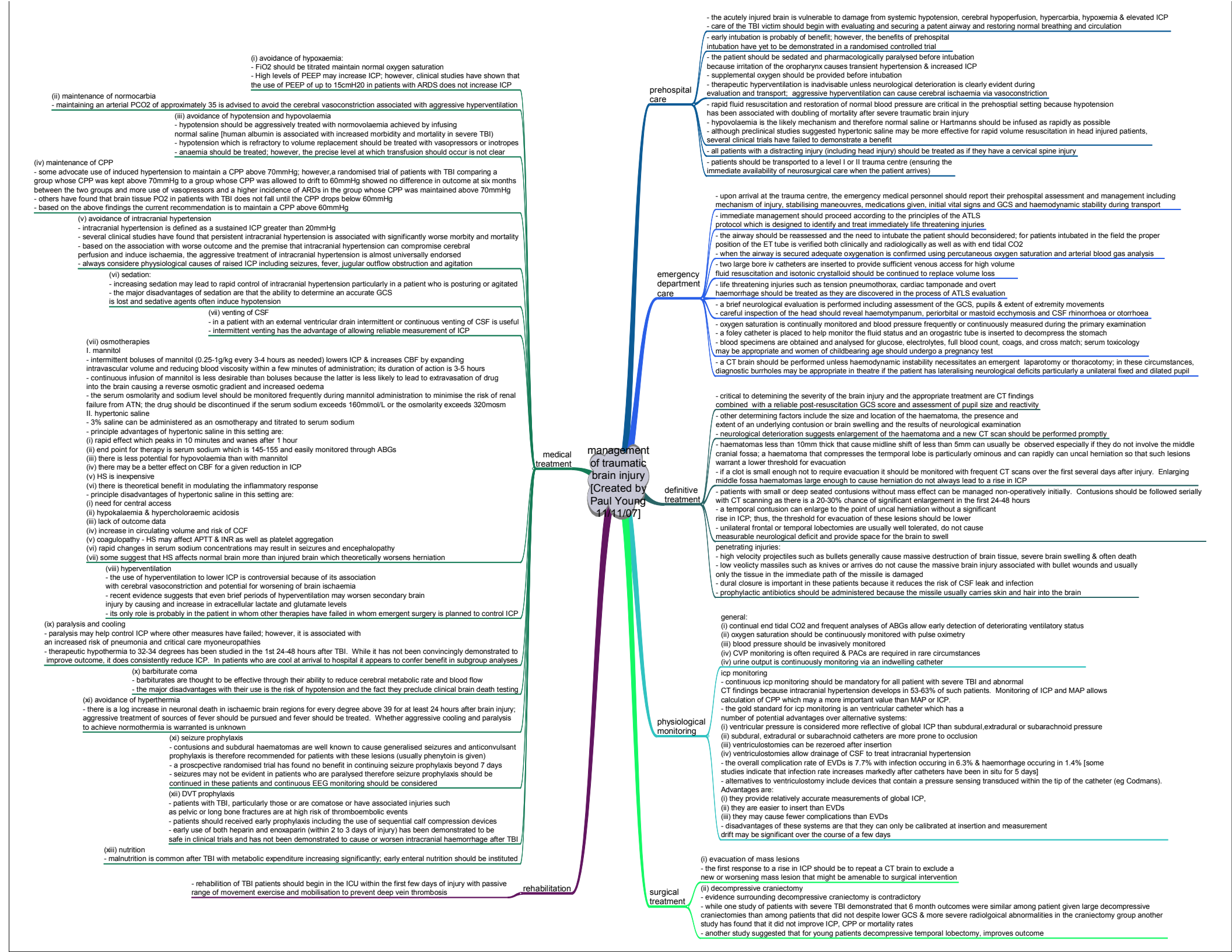
management of traumatic brain injury [Created by Paul Young 11/11/07]
prehospital care
- the acutely injured brain is vulnerable to damage from systemic hypotension, cerebral hypoperfusion, hypercarbia, hypoxemia & elevated ICP
- care of the TBI victim should begin with evaluating and securing a patent airway and restoring normal breathing and circulation
- early intubation is probably of benefit; however, the benefits of prehospital intubation have yet to be demonstrated in a randomised controlled trial
- the patient should be sedated and pharmacologically paralysed before intubation because irritation of the oropharynx causes transient hypertension & increased ICP
- supplemental oxygen should be provided before intubation
- therapeutic hyperventilation is inadvisable unless neurological deterioration is clearly evident during evaluation and transport; aggressive hyperventilation can cause cerebral ischaemia via vasoconstriction
- rapid fluid resuscitation and restoration of normal blood pressure are critical in the prehosptial setting because hypotension has been associated with doubling of mortality after severe traumatic brain injury
- hypovolaemia is the likely mechanism and therefore normal saline or Hartmanns should be infused as rapidly as possible
- although preclinical studies suggested hypertonic saline may be more effective for rapid volume resuscitation in head injured patients, several clinical trials have failed to demonstrate a benefit
- all patients with a distracting injury (including head injury) should be treated as if they have a cervical spine injury
- patients should be transported to a level I or II trauma centre (ensuring the immediate availability of neurosurgical care when the patient arrives)
emergency department care
- upon arrival at the trauma centre, the emergency medical personnel should report their prehospital assessment and management including mechanism of injury, stabilising maneouvres, medications given, initial vital signs and GCS and haemodynamic stability during transport
- immediate management should proceed according to the principles of the ATLS protocol which is designed to identify and treat immediately life threatening injuries
- the airway should be reassessed and the need to intubate the patient should beconsidered; for patients intubated in the field the proper position of the ET tube is verified both clinically and radiologically as well as with end tidal CO2
- when the airway is secured adequate oxygenation is confirmed using percutaneous oxygen saturation and arterial blood gas analysis
- two large bore iv catheters are inserted to provide sufficient venous access for high volume fluid resuscitation and isotonic crystalloid should be continued to replace volume loss
- life threatening injuries such as tension pneumothorax, cardiac tamponade and overt haemorrhage should be treated as they are discovered in the process of ATLS evaluation
- a brief neurological evaluation is performed including assessment of the GCS, pupils & extent of extremity movements
- careful inspection of the head should reveal haemotympanum, periorbital or mastoid ecchymosis and CSF rhinorrhoea or otorrhoea
- oxygen saturation is continually monitored and blood pressure frequently or continuously measured during the primary examination
- a foley catheter is placed to help monitor the fluid status and an orogastric tube is inserted to decompress the stomach
