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Lecture Notes

Head Injury

Scalp injury
Skull fracture
Intracranial haemorrhage (extradural, subdural, subarachnoid, intracerebral)
Brain injury
Complications of head injury

HEAD INJURY

The head is very vulnerable to injury, often with severe consequences. It is particularly susceptible to acceleration/deceleration and rotational forces because it is heavy in relation to its size (3-6 kg, avg 4.5 kg or 10 lb), is freely mobile in 3 dimensions and occupies a relatively unstable position, being secured only by the neck muscles and ligaments.

Analagy for head injury: blancmange (brain), wrapped in cling film (arachnoid), in a paper bag (dura), inside a cardboard box (skull), wrapped in brown paper (scalp).

Any layer may be damaged by:
Direct impact on the box (blow),
Dropping the box (fall) or
Shaking the box (acceleration/deceleration)

SCALP INJURIES
Scalp consists of several distinct layers: hairy skin, the subcutaneous fat and connective tissue layer, the galea or aponeurosis (a thin fibrous layer to which the flat epicranial muscles are attached), a thin layer of connective tissue and finally, the periostium of the skull.

The scalp is susceptible to all types of injury, particularly laceration as it is readily crushed and split against the underlying bone. Such lacerations are often linear due to the convexity of the underlying skull. The scalp often swells markedly due to oedema (waterlogged tissues) or haematoma formation (raised swelling) due to bruising above or below the galeal layer. Dense hair may mask scalp injuries.

SKULL FRACTURE

The adult skull is formed by the fusion of several plates of bone which become knitted solidly together along the cranial sutures. The skull bones each consist of a thick *outer layer* or table of bone, the spongey *diploe* and the thinner *inner table*. The inner skull table is lined by a thick fibrous adherent membrane (*the dura mater*). A small space (subdural space) lies between the inner surface of the dura and the thin membranous *arachnoid mater* which covers the surface of the brain.

The solid skull is deformed by localised impact which may damage the cranial contents even when the skull does not fracture. If the force and deformation is excessive the skull will fracture at or near the site of impact. Uncomplicated skull fractures themselves are rarely lethal - it is the associated intracranial damage which is lethal. A fracture indicates that substantial force has been applied to the head which is likely to have damaged the cranial contents. Skull factures may occur with no associated neurological damage and conversely, fatal injury to membranes, blood vessels and the brain may occur without overlying fracture.

Force required to cause fracture: is very varible and depends on the thickness of the hair, scalp and skull, upon which part of the skull is struck, the direction of impact and other imponderables. Skull fracture can result from merely walking into a fixed obstruction (73 Newtons or 5 foot pounds), from the 4.5 kg adult head falling from a height of 1 metre onto a hard surface (510 N), the head falling from a standing position (873 N), running into a obstruction (1020 N) or a 100g golf ball or stone thrown with moderate force against the temple.

Types of skull fracture:

Linear- from the point of impact along lines of anatomical weakness (eg diastasis of suture fusion lines).
frontal fracture (eg head-on RTA) often radiates into anterior cranial fossa
temporal fracture (eg blow to side of head) often radiates into middle cranial fossa
occipital fracture (eg backwards fall) often radiates into posterior cranial fossa

Radiating- outwards from the point of impact

Spider's web- radiating lines connected by concentric fracture rings

Depressed- where fragments are driven inwards

Hinge- passing across the base of the skull (motor cyclist or blow to chin)

Ring- encircling the hole through which the spinal cord passes downwards (foramen magnum). Due to a fall onto the feet or onto the top of the head

Contre-coup- a backwards fall, striking the back of the head (coup) may also cause fracture of the thin layer of bone over the roof of the orbits opposite the point of impact (contre-coup) due to suction forces transmitted through the brain tissue.

INTRACRANIAL HAEMORRHAGE (ICH)

The dural and arachnoid membranes and their associated blood vessels are readily torn by impact and by fractured bone fragments. The process of **haemorrhage** results in the formation of a localised accumulation of blood, or **haematoma**. There are 4 types of ICH:

1. Extradural haemorrhage (EDH)

A blow to the temple often fractures the thin temporal bone and tears the middle meningeal artery as it passes upwards within a groove between the inner skull table and the dura. In 15% of cases no fracture is identified. Arterial bleeding strips the dura off the inner skull table to form a haematoma which acts as a space-occupying lesion (SOL). This accumulation can be immediate or delayed. EDH is easily overlooked, as mild concussion is followed by a lucid interval before neurological symptoms and coma develop many hours later when the enlarging haematoma begins to exert pressure on the

brain. Amenable to surgical decompression in early stages.

