Factors Influencing Severe Head Injury in the ICU

Intracranial problems:

- Hematoma slowly developing hours after the first CT in ED
- Seizures which may not be obvious because of sedation or paralysis
  Phenytoin is only recommended in patients with a destructive parenchymal lesion; and it should continue for 10 days. This will NOT decrease the chance that you will go on to develop a late post-traumatic seizure disorder
- Subarachnoid hemorrhage
- Vasospasm as a result of the SAH
- Hydrocephalus as a result of SAH
- Infection meningitis, encephalitis, ventriculitis

Extracranial influences

- Hypoxia = neuronal death, …maintain a PaO$_2$ of at least 100!
- Hypotension cerebral hypoperfusion = neuronal death
- Hypocapnia = vasospasm
- Hypercapnia = vasodilation = increased ICP…maintain PaCO$_2$ of 35-40!

- Hyperthermia = increased cerebral metabolic demand
- Hypoglycaemia = neuronal death, +/- seizure
- Hyperglycaemia = shown to be associated with poorer outcome
- Hyponatremia
- Hypernatremia
- Hyperosmolality
- Systemic sepsis
Chronology of Cerebral Perfusion in Severe Head Injury

Hypoperfusion phase
- In the first 72 hrs, autoregulation is severely impaired
- THUS, cerebral blood flow is directly dependent on MAP
- One must manipulate MAP to achieve a CPP of about 60-70
- Cytototoxic oedema is due to the ischaemic neurons dying
- There is good evidence: **START INOTROPES EARLY** in the hypoperfusion phase

Hyperaemic phase
- Autoregulation starts to recover
- Blood-brain permeability is altered, and so there is vasogenic oedema, with leaking of fluid out of the damaged vessels
- Not everyone enters this phase
- A recommended CPP is 50-70 (i.e. you relax your strict targets because the autoregulation is recovering; provided their CT remains unchanged)

Vasospastic phase
- Not everyone ends up in this phase
- Typically you would have had to get a nasty SAH to get into this position
- Produced by arterial vasospasm, hypometabolism, and impaired autoregulation
- Again, the key is maintaining a MAP to maintain a target CPP

Neurogenic Hypertension
- Usually around day 5
- There may also be ECG changes and SVT
- AVOID VASODILATORS! Typically clonidine and beta-blockers are more useful
Interpretation of the ICP waveform

The EVD catheter is zeroed at the level of the external auditory meatus (equivalent to the circle of Willis)
The vascular ICP waveform correlates with the arterial blood pressure waveform
The respiratory ICP waveform correlates with the respiratory cycle

A reasonable maximum ICP to tolerate is 25.

What the hell is causing these waves?
- Experts disagree.
- It is theorized that the P1 and P2 waves are arterial and P3 is venous
- Generally, it is thought that the pulsations of the intracranial vessels are transmitted to the CSF via the choroid plexus, the vessels themselves, and the brain parenchyma
- But, in general, nobody really knows what exactly generates these waveforms

Interpretation of the ICP waveform trends:
These are patterns in intracranial pressure over several minutes

“A” waves, or plateau waves:
- Cerebral perfusion is severely compromised due to increased intracranial pressure
- One ought to get on the phone to the neurosurgeon
Interpretation of ICP waveforms

My ICP trace is flat
- Your EVD is clogged or kinked.
- Your patient has died.

Increased (or decreased) amplitude of all waves (unchanged waveform components)
- Increasing CSF volume (or decreased);
- if you drain off a large volume of CSF, the waveform wont change shape, but it will decrease in amplitude.
- This will also happen in a patient with a missing bone flap

Prominent P1 wave
- The systolic BP is too high
- The ICP trace looks a lot like the art line trace

Diminished P1 wave
- If the systolic BP is too low P1 decreases and eventually disappears, leaving only P2.
- P2 and P3 are not changed by this

Prominent P2 wave
- The mass lesion is increasing in volume
- This trace means the intracranial compliance has decreased; you can also get this with an inspiratory breath hold (as ICP will also rise)

Diminished P2 and P3 waves
- This happens in a hyperventilated patient

Rounded ICP Waveform
- ICP is critically high

When to pull the EVD out? Once there is CT evidence of resolution of cerebral oedema, and provided there is improvement of ICP (i.e. it is consistently under 20-25)
Or… if the EVD is infected.
Maintaining the cerebral perfusion pressure

- Recent changes to guidelines recommend a CPP of ~ 60mm/Hg
- Assuming a normal ICP, that means a MAP over 70

**CPP = MAP minus ICP**

**What to increase the MAP with?**
- Whatever crystalloid you were going to use anyway; normal saline is satisfactory
- What you DON’T want is an increase in free water, so avoid dextrose or 4%-and-5th
  - According to the SAFE investigators, **albumin increases mortality in head injury.**
  - Aim for a Hematocrit of 30
  - Maintain a Hb of 85 to 100
  - Maintain a normal osmolality

**How shall I decrease the ICP?**

**Hyperventilation**
- Reduced CO2 = vasoconstriction and thus decreased cerebral blood flow; thus, ICP will also fall
- But, immediately after a head injury, the cerebral perfusion pressure is already low: hyperventilation will drop it even further.
- This will make the existing hypoxia worse; **THUS:**
  - HYPOVENTILATION IS NOT RECOMMENDED IN THE INITIAL RESUSCITATION OF HEAD INJURY …UNLESS THEY ARE CONING IN FRONT OF YOU

**Mannitol**
- 1g/kg
- ONLY in the normovolemic patient – it will drop the blood pressure
- ONLY useful in an osmolality range of 290-330; thereafter, side-effects outweigh the benefits
- It also enters the brain if the blood brain barrier is damaged, actually worsening the oedema
- Massive hypotension will ensue, as it is a potent diuretic.
  - **THUS:**
    - MANNITOL INFUSION IS NOT RECOMMENDED IN THE INITIAL RESUSCITATION OF HEAD INJURY …UNLESS THEY ARE CONING IN FRONT OF YOU

**Hypertonic Saline may prove to be better (3% saline)** - at least in one study, its superior to mannitol.
- 20% saline, 20 ml, over 20 minutes (“20 of 20 in 20”)

**Keep the PEEP low: 5 - 10**
- Or else you might compromise cerebral venous return

**Maintain normocapnea**
- 35-40 mmHg PCO2

**Head up, 45 degrees**
- To improve cerebral venous return and reduce aspiration risk

**Keep the head straight and try to avoid jugular CVCs**
- Also to improve cerebral venous return

**Keep them sedated (propofol seems to be the poison of choice)**
- To prevent coughing which will also increase the ICP, and to reduce cerebral metabolic demand
- Paralysis for long periods is not indicated- it is associated with a bad outcome
- Barbiturate coma is still practiced, but there isn’t much evidence for it