Management of Severe Traumatic Brain Injury

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Traumatic brain injury is an important cause of death and disability in young adults, causing 15-20% of deaths in the age group 5-35 year olds. In the UK 1 million people present with an acute head injury each year with men three times as likely as women to suffer such an injury. Frequently alcohol and drugs are involved in the mechanism of these injuries and should be considered at the time of presentation of hospital.

Primary brain injury occurs at the time of impact and is related to the forces applied to the brain. Of these injuries **extradural haematomas**, whilst uncommon, are rapidly fatal as the haematoma, the result of a tear in a dural artery (often middle meningeal artery), expands rapidly. Extradural haematomas have a good prognosis if evacuated as an emergency and such patients should be transferred to a neurosurgical centre immediately. **Subdural haematomas** are found in 30% of severe head injuries and carry a poorer prognosis than extradural haematomas due to the associated brain injury.

Skull fractures are common and, whilst not a cause of disability in themselves, are associated with brain injury and bleeding. Generally depressed skull fractures are elevated if the depth of depression of the bone is more than the thickness of the skull. Base of skull fractures can be difficult to diagnose radiologically and so recognition of the clinical signs (cerebrospinal fluid otorrhoea, haemotympanum, periorbital bruising and bruised mastoid) is important.

Secondary brain injury occurs following the initial impact in the area of the primary injury and the surrounding region. At a cellular level the injury is followed by an increase in glutamate and excitatory amino acids, an increase in intracellular calcium, release of free radicals leading to vasodilation, cell dysfunction and ultimately cerebral oedema with loss of cerebral blood flow autoregulation. Many studies have demonstrated that the extent of this secondary brain injury can be reduced by avoiding hypoxia, systemic hypotension and raised intracranial pressure (ICP). Hence the management of severe traumatic brain injury, both immediately in A+E and later in ITU, is aimed at limiting these factors.

Immediate management of a patient with severe traumatic brain injury in A+E

Airway	GCS < 8	Secure airway with endotracheal intubation	Rapid sequence induction with suxamethonium (transient increase in ICP outweighs benefit of securing the airway)
Breathing	PaO2 > 13 kPa	Oxygen via face mask	Slight hyper-oxygenation may improve delivery of oxygen in ischaemic or threatened areas of brain tissue
	PaCO2 4.5 – 5.0kPa	Control rate and VTe.	Hypercapnia causes cerebral vasodilation and therefore raised ICP. Hyperventilation causes vasoconstriction, loss of perfusion and therefore ischaemia.
	PEEP = 5cm H2O		Moderate levels of PEEP have no impact on ICP. If FiO2 is > 60% or if significant airway collapse, increase PEEP but monitor PaCO2 carefully
Circulation	MAP > 90 mmHg	Intravenous access Consider arterial/ central access early Fluid resuscitation	Avoid glucose containing fluids as blood glucose > 10 mmol/l is associated with a poor outcome. Consider inotrope (noradrenaline) if hypotension persists despite fluid resuscitation and exclusion of other injuries
	Orogastric tube		

Indications for intubation

IMMEDIATE INDICATIONS	BEFORE TRANSFER
GSC < 8	Decreasing GCS (fall of > 2 in an hour)
PaO2 < 9 in air	Potential airway compromise
PaCo2 > 6	Seizure
Spontaneous tachypnoea (PaCO2 < 3.5 kPa)	
Associated chest injury	

Management of a patient with severe traumatic brain injury in the general ICU

	Aim	Method	
Cardiovascular	MAP = 90mmHg	1. Identify and treat any haemorrhage 2. Adequate filling (aim for normovolaemia), using: a. Enteral feed as appropriate b. Normal saline c. Colloids (or blood, see below) 3. Noradrenaline	Optimal cerebral perfusion pressure (CPP = MAP – ICP) is > 60mmHg. In the absence of ICP monitoring, a MAP of 90 allows for an ICP up to 30 without compromising CPP Noradrenaline is the inotrope of first choice
	Haematocrit = 30%	Blood products as required	
	Temperature < 37.5	Paracetamol. Surface cooling. NSAIDS if resistant. Treatment of infections. Do not actively warm hypothermic patients unless temp < 32.0 (and then only to 33.0) unless coagulopathy is a concern	Raised temperature is detrimental to patients with raised ICP. Evidence for the benefits of elective hypothermia is conflicting. Active warming may compromise haemodynamic stability
Respiratory	PaO2 > 13kPa		Slight hyper-oxygenation may improve delivery of oxygen in ischaemic or threatened areas of brain tissue

	PaCO2 4.5 – 5.0kPa	Control rate and VTe.	Hypercapnia causes cerebral vasodilation and therefore raised ICP. Hyperventilation causes vasoconstriction, loss of perfusion and therefore ischaemia. Pressure-controlled modes will reduce intra-thoracic pressure but may produce erratic VTe and therefore swings in PaCO2. Volume-controlled modes give better CO2 control but raise airway pressures. Choice should be individualised to the patient.
	PEEP = 5cm H2O		Moderate levels of PEEP have no impact on ICP. If FiO2 is > 60% or if significant airway collapse, increase PEEP but monitor PaCO2 carefully
Abdominal	Early feeding	Oro-gastric tube Enteral feed at 1500kcal / 24 hrs until seen by dietitian	Naso-gastric tubes should only be used if a base-of-skull fracture has been definitively excluded. Early feeding is associated with improved outcome. Prompt dietetic assessment is essential.
	Stress ulcer prophylaxis	Until feeding established, H2 receptor inhibitor	
	Normo-glycaemia	Insulin infusion if required for blood glucose < 10 mmol/l	Hyperglycaemia is associated with poor outcome following head injury. No additional benefit of tight glycaemic control below 10 mmol/l
	Avoid constipation	Prompt use of aperients	

Neurology	Sedation	1. Sedate until the patient does not respond to pain 2. Daily sedation hold to assess neurology. 3. Avoid paralysing agents unless necessary for ventilation	Inadequate sedation will cause a rise in ICP. Generally severe head injuries (GCS < 8) are sedated for 48 hours following injury. Propofol is preferred but may entail additional inotropic support. An opiate should be included. Duration of hold will depend on agents used. Resume sedation if the patient localises or if ventilation is compromised, or if no improvement in GCS after 1 hour of hold
	Spinal clearance	Clear cervical and thoraco-lumbar spine according to local protocol	Continue with spinal precautions until spine cleared
	Reduce raised ICP	1. Position 30° head up (if spine cleared) 2. Avoid neck rotation/flexion/extension	Head elevation promotes venous drainage. Elevation above 30° may compromise cerebral arterial pressure
	Seizure control	Treat if patient has more than one seizure. Phenytoin load and regular dose	
Micobiological	Prevent HAIs	Careful infection control measures	Neurosurgical patients are particularly prone to Staphylococcal infections
	Treat infections	Local antibiotic policy for infection	Routine use of prophylactic antibiotics for base of skull fracture are not currently advised
Haematological	Coagulation	Normal clotting profile	
		Prevent DVT	Anti-coagulants are contra-indicated in cases of intracranial haemorrhage of any sort, or if the patient may need urgent neurosurgery. Anti-embolic stockings *and* pneumatic compression boots should be used

Please send any comments on this draft to chris.brunker@stgeorges.nhs.uk. Thank you.