Head Injury

- The most common cause of death in young adults.
- Males > Females
- RTA is the commonest cause.
- Severity of head injury is classified according to the post resuscitation Glasgow Coma Scale (GCS), as it is the GCS score, and in particular the motor score, that is the best predictor of neurological outcome.

- Severity of head injury
  - Minor- GCS 13-15 (80%) 1% mortality
  - Moderate- GCS 9-12 (10%) 5% mortality
  - Severe- GCS <8 (10%) 40% mortality
- In broad terms, significantly obtunded patients have moderate injuries and comatose patients have severe injuries; alcohol and drug effects often complicate the classification.

MINOR AND MILD HEAD INJURY

- After exclusion of associated cervical spine injury, the major concern for these patients is to avoid discharge during the ‘lucid interval’ that may precede delayed deterioration due to an expanding intracranial haematoma.
- In general, patients with isolated head injuries and without ongoing deficits can safely be discharged from the emergency department, provided they meet suitable criteria.

- Patients who do not meet all the discharge criteria will need admission for a further period of observation, and/or brain imaging.
- Early computed tomography (CT) imaging is desirable in patients with a persistent reduced conscious level, focal deficits, suspected fractures or risk factors for intracranial bleed.
- Significant clinical or radiological abnormalities should be discussed with the neurosurgical service.
- Many of these patients will struggle with features of concussion for a period after their injury, with headaches and somnolence typical.
- Follow-up by a head injury specialist nurse or equivalent is therefore desirable.

Non-accidental injury

- Head injury in children and vulnerable adults may be due to abuse.
- Significant findings include delayed presentation, injuries of disparate age, retinal haemorrhages, bilateral chronic subdural haematomas, multiple skull fractures and neurological injury without external signs of trauma.

Concussion, second impact syndrome and postconcussive syndrome

- **Concussion** is defined as alteration of consciousness as a result of closed head injury, but is generally used in describing mild head injury without imaging abnormalities; loss of consciousness (LOC) at the time of injury is not a prerequisite.
- Key features include confusion and amnesia.

- The patient may be lethargic, easily distractable, forgetful, slow to interact or emotionally labile.
- Gait disturbance and incoordination may be seen.
- It is claimed that while symptomatic following a head injury, patients may be especially vulnerable to repeat impacts.
- It is proposed that in the context of disordered cerebral autoregulation, a second minor injury may trigger a form of malignant cerebral oedema refractory to treatment.
- Although the existence of the syndrome is disputed, and it is certainly rare, it should be considered in advice to individuals engaged in sports or activities carrying a risk of further injury: **symptomatic players should not return to play.**

- **Postconcussive syndrome** is a loosely defined constellation of symptoms, persisting for a prolonged period after injury, and exacerbated in some patients by the potential for secondary gain (compensation).
- Patients may report somatic features such as headache, dizziness and disorders of hearing and vision.
- They may also suffer a variety of neurocognitive and neuropsychological disturbances, including difficulty with concentration and recall, insomnia, emotional lability, fatigue, depression and personality change.

MODERATE AND SEVERE TRAUMATIC BRAIN INJURY

Resuscitation and evaluation

- Performed according to Advanced Trauma and Life Support (ATLS) guidelines, beginning with management of the airway and cervical spine control, and proceeding to assess and manage breathing and circulation.
- History obtained in parallel is key to shaping ongoing management.

History

- Bystanders and paramedics may give vital information on the:
  - preinjury state (fits, alcohol, chest pain)
  - mechanism and energy involved in the injury (speed of vehicles, height fallen)
  - conscious state and haemodynamic stability of the patient after the accident
  - length of time taken for extrication
- Check the medication history especially anticoagulants and antiplatelet agents.

Examination: primary survey

- ATLS guidelines address a fundamental priority, **ensuring uninterrupted perfusion of the brain with oxygenated blood**.
- This is especially important after a head injury given the disturbance to intracranial autoregulation and the sensitivity of the primary injured brain tissue to further insult.
- Bleeding from scalp lacerations may require management as part of the primary survey, as the blood loss can be substantial and ongoing.
- Check the responsiveness of the pupils, conscious level and for any gross focal neurological deficits.
- Blood glucose level should also be measured as early as possible as hypoglycaemia is very dangerous and easily reversible.

- The **pupil size** should be recorded in millimetres, and reactivity documented as present, sluggish or absent.
- Uncal herniation can compress the third nerve, compromising the parasympathetic supply to the pupil.
- Unopposed sympathetic activity produces a sluggish enlarged pupil, progressing to fixed and dilated under continued compression.
- Established pupil changes may reflect pathology anywhere in the eye or the reflex loop made up by the optic nerve, the oculomotor nerve and the brainstem.
- Direct ocular trauma or nerve injury in association with a skull base fracture can cause mydriasis (dilated pupil) present from the time of injury.
- Pre-existing discrepancy in pupil size (anisocoria), as a result of Holmes–Adie pupil or cataracts for example, may also complicate assessment.