- blood specimens are obtained and analysed for glucose, electrolytes, full blood count, coags, and cross match; serum toxicology may be appropriate and women of childbearing age should undergo a pregnancy test
- a CT brain should be performed unless haemodynamic instability necessitates an emergent laparotomy or thoracotomy; in these circumstances, diagnostic burrholes may be appropriate in theatre if the patient has lateralising neurological deficits particularly a unilateral fixed and dilated pupil
definitive treatment
- critical to detemining the severity of the brain injury and the appropriate treatment are CT findings combined with a reliable post-resuscitation GCS score and assessment of pupil size and reactivity
- other determining factors include the size and location of the haematoma, the presence and extent of an underlying contusion or brain swelling and the results of neurological examination
- neurological deterioration suggests enlargement of the haematoma and a new CT scan should be performed promptly
- haematomas less than 10mm thick that cause midline shift of less than 5mm can usually be observed especially if they do not involve the middle cranial fossa; a haematoma that compresses the termporal lobe is particularly ominous and can rapidly can uncal herniation so that such lesions warrant a lower threshold for evacuation
- if a clot is small enough not to require evacuation it should be monitored with frequent CT scans over the first several days after injury. Enlarging middle fossa haematomas large enough to cause herniation do not always lead to a rise in ICP
- patients with small or deep seated contusions without mass effect can be managed non-operatively initially. Contusions should be followed serially with CT scanning as there is a 20-30% chance of significant enlargement in the first 24-48 hours
- a temporal contusion can enlarge to the point of uncal herniation without a significant rise in ICP; thus, the threshold for evacuation of these lesions should be lower
- unilateral frontal or temporal lobectomies are usually well tolerated, do not cause measurable neurological deficit and provide space for the brain to swell
penetrating injuries:
- high velocity projectiles such as bullets generally cause massive destruction of brain tissue, severe brain swelling & often death
- low veolicty massiles such as knives or arrives do not cause the massive brain injury associated with bullet wounds and usually only the tissue in the immediate path of the missile is damaged
- dural closure is important in these patients because it reduces the risk of CSF leak and infection
- prophylactic antibiotics should be administered because the missile usually carries skin and hair into the brain
physiological monitoring
general:
(i) continual end tidal CO2 and frequent analyses of ABGs allow early detection of deteriorating ventilatory status
(ii) oxygen saturation should be continuously monitored with pulse oximetry
(iii) blood pressure should be invasively monitored
(iv) CVP monitoring is often required & PACs are required in rare circumstances
(iv) urine output is continuously monitoring via an indwelling catheter
icp monitoring
- continuous icp monitoring should be mandatory for all patient with severe TBI and abnormal CT findings because intracranial hypertension develops in 53-63% of such patients. Monitoring of ICP and MAP allows calculation of CPP which may a more important value than MAP or ICP.
