REFRESHER COURSE FOR GENERAL PRACTITIONERS

HEAD INJURIES—I. BRAIN INJURY AND ITS DIAGNOSIS

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Injuries of the head derive their seriousness largely from the degree to which the brain has been damaged or may subsequently suffer damage by complications. The brain may sustain concussion, contusion, or laceration; it may be gradually compressed by an effusion of blood; or it may become the seat of inflammation if infection invades the cranial cavity as a result of the injury providing a portal of entry. The only importance of fractures of the skull, if not compound or depressed, is that they give some indication of the severity of the impact.

The diagnosis of the nature and extent of the brain injury, and of the presence or otherwise of complications, such as compression of the brain, rests essentially on the clinical picture.

Concussion

Concussion of the brain is characterized by the immediate cessation of consciousness after a blow on the head. If examined within a few moments of the injury the patient will be found to be profoundly unconscious, pale, and to have a slow feeble pulse and shallow respirations; the limbs will be flaccid; and all reflexes, such as the pupillary, corneal, tendon, and plantar, will be unobtainable. If the blow was not severe this state will rapidly resolve and within a few minutes the patient will be fully conscious and the vital activities and all reflexes will be normal. It is now generally accepted that concussion is a reaction of the nerve cell to certain physical stresses which is characterized by immediate paralysis of its function, with minimal or no structural damage, and a natural tendency to rapid recovery. This is in keeping with Trotter's classical definition: "An essentially transient state due to head injury which is of instantaneous onset, manifests widespread symptoms of purely paralytic kind, does not as such comprise any evidence of structural cerebral injury, and is always followed by amnesia for the actual moment of the accident."

Experimental work carried out in recent years shows that the brain is most readily "concussed" by a force which causes abrupt acceleration—when a rapidly moving object strikes the stationary head—or deceleration—when the falling head strikes the ground. If the head does not move but is suddenly crushed, as in shunting accidents, consciousness is often retained in spite of extensive fracture of the skull. A much greater force must be expended in order to cause "compression" concussion as compared with "acceleration" concussion. The energy dissipated in producing concussion is usually supplied by a blunt or smooth-surfaced heavy object; if the force comes from a small rapidly moving object, such as a bullet, the energy is dissipated in penetrating the skull, causing only local damage to the brain. Provided the missile does not damage vital centres, consciousness is often retained, the reason being that insufficient acceleration has been imparted to the brain to bring about the diffuse neuronal disturbance responsible for concussion.

Contusion and Laceration

In addition to the diffuse disturbance of neuronal function called concussion, injury may, and often does, bring about structural brain damage. Necropsy reveals haemorrhages, petechial or coarse, subpial or within the substance of the brain, scattered diffusely or localized to the several poles of the brain. Microscopical examination shows areas of structural damage to the nerve cells and their tracts, and to

small and large blood vessels; areas of glial reaction to this tissue injury will be found if some hours have elapsed before death. The term "contusion" is given to such breaches of anatomical continuity. When the damage is more severe naked-eye examination may reveal laceration and dissolution of brain tissue and extensive haemorrhage. Contusion, laceration, and haemorrhage may be found immediately under the site of impact, particularly if there is a fracture of the overlying skull; or the damage may be at a remote part of the brain, and then constitutes contre-coup injury. These coarse lesions of the brain arise as a result of the sudden distortion of the skull, with or without fracture, brought about by the impact, and of the various shearing stresses imparted to the brain, which tends to swirl about within the cranium. Such physical stresses will be prominent where the poles of the brain are brought in contact with the buttresses of the cranium—for example, the temporal poles against the sphenoidal ridges—and where such movement is restricted by the great dural partitions, the falx and the tentorium,

It is not clear yet to what degree the coarse lesions of brain injury affect recovery from concussion. It is quite certain that contusion may occur without concussion; delayed symptoms of headache, dizziness, sleeplessness, and lack of concentration may arise after a glancing blow on the head which does not cause loss of consciousness. If the unconsciousness of concussion is a transient functional disturbance, then the prolonged coma and slow recovery after severe head injury may be regarded as the result of structural brain damage. Whether or not slowness of recovery of consciousness is due to contusion, it is certain that many of the permanent residual disabilities are due to the structural brain damage. Impairment of intellect and personality changes may follow widespread bifrontal contusion, and hemiparesis, dysphasia, hemianopia, etc., will result if there has been extensive tissue injury in the anatomically appropriate areas of the brain.