2. Subdural haemorrhage (SDH)

More common than EDH. Not usually associated with skull fracture. Sudden jarring or rotation of the head (often a trivial blow or fall) causes movement of the brain relative to the dura. This shears and tears the small veins which bridge across the gap between the dura and the cortical surface of the brain. The leaking blood accumulates over several hours and usually tracks extensively as a thin film over the surface of the brain. SDH is especially common in the elderly (brain atrophy widens the gap), children (shaking injury as part of the child abuse syndrome) and alcoholics (frequent unprotected falls and prolonged bleeding times). A small, self-limiting SDH may remain asymptomatic and be an incidental finding at autopsy.

3. Subarachnoid haemorrhage (SAH):

May be *natural*, due to rupture of a dilated blood vessel (an aneurysm), or *traumatic*. SAH is highly irritant to the brain-stem and is usually rapidly fatal. Traumatic SAH may is usually associated with contusion or laceration to the brain surface.

Rarely SAH is due to a kick or blow to the side of the neck which stretches and ruptures the vertebral artery as it enters the cranial cavity. This phenomenon is called traumatic basal SAH and is most often due to a blow to the side of the chin or jaw in an alcoholic fist-fight. The degree of force required to cause death in this way is less than would be reasonably expected, resulting in prosecution for culpable homicide, rather than murder.

- References:

G. Dowling & B. Curry. Traumatic Basal Subarchnoid Haeomorrhage: Report of 6 Cases and Review of the Literature. *American Journal of Forensic Medicine & Pathology*. 1988. 9 (1): 23-31

B. Koszyca, J. Gilbert & P.C. Blumbergs. Traumatic Basal Subarchnoid Haeomorrhage and Extracranial Vertebral Artery Injury. *American Journal of Forensic Medicine & Pathology.* 2003. 24 (2): 114-118

4. Intracerebral haemorrhage:

May be *natural*, due to spontaneous rupture of a small blood vessel (arteriole) which has been weakened by the effects long-standing high blood pressure. Rupture is likely to occur at a time of stress or excitement when the blood pressure is acutely elevated.

May be *traumatic*, due to extension of haemorrhage from surface contusions deep into the substance of the brain. Traumatic intracerebral haemorrhage may also be the result of rupture of small blood vessels deep within the brain due to shearing stress.

BRAIN INJURY

The brain consists of the cerebrum, cerebellum and brain stem. The *cerebrum* is the largest component and consists of the right and left cerebral hemispheres, each of which has a frontal (front), parietal (upper side), temporal (lower side) and occipital (back) lobe. The cerebral hemispheres are separated at the midline by a vertical fold of dura called the falx. The occipital lobes sit above two further horizontal folds of dura (tentorium cerebelli) which separate them

from the *cerebellum* below. Below the cerebral hemispheres and in front of the cerebellar hemispheres lies the *brain stem*. Although small this houses the vital nerve centres controlling consciousness, breathing and heart function. The brain stem passes down through the only exit from the cranial cavity (the foramen magnum) to become the spinal cord.

Minor jarring of the brain causes **concussion**, a clinical state of transient loss of consciousness due to temporary nerve cell dysfunction. Retrograde amnesia is common. Bruising on the surface of the brain is called **cerebral contusion**, and is of 2 types:

"Coup type" contusions: a blow to the head when it is free to move, accelerates the head and causes cerebral contusion at the point of impact. Scalp injury (bruise, abrasion or laceration) is likely to occur at the point of primary impact. Contusion or laceration of the brain surface often occur at the site of a fracture, especially if it is depressed.

"Contre-coup type" contusions: when the falling head strikes the ground it decelerates abruptly while the semi-fluid brain continues moving towards the point of impact. This causes more severe contusions in the area diametrically *opposite the point of impact*. Such "contre-coup" contusions occur where the brain glides over the irregular, jagged contours of the skull interior and are usually more severe than the corresponding coup-type contusions. Negative suction pressures which develop opposite the point of impact are also involved in their causation. Greater contre-coup force may also lacerate the brain surface. In this way a backwards fall causes contre-coup contusions at the front of the brain (to the frontal and temporal poles). Similarly, a fall onto one side of the head causes contre-coup contusions at the opposite side of the brain (temporal lobe). This pattern is useful in distinguishing at autopsy head injuries due to falls from those due to blows. However, a forwards fall does not cause contre-coup contusions over the back of the brain due to the interior of the skull being smooth at this point.