- The **GCS** is the sum of scores on three components.
- The breakdown of the GCS into eye opening, verbal and motor components should always be recorded and used when communicating the status to other doctors.
- **Gross focal neurological deficits**, such as paraplegia, may be evident at the primary survey, and an assessment to exclude such deficit should be carried out, especially if the patient is to be intubated so that subsequent examination will be impossible.
- Detailed neurological examination is included in the secondary survey.

Examination: secondary survey

- A full secondary survey will be required. Particular attention must be paid to head, neck and spine.
- Examination of the head should include inspection and palpation of the scalp for evidence of subgaleal haematoma and scalp lacerations, which may bleed profusely, and potentially overlie fractures.
- Examine the face for evidence of fractures.
- Clinical evidence of a skull base fracture may include Battle's sign and 'racoon' or 'panda' eyes (bilateral periorbital bruising).
- A complete examination of the cranial nerves will reveal cranial nerve palsies due to underlying fractures.

- Cervical spine injury must be presumed in the context of head injury until actively excluded.
- These patients should be managed in a hard collar until the neck can be cleared clinically.
- A peripheral nerve examination with documentation of limb tone, power, reflexes and sensation needs to be performed early to identify spinal pathology.
- The patient will need to be log-rolled to palpate for thoracic or lumbar deformity, and any cervical collar should be removed at this stage to allow palpation of the cervical spine, before it is replaced.

- If there is associated spinal injury, a thoracic sensory level is much more easily established by sensory examination on the back.
- A per rectal examination is also performed at log-roll, assessing for anal tone, sensation in the awake patient and anal wink (sphincter seen to contract in response to a pinprick stimulus). Priapism is a strong predictor of severe cord injury even in intubated patients.

Surgical management

Fractures: skull vault

- Closed linear fractures of the skull vault are managed conservatively.
- Open or comminuted fractures should be considered for debridement and prophylactic antibiotic therapy.
- Depressed skull fractures require exploration and elevation, especially where intracranial air is present pointing to a breach in the dura mater.
- Fractures that involve the air sinuses should generally be managed as open fractures, using broad spectrum antibiotics with or without exploration.

Fractures: skull base

- Clinical signs of skull base fracture include bleeding or CSF leak from the ears (otorrhoea) or nose (rhinnorrhea), and bruising behind the ear or around the eyes.
- Skull base fractures may be complicated by pituitary dysfunction, arterial dissection or cranial nerve deficits, with anosmia, facial palsy or hearing loss typical.
- CSF leak will generally resolve spontaneously but persistent leak can result in meningitis so repair may be required.
- Blind nasogastric tube placement is contraindicated in these patients.

Extradural haematoma

- A neurosurgical emergency.
- It results from rupture of an artery, vein or venous sinus, in association with a skull fracture.
- The classical injury is a fracture to the thin squamous temporal bone, with associated damage to the middle meningeal artery.
- Transient loss of consciousness is typical, and the patient may then present in the subsequent lucid interval with headache but without any neurological deficit.
- As the haematoma expands, compensation is exhausted (see Monro Kellie doctrine above), with rapid deterioration.
- There is contralateral hemiparesis, reduced conscious level and ipsilateral pupillary dilatation, the cardinal signs of brain compression and herniation.

- On CT, appear as a lentiform (lens-shaped or biconvex) hyperdense lesion between skull and brain, constrained by the adherence of the dura to the skull.
- Mass effect may be evident, with compression of surrounding brain and midline shift.
- Areas of mixed density suggest active bleeding.
- A skull fracture will usually be evident.

- Extradural haematoma mandates urgent transfer to the most accessible neurosurgical facility, for immediate evacuation in deteriorating or comatose patients or those with large bleeds, and for close observation with serial imaging in other cases.
- The prognosis for promptly evacuated extradural haematoma, without associated primary brain injury, is excellent.

Acute subdural haematoma

- Encountered in two broadly distinct contexts.
- Firstly, high-energy injury mechanisms can result in the rupture of cortical surface vessels with significant associated primary brain injury.
- This results in expanding haematoma with rapid deterioration and developing signs of raised ICP, reminiscent of extradural haematoma without the lucid interval.
- These collections require prompt evacuation, typically by craniotomy or craniectomy.

- In a second group of patients, older and often anticoagulated, a lower-energy injury leads to venous bleeding around the brain.
- Depending on the total volume of bleeding, the resulting haematoma may present early as acute subdural haematoma, after delay and osmotic expansion as chronic subdural haematoma or may even remain clinically silent.
- This latter group may present much later with a further ‘acute-onchronic’ subdural haematoma.
- On diagnosis, clotting function should be corrected wherever possible.
- Bleeds of significant size, with significant associated midline shift or with deteriorating neurology, require urgent evacuation.
- Smaller bleeds in neurologically stable patients may be managed conservatively, at least initially: liquefaction of the clot over 7–10 days after the bleed may allow for a much less invasive evacuation through burr holes.

