condition no longer is one of uncomplicated concussion. Some other brain lesion then exists.

One point requires emphasis. Loss of consciousness, no matter how transitory, must have been present if the condition is to be regarded as concussion. This, therefore, is the most distinctive sign, while slowing of the pulse is the objective symptom.

As a rule, fracture of the skull is accompanied by concussion of the brain, for a force of sufficient violence to fracture the skull will suffice to cause concussion. There are cases, however, in which the force is concentrated upon so small an area of the surface of the skull that the bone is broken, but the brain escapes general concussion. This point is of importance from a medico-legal standpoint.

Compression of the brain (not to be confused with the normal brain pressure, which is constantly present) is caused by some new factor which increases the intracranial contents. The simplest example of this is an intracranial hemorrhage, as, for instance, rupture of the middle meningeal artery. The escaping blood is added to the already present cranial contents (brain and cerebro-spinal fluid). As the cranial cavity is non-elastic, the brain is compressed by the superadded blood. A similar example is furnished by a circumscribed depressed fracture of the skull. Here the intracranial contents is not increased by a new factor, but the cranial capacity is diminished, and becomes too small for its normal contents. Relatively the two cases are the same.

As a result of compression the following symptoms arise: Nystagmus, vomiting, circulatory disturbance (slowing of the pulse-rate), disturbance of respiration, general convulsions, coma, and death.

These, however, are the symptoms experimentally obtained on animals.

The picture at the sick-bed may follow one of two courses:

By compressing the tumour in an individual suffering from hernia cerebri, and thus increasing the contents of the cranium and raising intracranial pressure, the following symptoms result: *At times*, besides a few convulsive movements, the patient sinks into stupor and the pulse-rate is very much decreased.

After rupture of the middle meningeal artery, the patient first suffers from paralysis of the extremities of the opposite side. The paralysis then becomes general, consciousness is lost, and the pulse is slowed.

Both these clinical pictures have in common the loss of voluntary motion and of consciousness, combined with a slowing of the pulse-rate.

The theory of compression is still unsettled and under discussion. I do not desire to discuss a doctrine which is still doubtful or incomplete. I shall therefore content myself with this outline of the clinical picture, which can be used as a foundation for the theory of compression of the brain. The second form alone can, in the strict sense of the word, be called compression. If a large clot of blood rests upon a hemisphere, and if the brain surface is not only flattened but depressed, so that a true concavity exists at the spot at which the extravasation is situated, no doubt can be entertained but that the brain is *compressed.* It is not only pressed upon, but also depressed. True, at first the compression is merely local, but it has been experimentally ascertained that if pressure is applied to circumscribed portions of the brain, the increase of intracranial pressure is transmitted in all directions through the brain substance. The pressure, therefore, affects the whole brain, as the local pressure soon becomes general. The theory explaining this last fact has not been fully elaborated.

If a theory is not quite ready for us we may as well stick to old traditions. Now, what are these?

If consciousness, lost immediately after an injury to the skull, returns, we heave a sigh of relief and call the disease concussion. But if unconsciousness again sets in after a short interval, we try to explain this by assuming that an extravasation of blood has taken place in the meantime, and has now reached sufficient proportions to compress the brain. If, after the trauma, consciousness does not return—although the patient continues to live day after day—and if depression of the skull is present, we explain the case by declaring that the brain is compressed by the depressed fragment of bone. But if consciousness finally returns and the paralyses are recovered from, we assume that the brain has accommodated itself to the increased pressure, or that an extravasation of blood which was present has been absorbed.

There are cases in which the paralysis persists although consciousness returns. If the paralysis disappears, after elevating the depressed bone, the palsy was due to purely local causes; if it continues, it was due to contusion of the brain.

If we attempt to apply these theoretical conclusions to the clinical facts, we frequently come face to face with great difficulties. For, in the majority of cases, local lesions and compression of the brain are found in combination; it could hardly be otherwise. Primary symptoms of compression can result from only two causes; either from a depressed piece of bone or from an exudate of some size, often further complicated by a local lesion, for, in this latter instance, the force causing the injury frequently produces some lesion of the brain substance in addition.

As a rule, however, the problem is soon solved. In a large majority of cases the course of events is as follows: The diffuse lesion—that is, the concussion—is the first symptom. As this is recovered from, the local lesion, with its symptom-complex, comes into prominence. The injured person suffers from unconsciousness, vomiting, and slowing of the pulse. But, sooner or later, consciousness returns, and consequently compression is no

longer present. Then, with return of consciousness, aphasia, hemiplegia, or monoplegia develop. Therefore, we are dealing with a local lesion.

A single difficulty still requires explanation. Some symptoms of a local lesion—for instance, paralysis in the distribution of a cranial nerve—can be present without injury to the brain itself. If a fracture of the base tears or compresses a cranial nerve, the functions of the nerve will be interfered with. Thus blindness, deafness, anosmia, paralysis of the facial or of the abducens, etc., may result. The same symptoms may be produced by a bullet or stab wound severing any of these nerves in their course. It is often impossible to decide, on the spur of the moment, whether the penetrating instrument has reached the inside of the skull or not. At such times we are left in doubt as to whether a nerve or a centre has been injured until the combination of symptoms clears up the uncertainty.

Let us take the case of a penetrating body—for instance, of a projectile—which produces a circumscribed paralysis in the distribution of a cranial nerve. Certain facts taken for granted, two possibilities are then to be considered. Either some portion of the brain, or the cranial nerve itself, may have been injured. If the bullet has entered the forehead, either the optic nerve or the optic centre may have suffered. A bullet penetrating in the region of the ear may injure the auditory or facial nerve, or may reach their centres. In such cases the question arises, Is the paralysis of centric or peripheral origin? Neurologists have made strenuous efforts to solve this question, which is of such vital importance in the diagnosis of nervous diseases. They have endeavoured to discover certain functional symptoms by which we might hope to distinguish peripheral from central paralyses. When, for instance, not all the muscles supplied by the facial are paralyzed (the orbicularis and frontalis retaining its function), and when the paralyzed muscles retain their proper electrical reaction, neurologists conclude that the lesion is central. However, these theories have not as yet been sufficiently developed to prove of much assistance to the surgeon. Familiarity with the course of the nerves still remains of prime importance. For instance, as the result of an injury, the function of two or more nerves, which are close together in some part of their course along the base of the skull, may be disturbed. Their centres in the brain, however, are known to be separated by a large interval. We therefore can conclude that the point of injury is situated at that spot in their course at which they are in close proximity. If disturbance of the function of *spinal* nerves (as hemiplegia) is added to disturbances of *cranial* nerves, the obvious conclusion must always be that the brain itself has been injured, for the tracts from the higher centres pass through the brain on their way to the spinal cord.

The following examples will be used to illustrate some of the rules mentioned above:

A man, fifty years of age, was picked up unconscious in the street and taken to the hospital. A lacerated wound, extending down to the periosteum, was found over the parietal bone. The patient's beard and clothing were soiled with vomitus. The pulse was about seventy-two; the face pale. When loudly spoken to he mumbled unintelligible words. As the vomitus smelt strongly of alcohol, and the patient's breath smelt of the same, the question arose whether the unconsciousness and vomiting were symptoms of alcoholism or of concussion. The pallor might have been accounted for by the coolness of the autumn night. During the dressing of the wound, which caused