tricity has not yet been largely used by surgeons for purposes of diagnosis, there is one way in which it affords most valuable aid in localising the position of a paralysing lesion. It depends upon the fact that when a nerve trunk in any part or its centre is injured, the irritability of the paralysed muscles to the faradic current rapidly diminishes until it finally disappears, while they respond more readily to the constant current. This increase of voltaic irritability, and diminution or loss of faradic irritability, are known as the *reaction of degeneration*. This sign can be observed within a few days of the onset of paralysis, and becomes more and more marked; it is always associated with rapid wasting of the affected muscles; it is applicable to both the cranial and spinal nerves. Where, therefore, paralysed muscles show this reaction of degeneration, it is to be accepted as certain evidence that the lesion causing the paralysis is affecting the nerve supplying the muscles, or the nerve nucleus; if no such reaction of degeneration be found, it shows that the lesion is situated in some part of the nervous apparatus above the nerve centre. This sign tells us nothing of the cause of paralysis, only its site.

(2) **The nature of a lesion** is indicated partly by the class of symptoms which it occasions, and partly by their time of onset. (*a*) *Irritation* is caused by slight compression, by superficial or partial laceration of the brain substance, or by inflammatory congestion; it rarely affects the cranial nerve trunks. (*b*) *Paralysis* is caused by complete laceration of brain or nerve, by firm compression by bone, blood, or inflammatory exudation, or by shaking up of the brain with more or less appreciable contusion, or by shock. (*See* page 57.) (*c*) Symptoms produced immediately by an injury are caused either by *concussion* of the brain or by *compression* by displaced bone. (*d*) Symptoms coming on within a short interval of an injury (an interval measured by hours) are due to the pressure of *effused blood*. (*e*) Symptoms arising later on are the result of *inflammation*.

Cerebral localisation.—It has in quite recent years been shown that the cortex of the cerebrum is functionally differentiated into a number of centres. When a lesion of the cortex has been produced, observation of the function or functions affected enables, or may enable, a diagnosis of the exact seat of the lesion to be made; when the lesion is one that admits of surgical treatment (abscess, a depressed splinter of the internal table, etc.) this becomes of great importance. There is still some difference of opinion as to the precise seat of the various motor and sensory areas; and in what follows I am accepting the views put forth by Dr. Ferrier.

The cortex of the anterior two-thirds of the frontal lobe of the brain (that corresponding to the frontal bone) is not the seat of any centre for voluntary motion, or of perception of sensations, but is probably concerned with the higher mental functions only. The region of the brain bounding the fissure of Rolando, including the posterior extremities of the three horizontal frontal convolutions, the whole of the descending frontal and ascending parietal, and the parietal lobule, together with the inner surface of the marginal convolution, is the motor area, which is further divided up into special centres; the temporo-sphenoidal lobe, with the supra-marginal and angular gyri, are the seats of the perception of the special centres. The part of the motor area adjacent to the great longitudinal fissure is the centre for the movements of the legs; the lower part of this area is the centre for the arms; and the part in front of the lower extremity of the fissure of Rolando, that part of the area in front of the ear, is the centre for the face. The mesial surface of the marginal convolution is the centre for the trunk

muscles, the anterior part representing the neck, the middle part the shoulders, and the hinder part of it being the centre for the lower muscles of the trunk. The superior temporo-sphenoidal convolution has been shown to be the centre for hearing. To delineate these centres on the scalp, the following directions will suffice: Place the head so that a line drawn from the lower edge of the alveolar process of the upper jaw to the lowest part of the occipital bone is horizontal. The motor area is limited above by a line half an inch from the middle line, and below by a horizontal line drawn backwards from the external angular process of the frontal bone. Its anterior limit corresponds with the coronal suture, and is approximately marked by a line drawn vertically up from the middle of the zygomatic arch. Now draw two vertical lines, one through the external auditory meatus, and the other 50 mm. behind this; the fissure of Rolando will be under a line drawn obliquely from the top of the posterior of these two lines to the spot where the anterior of them crosses the line drawn horizontally back from the external angular process. The centre for hearing lies immediately below this horizontal line, and parallel with it; that for vision will be found above this line, and just behind the posterior of the two vertical lines above mentioned. Broca's speech-centre is immediately above the level of the external angular process of the frontal bone, and one inch behind that point of bone. For many further details regarding this most important subject, the reader is referred to the writings of Bastian, Ferrier, Gowers, and others.

The primary lesions of the cranial contents are:

Concussion.
Contusion and laceration.
Compression.