Compression of the Brain

The essential injury of the brain, be it concussion, contusion, or laceration, produces a clinical syndrome in which there is a strong tendency towards recovery. Provided the vital centres are not irreparably damaged and complications do not develop, and provided treatment is along correct lines, there should be a gradual and steady improvement in the patient's condition. To this picture, produced by the immediate brain damage, Jefferson has applied the term "basic state." The value of this conception resides in its emphasis on the word "basic," from which datum line one can take measurements of improvement or of deterioration. This implies observation over a period of time during which symptoms and signs may develop, indicating the presence of complications, the most important of which is compression by blood. Jefferson emphasizes the importance of such fresh developments by calling them "epiphenomena."

Injuries of the skull, either by fracture or by distortion, may tear meningeal vessels, usually the middle meningeal ones. Haemorrhage occurs between the dura mater and the skull and gradually separates the membrane from the bone, the brain being compressed during this process. The rapidity of the bleeding is such that an extradural haematoma manifests itself usually within 6 to 24 hours of the accident and if unrelieved by operation it swiftly kills. Bleeding may also occur into the potential space which

exists between the dura mater and the arachnoid, forming a subdural haematoma. The bleeding arises from veins passing from the cortex to the large dural sinuses; it is often bilateral, and the blood, which for the most part remains unclotted, spreads diffusely over the convexity. The haematoma increases in size more slowly than the extradural variety and manifests itself somewhat later. If the quantity is small its presence may evade detection for weeks, particularly if the head injury was trivial, and it then tends to encapsulate itself by membrane formation; in such cases, and in elderly subjects in whom it is prone to occur, the head injury may be forgotten and compression of the brain by a chronic subdural haematoma may then simulate cerebral thrombosis or tumour. A similar condition can occur in infants, causing enlargement of the head, and may be mistaken for hydrocephalus.

Cerebral laceration usually causes an effusion of blood into the subarachnoid spaces, and sometimes into the ventricles. Such bleeding rarely of itself leads to compression of the brain. Laceration severe enough to cause a serious degree of subarachnoid or ventricular bleeding is usually fatal because of the intrinsic brain damage. Intracerebral haematoma, other than in rapidly fatal injuries, is relatively rare. It may arise from focal laceration and vessel damage, or it may occur as a sudden event some days after the accident, the so-called *spät apoplexie*.

As a result of cranial distortion the arachnoid may tear, allowing cerebrospinal fluid to seep into the subdural space. Owing to the poor rate of absorption of liquids from the subdural space, the cerebrospinal fluid may collect in quantities large enough to compress the brain. It is then called a "subdural hydroma"; it is generally of slow development and is clinically indistinguishable from subdural haematoma.

Fractures of the Skull

As with fractures of other bones, skull fractures may be closed, when the overlying skin is not broken, or open (compound), when the fracture line communicates with the external air through a wound. The importance of this classification is precisely the same as that in other bones—the risk of infection if the fracture is open. A fracture of the frontal bone which appears closed, there being no overlying scalp wound, may nevertheless involve the posterior wall of the frontal sinus or the roof of the nose, and thereby tear the mucosa; in this way it is in fact open. A fracture of the middle cranial fossa which involves the middle ear is similarly open. The intact dura mater underlying an open fracture forms a good barrier against infection, but if the injury has also torn the dura mater a penetrating infection may lead to meningitis and even to brain abscess. This is particularly prone to occur in fractures involving the frontal sinus or roof of the nose, with tearing of the dura mater, and such fractures may easily pass unrecognized; a flow of cerebrospinal fluid from the nose (or from the ear in a case of fracture of the middle fossa) is of course pathognomonic.

In addition to being open or closed, a fracture may be fissured or comminuted. It may not alter the normal contour of the skull, or the force of the impact may cause depression of fragments. In children a depressed fracture often occurs without laceration of the overlying skin, but in adults the skin is usually broken. The area of damaged bone may form a saucer-like depression, or individual fragments may be so tilted and so pointed as to tear the dura mater and to damage the underlying brain. In severe injuries, and with missiles, the bone fragments may be projected fanwise into the brain. Depressed fractures compress the brain in the immediate locality, but if symptoms of generalized brain compression develop they are usually due to an accompanying extradural or subdural haematoma.

Scalp Wounds

Lacerations of the scalp may vary from minor superficial abrasions to extensive and complicated wounds. They may be widely contused or cleanly incised. There may be avulsion of flaps. Serious loss of blood may occur from a

scalp wound, and cause shock which may disguise the basic state. What may be regarded as "only a scalp wound" may in fact be an open fracture. Damage to underlying brain, a focal contusion, may be present in addition to the scalp wound, whether or not there is a fracture of the skull; thus a scalp wound on one side of the head, without any fracture of the skull, may be associated with a weakness of the opposite arm or leg.