Chronic subdural haematoma

- A common cause of acute neurological deterioration in the elderly.
- Cerebral atrophy in this age group results in stretching of cortical–dural bridging veins, which are then vulnerable to rupture.
- The resulting haematoma can expand over days or weeks by osmosis, ultimately producing symptoms of raised ICP or focal deficits.
- There is usually a history of recent injury, but especially in the context of antiplatelet or anticoagulant medication even apparently trivial impacts may be responsible.

- Imaging reveals diffuse hypodensity overlying the brain surface.
- Recent bleeding may be isodense or hyperdense, and mixed density can indicate an acute-onchronic subdural haematoma.
- Anticoagulation should be reversed, either by administration of vitamin K, or urgently by transfusion of recombinant clotting factors in patients who have deteriorated acutely.
- Conservative management, sometimes with administration of corticosteroids, can be considered for small bleeds without symptoms or with headache alone.
- For the majority, drainage is performed using burr holes.

Traumatic subarachnoid haemorrhage

- Trauma is the commonest cause of subarachnoid haemorrhage, and this is managed conservatively.
- It is not usually associated with significant vasospasm, which characterises aneurysmal subarachnoid haemorrhage.
- The possibility of spontaneous subarachnoid haemorrhage leading to collapse and so causing a head injury needs to be borne in mind, and formal or CT angiography may be required to exclude this.

Cerebral contusions

- Common and are found predominantly where brain is in contact with the irregularly ridged inside of the skull, i.e. at the inferior frontal lobes and temporal poles.
- ‘Coup contre-coup’ contusions are brain injury at the site of impact and where the brain is struck by the inside of the skull on the far side, as the skull and brain accelerate and then decelerate out of synchrony with each other.
- Contusions appear heterogenous on CT, reflecting their composition of injured brain matter interspersed with acute blood.
- Contusions rarely require surgical intervention, but may warrant delayed evacuation to reduce mass effect.

Diffuse axonal injury

- A form of primary brain injury, seen in high-energy accidents, and which usually renders the patient comatose.
- It is strictly a pathological diagnosis made at postmortem, but haemorrhagic foci in the corpus callosum and dorsolateral rostral brainstem on CT may be suggestive, although the CT often appears normal.

Arterial dissection

- Occurs spontaneously or in the context of trauma.
- Presents with headache, neck pain and focal ischaemic deficits, due to occlusion by mural haematoma, thrombus and thromboembolism.

Medical Management

- First-line ICP control involves optimising sedation, ventilation and serum sodium levels.
- Paralysis and external ventricular CSF drainage are important adjuncts.
- There is little evidence for benefit with therapeutic hypothermia, barbiturate coma or decompressive craniectomy.
- Check pituitary function, consider seizure prophylaxis, commence enteral nutrition within 72 hours.

The role of neurosurgical centres

- Early discussion of patients and imaging with the regional neurosurgical service is advisable.

Control of intracranial pressure

- Intubation and ventilation is required early in the management of severe brain injury for airway control.
- It is often required in moderate brain injury to facilitate the safe management and transfer of unstable and frequently agitated patients and in order to control ICP.
- Where there is evidence of raised ICP, for example pupil changes, a bolus of mannitol may be administered to control pressure temporarily while scanning and transferring the patient.

- Management of the intubated patient, following evacuation of any focal haematomas, is guided by ICP monitoring using a bolt ICP monitor, or else an external ventricular drain inserted into the lateral ventricle, which can also contribute to ICP control by permitting CSF drainage.
- ICP can be controlled by simple measures including raising the head of the bed and loosening the collar to improve venous drainage.
- Seizures and pyrexia should be actively controlled.
- Medical management titrated to ICP includes escalating doses of sedatives, analgesics and ultimately muscle relaxants.

Pituitary dysfunction

- Electrolyte imbalance contributes to brain swelling and to causing seizures.
- Cerebral salt wasting, a poorly understood form of excretory dysregulation in association with brain insult, leads to volume depletion and hyponatraemia.
- The syndrome of inappropriate antidiuretic hormone (SIADH) leads to a water retention and hyponatraemia in the context of pituitary damage.
- This is of particular concern in head injury since low serum osmotic pressure can contribute to brain swelling, so hypotonic fluids are avoided in this setting.
- Conversely, ADH secretion may be compromised in the context of trauma, producing diabetes insipidus resulting in hypernatraemia.

- Routine screening of pituitary hormone levels and liaison with endocrinology is an important aspect of optimal medical management.
- Note that routine, rather than directed, administration of corticosteroids in severe head injury is associated with increased mortality and is not recommended.

Nutrition

- Enteral nutrition is preferred to intravenous parenteral nutrition on grounds of cost and associated complications, and should be commenced within 72 hours of injury.
- Prokinetics (e.g. metaclopramide, erythromycin) can be administered to promote absorption.

Outcomes and sequelae

- The long-term sequelae of moderate and severe traumatic brain injury include headache, memory and cognitive impairments, contributing to the postconcussive syndrome described above.
- Rehabilitation represents a complex and prolonged multidisciplinary challenge.
- Good recovery implies independence and potential to return to work rather than a full return to previous capacity.