(1) If after an injury to the head, or a general shake of the body, the patient be temporarily unconscious, with muscular relaxation (falling down) and pallor, or if he simply feel giddy and faint, with some nausea, the symptoms are to be attributed to *concussion of the brain* in its milder form.

(2) If immediately after such an injury a patient be found apparently quite unconscious, but yet capable of being roused to some slight extent by loud shouting or a strong sensory impression (pinching or a bright light), with complete muscular relaxation, the limbs being flaccid and motionless, and the urine and fæces have passed involuntarily from relaxation of the sphincters, the surface being pale and cold, the pulse small, soft, and frequent (sometimes irregular, and in extreme cases imperceptible at the wrist), the respiration shallow and noiseless, the pupils either unaltered in size or slightly contracted or dilated, or unequal on the two sides, but acting slowly to a bright light, the case is one of severe *concussion of the brain.* All grades of severity in the symptoms, from slight temporary disablement up to sudden death, are met with in cases which are to be grouped under the heading of *concussion.* Where the symptoms are severe, there is always a distinct amount of contusion and laceration of the brain substance, as proved by post-mortem evidence.

(3) If, along with the symptoms of concussion of the brain, there be twitchings of certain groups of muscles, or more general convulsions, or if the patient exhibit great irritability of manner, and lie curled up with all his limbs flexed, eyelids closed, and if he resent all interference, resisting attempts to open his eyelids or mouth, the symptoms may be accepted as evidence of *contusion and laceration* of the surface of the brain; by closely noting the muscles affected, the seat of the brain lesion may be approximately or accurately determined.

(4) If, in a case of injury to the head, brain matter (proved to be such by microscopical examination) be found mixed with the blood from the wound, in the hair of the scalp, or in the nose, ear, or mouth, or lying in a wound into the orbit, it of course demonstrates *laceration of the brain;* and it must be remembered that patients may present themselves with brain matter thus extruded from the cranium without any symptoms to indicate so grave a lesion, without unconsciousness or paralysis.

(5) Whenever after an injury cerebro-spinal fluid is found escaping from the skull, it proves that there is a *laceration* of the visceral layer of the *arachnoid membrane* communicating with a fracture of the bone. When the escape of this fluid is very abundant, and particularly if it flow from a fracture of the vault, it is accepted as evidence of a *laceration* of the brain opening up one of the *ventricles.* The cerebro-spinal fluid may escape from a wound in the soft parts, or form the cystic tumour of the scalp described on page 82; as a complication of fractures of the vault it is a very rare circumstance. The distinctive characters of cerebro-spinal fluid have been already given. (*See* page 83.)

(6) If after an injury to the head a patient be found absolutely unconscious, it being impossible to arouse him or to make him answer questions, with fixed dilated pupils, slow, deep, stertorous breathing with flapping of the cheeks during expiration, slow, full, laboured pulse, retention of urine and involuntary passage of fæces, and paralysis, general or of one side of the body only, these symptoms are caused by *compression of the brain.* An attempt must be made to determine the source of the compression, and first of all *depressed bone* must be sought; and if a depressed fracture be detected, and the symptoms of compression are known to have been instantly produced, it may be

regarded as the cause of the compression. If, however, the symptoms of compression have come on gradually and at a short interval after the accident (an interval during which the patient may have been quite conscious, or showing more or less evidence of brain concussion), or if while under observation it be noted that the signs of compression are increasing, the coma becoming more deep and the paralysis more extensive, it indicates clearly *intracranial hæmorrhage.* When there has been an interval of consciousness between the injury and the oncoming of the symptoms of concussion, showing that the brain has not been seriously shaken or contused; and, further, if the paralysis is or was at first unilateral, we may diagnose that the hæmorrhage has taken place between the bone and the dura mater from rupture of a meningeal artery or wound of a sinus; while under the other conditions we have evidence of more severe direct injury to the brain, making it probable that the blood clot is situated on or in the brain. The symptoms of compression coming on after an interval of days or weeks from the injury, and preceded by signs of intracranial inflammation, indicate that the cause is the presence of *inflammatory products.* (*See* page 97.)