Cranial Nerve Palsies

In addition to the injury sustained by the brain, the individual cranial nerves may be damaged between their points of emergence from the brain and their foramina in the skull, or in those foramina. The paralysis may be transient, of long duration, or permanent, according to the degree of damage and to the particular nerve involved.

Injury to one or both *olfactory nerves* often occurs in frontal-lobe contusion, and particularly when the impact is received on the forehead or the occiput. The sense of smell may be partly or completely lost, at one or both nostrils. If the anosmia is absolute, appreciation of flavour is also impaired. The disability is potentially serious, because the patient is unaware of the smell produced by a leak of gas or by petrol. A perverted sense of smell—*parosmia*—or olfactory hallucinations may sometimes occur.

One or both *optic nerves* or the *optic chiasma* may be injured; bizarre abnormalities of the visual fields may be detected, according to the site and degree of the lesion, or vision may be completely destroyed. There is rarely any accompanying fracture of the optic foramen, although a fracture through the floor of the anterior fossa may be evidence of the severity of the force involved. Loss of vision will not be revealed till full consciousness is gained, but if one optic nerve is severely damaged the pupil of that eye will not react to direct light stimulus, and the significance of such a single abnormal sign may not be immediately appreciated. Primary optic atrophy will develop after several weeks, confirming the nature of the injury.

The *motor nerves of the eyeball*—the oculomotor, the trochlear, and the abducens—may be injured within the cranium or more often within the orbit as a result of severe bruising, commonly due to fracture of the wall of the orbit. The intraorbital bleeding may produce proptosis. Disturbances of the pupillary reactions and squints will occur. Such signs may also be caused by damage to the nuclei in the brain stem.

Damage to sensory branches of the *trigeminal nerve* usually occurs only as a result of fracture of the facial skeleton. Paralysis of the masticatory muscles is very rare.

The *facial and acoustic nerves* may be involved in a fracture of the petrous bone. Damage of the vestibular portion of the eighth nerve may give rise to vertigo. Injury of the other cranial nerves does not occur except as part of a fatal injury.

Diagnosis

Loss of consciousness is an essential feature of concussion, and for this period of time there is complete loss of memory. If the patient has already regained consciousness when the doctor arrives concussion can be assumed to have occurred if there is no recollection of the actual injury. If return of full consciousness is delayed for more than a few minutes, if abnormal neurological signs persist for more than 24 hours, or if there are signs of a coarse lesion of the brain—for example, monoparesis or hemiparesis, etc.—then the basic state indicates structural damage—namely, contusion (with or without laceration). Consciousness may not return for hours or days, and the patient passes successively through periods of stupor and mental confusion, and intermittent periods of full and clouded consciousness, until the normal is reached. There may be stages of mental and physical excitement, with resistance to nursing care—so-called irritability, which should be more accurately regarded

as being due to paralysis of the higher inhibitory mental processes. Incontinence of urine and faeces may occur, or retention of urine may aggravate restlessness. Fever, neck stiffness, and a positive Kernig's sign indicate irritation of the meninges by blood. Such signs will also develop if meningitis occurs, and the possibility of infection spreading through a hidden basal fracture must be borne in mind. Persistent headache, vomiting, and giddiness are common symptoms of brain contusion and, as pointed out earlier, may occur without loss of consciousness—that is, without concussion. The pulse rate is commonly raised for a day or so after brain contusion, and then is often succeeded by a bradycardia about the fourth day lasting for some days. The temperature may rise to 103° F. (39.4° C.), or even higher, within a few hours of a brain injury, although occasionally the injury is so severe that a pyrexial reaction does not occur; the thermo-regulatory mechanism is paralysed and the temperature falls; such injuries are commonly fatal.

When confronted with a case of head injury the presence of concussion with or without contusion may be self-evident; but a common difficulty is to decide whether the patient is also suffering from another disease or intoxication which may have caused him to fall and stun himself—for instance, epilepsy, acute alcoholism, uraemia, diabetes, or cerebral haemorrhage from atheroma or aneurysm. An account of the accident given by an eye-witness may be of great value; examination of the exterior of the cranium may reveal ample evidence of a severe injury. Information from relatives or the doctor's own knowledge of the past history of the patient may clarify the possible importance of non-traumatic causes of coma.

