mechanisms of traumatic brain injury [created by Paul Young 10/11/07]

general

general:
- trauma to the head causes primary injury such as skull fracture, cerebral contusion, and haemorrhage that is a direct consequence of the traumatic incident
- secondary injury occurs hours or days after the injury and may be the major determinant of the patient's ultimate neurological outcome

primary injury:
(i) contact forces
- produce focal injuries such as skull fractures, contusions and extra- or subdural haematomas

(ii) inertial forces:
- result from the brain undergoing acceleration or deceleration and can occur without head impact
- inertial forces can produce focal or diffuse brain injuries: pure translational acceleration leads to focal injuries such as contrecoup contusions, intracerebral haematomas and subdural haematomas, whereas, rotational or angular acceleration, common with motor vehicle accidents leads to diffuse injuries

secondary injury:
- post traumatic ischaemia initiates a cascade of metabolic events that lead to the surplus production of oxygen free radicles, excitatory amino acids, cytokines & other inflammatory agents
- post traumatic non-ischaemic events such as increase in intracellular free calcium via receptor gated or voltage gated ion channels induce release of oxygen free radicals from the mitochondria
- excessive production of oxygen free radicals causes lipid peroxidation of cell membranes, oxidation of intracellular proteins and nucleic acids and activation of phospholipases A2 and C which hydrolyse membrane phospholipids releasing arachidonic acid which generate free fatty acids, leukotriennes and thromboxane B2 all of which are associated with neurodegeneration and poor outcome after experimental traumatic brain injury
- TBI also increase intracellular potassium levels leading to an imbalance of intracellular and extracellular potassium, disruption of the Na+/K+ ATPase cell membrane regulatory mechanisms and subsequent cell swelling
- severe TBI also causes a substantial decrease in extracellular magnesium which impairs normal glycolysis, cellular respiration and oxydative phosphorylation contributing to brain injury

Systemic	Intracranial
Hypoxia	Seizure
Hypotension	Delayed haematoma
Hypocapnia	Subarachnoid haemorrhage
Hypercapnia	Vasospasm
Hyperthermia	Hydrocephalus
Hypoglycaemia	Neuroinfection
Hyperglycaemia	
Hyponatraemia	
Hypernatraemia	
Hyperosmolality	
Infection	

epidemiology

- traumatic brain injury is the leading cause for morbidity and mortality among people aged 1 to 45 years
- motor vehicle crashes are the major cause of head injuries among those aged 5 to 64 years old while falls are the major cause among over 65s
- males have twice the risk of traumatic brain injury across all age groups

skull fracture

- results from a contact force to the head that is usually severe enough to cause a brief loss of consciousness
- linear fractures are the most common and typically occur over the lateral convexities of the skull (most often they are non-displaced)
- a depressed skull fracture usually results from a blunt force from an object with a small surface area such as a hammer
- the base of the skull can be fractured by severe blunt trauma to the forehead or the occiput.

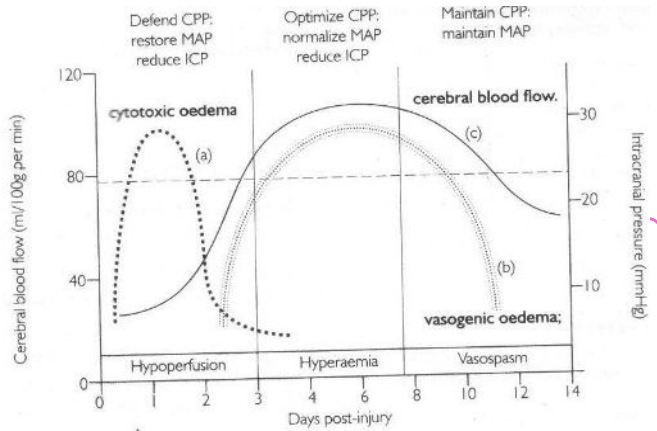
examples of injuries associated with skull fracture:
(i) anterior skull base fractures most often involve the cribriform plate disrupting the olfactory nerves.
(ii) Posterior skull base fractures may extend through the petrous bone and internal auditory canal thereby damaging the acoustic and facial nerves
(iii) fractures of the squamous temporal bone are frequently accompanied by a tear in the middle meningeal artery causing an extradural haematoma
(iv) depressed skull fractures are often accompanied by cerebral contusion
(v) dura is often disrupted with basilar skull fracture resulting in CSF leak from the nose or ear which may allow bacteria to enter the intracranial space