If the surgeon should trephine the skull on the supposition that there is a clot of blood between the bone and the dura mater, but on removing the piece of bone fail to find the clot, but notice that the untorn dura mater bulge up into the trephine hole, and is pulseless, and of a dark-blue colour, he may diagnose *arachnoid hæmorrhage,* and make an incision into the dura mater. When the history and symptoms indicate extrameningeal hæmorrhage, the surgeon must determine on which side the clot of blood is. Two signs will guide him. If there be paralysis on one side only, or if the paralysis be more marked on one side than the other, or if the general paralysis

have been preceded by unilateral spasm, the blood-clot is on the side opposite to the paralysis or spasm. And further, the hæmorrhage will be on the same side of the skull as the injury; and therefore if a bruise can be detected on one side, or if there be a wound, this will guide the surgeon as to the position of the clot, and indicate where the trephine should be applied if that treatment be indicated. It must be stated that the symptoms of intracranial hæmorrhage are often mixed with and obscured by those of severe concussion and laceration of the brain; they are mainly characterised by paralytic phenomena.

(7) Cases often present themselves in which there is great difficulty in determining whether the symptoms are due to the effects of *alcohol* or to a serious lesion of the brain. Whenever there is this doubt, and whenever a drunken man is known to have received a severe injury to the head, he should be kept quiet for some hours, until the effects of the alcohol have passed off. The signs of alcoholism upon which reliance is generally placed are the peculiar odour of the breath, the flushed face, heavy but not stertorous breathing, the contracted pupils, which dilate on arousing the patient, incoherent delirium, a tendency to become quarrelsome, tremor and unsteadiness of gait if able to walk at all, the absence of paralysis, and finding the bladder full of urine, in which alcohol may sometimes be detected. The history where obtainable is of course of the highest importance.

(8) In other instances the question arises whether a person found insensible and paralysed has had an attack of *apoplexy*, and fallen down, or has received a serious head injury causing the paralysis. Here again there are cases in which it may be necessary to suspend judgment for a time, or even altogether. For the symptoms produced by a clot of blood in the brain are of course the same whether the rupture of the blood-

vessel be spontaneous or excited by a blow. In all such cases the age of the patient and all the attendant circumstances must be carefully noted, and the heart and the urine should be examined, as the detection of a cardiac murmur or aortic aneurism, or of albuminuria, would be strong corroboration of the diagnosis of apoplexy. Careful search should be made for a wound or contusion of the scalp, depression of bone, bleeding from the nose, ear, or pharynx. A disproportion between the loss of consciousness and the paralysis, if present, would be of great assistance in the diagnosis, for in traumatic cases the sensorium, as a rule, suffers much more than the motorium, while in the idiopathic cases there may be complete hemiplegia with only very transient insensibility.

With regard to **lesions of cranial nerves,** the following points are of practical importance.

Loss of smell is the sign of injury to the olfactory nerve; but care must be taken to ascertain that the nasal fossa is clear, and not obstructed with blood-clot, displaced bone, or cartilage.

Injury of the third nerve often affects only a part of the nerve, and causes partial palsy; thus a combination of ptosis and a dilated fixed pupil is not infrequent.

Paralysis of the superior oblique muscle causing diplopia below the horizontal meridian, and internal strabismus only when the eye-ball is directed downwards, may be due to a lesion of the **fourth nerve,** or of its centre, or to interference with the play of the tendon of the muscle in its pulley; any thickening, pain, or tenderness at the seat of the pulley would render the latter the more probable diagnosis.

The fifth nerve is rarely affected alone; when the lesion is partial the cornea rapidly sloughs.

Internal strabismus without palsy of any

muscles except the external rectus points to injury of the **sixth cranial nerve** alone.

The seventh nerve is the one most frequently injured in fracture of the skull; the signs of facial palsy are unmistakable. In estimating deafness from injury to the portio mollis, reliance must be placed upon failure to appreciate sounds transmitted through the cranial bones rather than upon those transmitted through the auditory meatus and tympanum, as these may be obstructed with blood-clot.

The eighth and ninth nerves are rarely injured; dysphagia, dyspnœa with slow respiration, altered rhythm of the heart's action, palsy of the tongue, and anæsthesia of the pharynx and back of tongue, are the signs by which the lesion would be recognised.

The secondary lesions of the cranial contents are:

(1) Hernia cerebri.
(2) Intracranial inflammation and suppuration.

(1) If after a compound fracture of the skull, or the operation of trephining, or the removal of a sequestrum from the vault of the skull, a soft mottled grey and dark-red tumour protrudes through the aperture in the bone, and if of large size, grows in a mushroom-like form, bleeds easily when touched but is not sensitive, exhibits an expansile pulsation synchronous with the carotid pulse, and is distended by crying or coughing, or any effort which increases the intracranial pressure, we have to do with a *hernia cerebri*. Microscopic examination of a portion of the mass will prove beyond all doubt the nature of the fungus if large irregular nerve cells be detected. The history of injury to the bone preceding the appearance of the fungus at once distinguishes it from pulsating malignant tumour. (*See* page 382.)