In every case of head injury the problem arises whether there is cerebral compression or whether it is going to develop later. An unpredictable period of time must elapse after the injury before compression of the brain can occur. Consequently, immediately after an injury *no one* can predict whether or not compression will occur. If the concussion is brief and consciousness rapidly returns, the symptoms of compression, the epiphenomena, will be more easily recognized. As the clot enlarges, the fully conscious patient complains of increasingly severe headache, he may vomit, and consciousness becomes clouded and is finally lost. But if the brain injury is severe there may be no lucid interval—that is, consciousness is not regained but gradually deepens as compression progresses, and the most critical observation is necessary in order to detect this change at the earliest moment. Accompanying the recurrence or the deepening of unconsciousness will be found fresh neurological signs of a paralytic order. The pupil on one side (usually the side of the bleeding) becomes dilated and fixed (non-reactive to light) and the other pupil later follows suit. The opposite limbs become paralysed, their tone abnormal (sometimes spastic and sometimes flaccid), the tendon reflexes increase, and the plantar response becomes extensor. If the compression is not relieved the limbs on both sides become paralysed, and the patient may finally present a state of decerebrate rigidity with bilateral extensor plantar responses. Respirations become deep, laboured, and stertorous, and may terminally show Cheyne-Stokes rhythm; the patient's face is congested. Occasionally increase of blood pressure and a bradycardia may occur as cerebral compression develops; more commonly, however, little change occurs in the blood pressure after the patient comes under observation, and the pulse rate steadily mounts.

The crux of the matter lies in the repeated observation and recording of the patient's condition in order to detect at the earliest moment any deterioration of, or deviation from, the basic state. Where coma persists without improvement, or where abnormal signs lead to suspicions of the presence of a compressing lesion, it is advisable to make certain of the diagnosis by exploratory operation. This comprises the drilling of small burr-holes or trephine-holes over appropriate areas of the cranium; it should be done under local analgesia, and causes little disturbance of the

patient. It may prove a life-saving measure and should not be unduly deferred, for when compression has developed the patient's condition may worsen with incredible rapidity. Whether the haemorrhage is extradural, subdural, or intracerebral is of more moment to the surgeon who has to deal with the case. The time of onset of the change in the patient's condition, the rapidity of deterioration, the crispness of localizing signs or their diffuseness and insidious march, and the presence of local scalp or skull damage, all may help in locating the site of bleeding.

[Part II will appear in our next issue.]

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Nova et Vetera

EARLY CONTRACEPTIVE SHEATHS

During a recent examination of the contents of a muniment room in a large English country mansion a locked box was discovered which, on being opened, was found to contain a number of sheaths of an early type. They were in packets of eight, of three different sizes, and the inner (white) and outer (blue) wrappers were apparently those in which they had been delivered.

Some of these sheaths were brought to me at the British Museum for examination and report. The specimen submitted is apparently made from some animal membrane, and as far as could be discovered is seamless, the edge of the open end being turned over and roughly stitched with cotton to form a hem through which is threaded a strip of silk. Its approximate dimensions are: length, 190 mm.; diameter, 60 mm.; thickness, 0.038 mm. (as compared with the thickness of a modern thin rubber sheath of 0.075 mm. The membrane was found to be permeable to water and to a solution of salt and water. Both white and blue wrappers were made from rag and neither showed any watermark, the chain and laid lines being clearly visible; the blue colour of the outer wrappers was due to indigo.

If it be true, as is held by some authorities, that the appearance of a piling up of the pulp beside the chain lines (which is present in the white wrapper) indicates a date of manufacture probably prior to 1800, then this, with other features in the case, suggests that the date to be assigned to these sheaths is between about 1790 and 1810.

Although the inventor of the contraceptive and preservative sheath is still unknown, in spite of the attempts to prove that he was Colonel Condon or Cundum living in the reign of Charles II, much has been recorded about it in later times, especially in the eighteenth century. At that time skin sheaths were often used, and such devices continued to be sold till the twentieth century, when they gradually fell into disuse in England, being replaced with the more durable rubber condoms.

References in Literature

The specimens recently discovered are very similar to that described by Casanova (1725-98) in his *Mémoires* (Paris, 1931-2 ed., vol. 5, p. 275), where he speaks of a "petit habit d'une pellicule transparente d'environ huit pouces, sans issue et armée à son entrée d'une faveur rose passée dans une coulisse." Similarly, elsewhere, he writes of being shut up "dans un morceau de peau de mort pour avoir le bonheur de vous prouver que je suis parfaitement en vie."