- the gold standard for icp monitoring is an ventricular catheter which has a number of potential advantages over alternative systems:
(i) ventricular pressure is considered more reflective of global ICP than subdural,extradural or subarachnoid pressure
(ii) subdural, extradural or subarachnoid catheters are more prone to occlusion
(iii) ventriculostomies can be rezeroed after insertion
(iv) ventriculostomies allow drainage of CSF to treat intracranial hypertension
- the overall complication rate of EVDs is 7.7% with infection occuring in 6.3% & haemorrhage occuring in 1.4% [some studies indicate that infection rate increases markedly after catheters have been in situ for 5 days]
- alternatives to ventriculostomy include devices that contain a pressure sensing transduced within the tip of the catheter (eg Codmans). Advantages are:
(i) they provide relatively accurate measurements of global ICP,
(ii) they are easier to insert than EVDs
(iii) they may cause fewer complications than EVDs
- disadvantages of these systems are that they can only be calibrated at insertion and measurement drift may be significant over the course of a few days
surgical treatment
(i) evacuation of mass lesions
- the first response to a rise in ICP should be to repeat a CT brain to exclude a new or worsening mass lesion that might be amenable to surgical intervention
(ii) decompressive craniectomy
- evidence surrounding decompressive craniectomy is contradictory
- while one study of patients with severe TBI demonstrated that 6 month outcomes were similar among patient given large decompressive craniectomies than among patients that did not despite lower GCS & more severe radiolgoical abnormalities in the craniectomy group another study has found that it did not improve ICP, CPP or mortality rates
- another study suggested that for young patients decompressive temporal lobectomy, improves outcome
medical treatment
(i) avoidance of hypoxaemia:
- FiO2 should be titrated maintain normal oxygen saturation
- High levels of PEEP may increase ICP; however, clinical studies have shown that the use of PEEP of up to 15cmH20 in patients with ARDS does not increase ICP
(ii) maintenance of normocarbia
- maintaining an arterial PCO2 of approximately 35 is advised to avoid the cerebral vasoconstriction associated with aggressive hyperventilation
(iii) avoidance of hypotension and hypovolaemia
- hypotension should be aggressively treated with normovolaemia achieved by infusing normal saline [human albumin is associated with increased morbidity and mortality in severe TBI)
- hypotension which is refractory to volume replacement should be treated with vasopressors or inotropes
- anaemia should be treated; however, the precise level at which transfusion should occur is not clear
(iv) maintenance of CPP
- some advocate use of induced hypertension to maintain a CPP above 70mmHg; however,a randomised trial of patients with TBI comparing a group whose CPP was kept above 70mmHg to a group whose CPP was allowed to drift to 60mmHg showed no difference in outcome at six months between the two groups and more use of vasopressors and a higher incidence of ARDs in the group whose CPP was maintained above 70mmHg
- others have found that brain tissue PO2 in patients with TBI does not fall until the CPP drops below 60mmHg
- based on the above findings the current recommendation is to maintain a CPP above 60mmHg
(v) avoidance of intracranial hypertension
- intracranial hypertension is defined as a sustained ICP greater than 20mmHg
- several clinical studies have found that persistent intracranial hypertension is associated with significantly worse morbity and mortality
- based on the association with worse outcome and the premise that intracranial hypertension can compromise cerebral perfusion and induce ischaemia, the aggressive treatment of intracranial hypertension is almost universally endorsed
- always considere phyysiological causes of raised ICP including seizures, fever, jugular outflow obstruction and agitation
(vi) sedation:
- increasing sedation may lead to rapid control of intracranial hypertension particularly in a patient who is posturing or agitated
- the major disadvantages of sedation are that the ability to determine an accurate GCS is lost and sedative agents often induce hypotension
(vii) venting of CSF
- in a patient with an external ventricular drain intermittent or continuous venting of CSF is useful
- intermittent venting has the advantage of allowing reliable measurement of ICP
(vii) osmotherapies
I. mannitol