(2) When, after an interval of from two or three

days to weeks, months, or even years, after an injury to the head, there is persistent headache with intolerance of light and sound, contraction of the pupils, flushed face, fever, quick full sharp pulse, vomiting, with a thickly furred tongue and foul breath, and if these symptoms run on into delirium, followed by twitchings, convulsions, strabismus and coma, it is undoubted that *diffuse traumatic meningitis* exists. Of this group of symptoms there are, however, many modifications; pain alone may be present, and is of all symptoms the most frequent and constant, and when localised to the seat of the injury indicates a limited inflammation. In other cases muscular twitchings or paralysis may be the only sign of secondary intracranial mischief. It is important to remember that *abscess of the brain* may exist without any symptoms, and that not only may there be no fever, but it would even seem that a subnormal temperature is a frequent event in this complication. Sudden coma from the bursting of an abscess into the arachnoid cavity, or a sudden discharge of pus from the nose or ear, may be the first sign of a cerebral abscess. If, on account of symptoms of local intracranial inflammation, the interior of the cranium be explored, pus may be found outside the dura mater; and whether or not it be found, the surgeon should carefully note whether that membrane bulge up into the hole in the skull, and whether it pulsate. If it thus bulge up, and do not pulsate, he may diagnose subdural suppuration, and incise the membrane; but if these symptoms are not present, the surgeon should, with a fine exploring needle or trocar, puncture the brain in one or more directions in the hope of reaching a cerebral abscess.

Intracranial inflammation may cause, as its earliest symptom, spasm of muscles on the opposite side of the body; this is particularly characteristic if the muscles have been previously paralysed by compression

of their cortical centre, or if the spasms affect muscles under the governance of centres immediately adjacent to those connected with paralysed muscles. By noticing the distribution of the spasm, an exact diagnosis of the seat of the lesion can be made, and a gradual extension of the convulsions or paralysis from group to group of muscles is characteristic of cortical lesions, progressively implicating neighbouring centres.

CHAPTER VI.

THE DIAGNOSIS OF INJURIES OF THE SPINE.

Injuries of the spine derive their chief importance from the liability to implication of the spinal cord or of the spinal nerves. Paralysis at a distance from the seat of injury is the great symptom of injury to the cord or nerves, and as, in cases where these parts have been injured, any forcible manipulation may lead to further and serious mischief, the examination should be so conducted as to determine, first, whether the cord or nerves have been injured, and, if so, where; then whether the bones are fractured or dislocated; thirdly, whether the ligaments or muscles have been strained, torn, or bruised; while, last of all, lesions of the skin must be investigated.

1. **Injuries of the spinal cord and nerves** are indicated by *paralysis* and sometimes also by signs of *irritation;* what has been written in reference to these signs of intracranial mischief (page 87, *et seq.*), applies with almost equal force to injuries of the spine. In the investigation of traumatic paralysis for the purpose of diagnosis, two points must receive especial attention, its *extent*, and the *time and mode of onset*

of the palsy. By the latter we are able to judge of the actual cause of the paralysis; by the former we are able to localise the lesion.

The *extent of the paralysis* should be carefully determined; the power of voluntary motion, of sensation, and the condition of the various reflexes being separately and systematically investigated. The first use of the information thus obtained will be to decide whether the lesion is situated in the spinal cord, or in the nerves arising from it. Wherever the palsy is complete below a certain horizontal plane, involving both voluntary motion and sensation, it is undoubtedly due to *injury of the spinal cord.* But it requires a very complete crush of the cord to abolish entirely all sensation in the parts below, for if even a small portion of the cord be intact it will suffice to conduct some sensation, and hence it is that complete and absolute loss of sensation is more often met with in cases of injury to nerve trunks. Where the paralysis corresponds to the distribution of an individual nerve or nerves, it is due to lesion of those nerves. But a limited lesion of the cord may produce a limited paralysis of sensation or motion, according to the part of the cord affected; *e.g.* a lesion of the lateral column of the cord may cause paralysis of voluntary motion on the same side without affection of sensation or of the reflexes, and without marked wasting of the paralysed muscles; and similarly, a lesion limited to the posterior column of the cord may cause disorders of sensation without loss of power of voluntary motion. Injuries do not observe the strict regional limitations often found in diseases of the cord.

If paralysis ensue immediately on the receipt of an injury, it is due to some interference with the functions of the part caused directly by the injury, either *concussion* or *crushing*, and the diagnosis between these two will be readily made by