Severe brain injury may still have occured even in the absence of any associated scalp injury, skull fracture, intracranial haemorrhage or cerebral contusions. Rotation & acceleration/deceleration injury are more damaging to the brain than direct impact against the fixed, immobile head. This is because rotation causes the layers of brain tissue to glide over each other like a pack of cards, causing shearing of the delicate connections between the nerve fibres. Widespread subtle microscopic nerve fibre injury (**diffuse axonal injury)** of this type is common in road traffic accidents, due to the frequency of severe rotational impact and deceleration. Such microscopic damage to nerve fibres is generalised and not amenable to surgey. There may be nothing to see on the surface of the brain or on slicing at autopsy. Diagnosis usually requires expert neuropthological examination using special stains to demonstrate subtle microscopic damage to nerve fibres. **Analogy is shaking or kicking a TV set - damage to wiring may occur with no apparent damage to the exterior of the TV case or its interior at first glance.** Diffuse axonal injury is often associated with brain swelling (see below). Shearing or gliding injury may also rupture small blood vessels deep within the white matter of the brain cause numerous deep small haemorrhages.

Death from Blunt Force head injury in association with alcohol intoxication

Punching & kicking assaults directed at the head, face and neck and causing minor or moderate soft tissue injury may result in death if the individual is intoxicated with alcohol. Alcohol intoxication lessens muscular protective reflexes and also sensitises the brain and brainstem to concussive trauma. Assault may result in mild diffuse axonal injury and stretching of the brainstem (where the vital centres controlling respration and heart rhythm are located). Stretching & dysfunction of the nerve cells in these vital areas of the brain stem may result in potentially fatal central apnoea (cessation of breathing) or disturbance in the electrical rhythmn of the heart (cardiac arrhythmia). When death occurs rapidly, the usual neuropathological

features of Diffuse Axonal Injury may not have had time to develop, even microscopically.

Reference:

D.A. Ramsay & M.J. Shkrum. Homicidal Blunt Head Trauma, Diffuse Axonal Injury, Alcoholic Intoxication and Cardiorespiratory Arrest: a Case Report of a Forensic Syndrome of Acute Brainstem Dysfunction. *American Journal of Forensic Medicine & Pathology* 1995. 16 (2): 107-114

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COMPLICATIONS OF HEAD INJURY

Intracranial haemorrhage (extradural, subdural or intracerebral haemorrhage), see above. These complications create a space occupying lesion.

Brain swelling (cerebral oedema) is a common and frequently fatal complication of head injury which may develop within minutes or hours of injury. Swelling may accompany diffuse axonal injury or a space-occupying lesion such as an intracranial haematoma. In children brain swelling may be the only identifiable feature of head injury. A swollen brain is heavy, with visible enlargement of the surface convolutions (gyri) at the expense of obliteration of the the intervening gaps (sulci) and compression of the fluid filled cavities (ventricles) deep within the brain. Emergency neurosurgical procedures frequently attempted include drainage of an ICH or removal of severely damaged brain tissue in an attempt to reduce intracranial pressure. Brain swelling is frequently the fatal complication even after such measures.

Raised intracranial pressure (raised ICP): the skull is a rigid bony compartment with only one exit (the foramen magnum). Severe brain swelling or a large intracranial haemorrhage (a space-occupying lesion) may cause displacement of the brain tissue downwards towards the foramen magnum. The inner, lower edge of a cerebral hemisphere becomes compressed against the sharp edges of the dural folds (causing uncal necrosis) or can be actually forced out under the free edge of the dura (causing uncal herniation). The cerebellum is similarly squeezed down into the foramen magnum (causing tonsillar coning and necrosis). Pressure on the brain stem and secondary brain stem haemorrhage may occur and are fatal complications of raised ICP.

Meningitis and brain abscess: particularly common after penetrating (open) head injury and after fractures which disrupt the nasal and frontal air sinuses.

Post traumatic epilepsy: healing and scarring of the meninges and brain surface may be the focus of later epileptic fits.

Facial injury: common in accidents (RTA), assault by punching, kicking or blunt weapon. The fragile facial bones are susceptible to fracture. Bleeding from the nose, mouth and sinuses may obstruct the air passages.

Neck injury: a very vulnerable area with easy access to vital structures such as the trachea, large vessels (carotid arteries, jugular veins). Incisions, stabs, blows and manual pressure are very dangerous in this area. The cervical spine and spinal cord are also vulnerable. Sensitive nervous conections and reflexes can be stimulated with fatal results (vagal reflex).