- intermittent boluses of mannitol (0.25-1g/kg every 3-4 hours as needed) lowers ICP & increases CBF by expanding intravascular volume and reducing blood viscosity within a few minutes of administration; its duration of action is 3-5 hours
- continuous infusion of mannitol is less desirable than boluses because the latter is less likely to lead to extravasation of drug into the brain causing a reverse osmotic gradient and increased oedema
- the serum osmolarity and sodium level should be monitored frequently during mannitol administration to minimise the risk of renal failure from ATN; the drug should be discontinued if the serum sodium exceeds 160mmol/L or the osmolarity exceeds 320mosm
II. hypertonic saline
- 3% saline can be administered as an osmotherapy and titrated to serum sodium
- principle advantages of hypertonic saline in this setting are:
(i) rapid effect which peaks in 10 minutes and wanes after 1 hour
(ii) end point for therapy is serum sodium which is 145-155 and easily monitored through ABGs
(iii) there is less potential for hypovolaemia than with mannitol
(iv) there may be a better effect on CBF for a given reduction in ICP
(v) HS is inexpensive
(vi) there is theoretical benefit in modulating the inflammatory response
- principle disadvantages of hypertonic saline in this setting are:
(i) need for central access
(ii) hypokalaemia & hypercholoraemic acidosis
(iii) lack of outcome data
(iv) increase in circulating volume and risk of CCF
(v) coagulopathy - HS may affect APTT & INR as well as platelet aggregation
(vi) rapid changes in serum sodium concentrations may result in seizures and encephalopathy
(vii) some suggest that HS affects normal brain more than injured brain which theoretically worsens herniation
(viii) hyperventilation
- the use of hyperventilation to lower ICP is controversial because of its association with cerebral vasoconstriction and potential for worsening of brain ischaemia
- recent evidence suggests that even brief periods of hyperventilation may worsen secondary brain injury by causing and increase in extracellular lactate and glutamate levels
- its only role is probably in the patient in whom other therapies have failed in whom emergent surgery is planned to control ICP
(ix) paralysis and cooling
- paralysis may help control ICP where other measures have failed; however, it is associated with an increased risk of pneumonia and critical care myoneuropathies
- therapeutic hypothermia to 32-34 degrees has been studied in the 1st 24-48 hours after TBI. While it has not been convincingly demonstrated to improve outcome, it does consistently reduce ICP. In patients who are cool at arrival to hospital it appears to confer benefit in subgroup analyses
(x) barbiturate coma
- barbiturates are thought to be effective through their ability to reduce cerebral metabolic rate and blood flow
- the major disadvantages with their use is the risk of hypotension and the fact they preclude clinical brain death testing
(xi) avoidance of hyperthermia
- there is a log increase in neuronal death in ischaemic brain regions for every degree above 39 for at least 24 hours after brain injury; aggressive treatment of sources of fever should be pursued and fever should be treated. Whether aggressive cooling and paralysis to achieve normothermia is warranted is unknown
(xi) seizure prophylaxis
- contusions and subdural haematomas are well known to cause generalised seizures and anticonvulsant prophylaxis is therefore recommended for patients with these lesions (usually phenytoin is given)
- a proscpective randomised trial has found no benefit in continuing seizure prophylaxis beyond 7 days
- seizures may not be evident in patients who are paralysed therefore seizure prophylaxis should be continued in these patients and continuous EEG monitoring should be considered
(xii) DVT prophylaxis
- patients with TBI, particularly those or are comatose or have associated injuries such as pelvic or long bone fractures are at high risk of thromboembolic events
- patients should received early prophylaxis including the use of sequential calf compression devices
- early use of both heparin and enoxaparin (within 2 to 3 days of injury) has been demonstrated to be safe in clinical trials and has not been demonstrated to cause or worsen intracranial haemorrhage after TBI
(xiii) nutrition
- malnutrition is common after TBI with metabolic expenditure increasing significantly; early enteral nutrition should be instituted
rehabilitation
- rehabilition of TBI patients should begin in the ICU within the first few days of injury with passive range of movement exercise and mobilisation to prevent deep vein thrombosis