subdural haematoma

- seen in 20-25% of all comatose victims of traumatic brain injury
- develop between the surface of the brain and the inner surface of the dura and are believed to result from the tearing of the bridging veins over the cortical surface or from disruption of major venous sinuses or their tributaries
- typically spread over most of the cerebral convexity with the dural reflections of the falx cerebri preventing expansion to the contralateral hemisphere
- swelling of the cerebral hemisphere is common due to damage to the underlying brain tissue; cerebral contusions are found in 2/3rds

- classified as acute, subacute and chronic with each having a characteristic CT appearance
- acute subdural appears white, subacute lesions are isodense and chronic lesions are hypodense

aetiology of secondary brain swelling



diffuse axonal injury

- refers to lacerations or punctate contusions at the interface between the gray and white matter; such punctate contusions are thought to result from the disparate densities of the grey and white matter and the consequent difference in centripetal force assoicated with a rotational vector of injury
- occurs most commonly after a high speed motor vehicle accident in which severe angular and rotational forces are applied to the head
- present in almost 50% of patients with severe TBI and in almost a third of those who die
- a common cause of persistent vegatative state or prolonged coma

cerebral contusions

- contusions are heterogenous lesions comprising punctate haemorrhage, oedema & necrosis and often associated with other intracranial lesions
- one or more contusions occur in 20-25% of patients with severe TBI
- contusions are most common in the inferior frontal cortex and the anterior temporal lobes where the surface of the inner table of the skull is very irregular; they may result from shifting of the of the brain over this irregular surface at the time of impact
- direct blunt force trauma to the head can produce a contusion in the tissue underlying the point of impact (coup contusion); if the head was in motion upon collision with a rigid surface, a contusion may occur in the brain contralateral to the point of impact
- because they evolve over time contusions may not be visible on initial CT scan
- local neuronal damage & haemorrhage lead to oedema which may increase over the next 24-48 hours
- depending on their size and location, they may cause significant mass effect resulting in midline shift, transtentorial or subfalcine herniation
- initial signs and symptoms vary greatly depending on the size and location:
(i) small contusions may cause no symptoms or only mild headache
(ii) contusions in eloquent area of the brain such as speech or motor areas may cause focal deficits
(iii) large contusions, especially frontal ones, cause elevated ICP and coma

subarachnoid haemorrhage

- traumatic subarachnoid haemorrhage does not produce a haematoma or mass effect
- it may be associated with post-traumatic vasospasm

intraparenchymal haematomas

- a haemorrhage within the brain substance that occurs after a very severe TBI & is usually associated with contusions of the surrounding tissue
- Duret's haemorrhage is a haemorrhage into the base of the pons or midbrain thought to result from disruption of the perforating arteries at the time of uncal herniation. Such brainstem haemorrhage almost always leads to death or vegetative survival

epidural haematoma

- develop between the inner table of the skull and the dura, usually when the middle meningeal artery or one its branches is torn by a skull fracture
- occur in 8-10% of those rendered comatose by traumatic brain injury
- the majority are located in the temporal or parietal regions but they can also occur over the frontal and occipital lobes and rarely in the posterior fossa
- unlike subdural haematomas their spread is limited by suture lines of the skull where the dura is very adherent
- an epidural space does not usually exist so the clot must strip the dura from the inner table of the skull as it enlarges resulting in the classic lenticular shape
- epidural haematomas are uncommon in infants and toddlers presumably because their skulls are more deformable and less likely to fracture and in patients older than 60 because the dura is extremely adherent to the skull
- the classically described lucid interval after initial period of loss of consciousness followed by coma occurs in fewer than a third of patients with most either remaining comatose or remaining conscious after the initial injury