

# Neurocritical Care

8<sup>th</sup> March 2016 Tuesday

# Management of Severe TBI (from Brain Trauma Foundation guidelines of 2007 and DECRA Trial)

- Primary survey and airway protection
  - ATLS (EMST) Primary Survey: Airway, Breathing, Circulation, Disability and Exposure– the airway to be kept clear and unobstructed
  - RSI and intubation (eg thiopentone, suxamethonium +/- fentanyl). Sedation as appropriate. Neuromuscular blocker until stable and imaging completed
- Monitor ETCO<sub>2</sub>
  - Keep normocarbica PaCO<sub>2</sub> 36-40mmHg
  - All ventilated head injury patients should have continuous capnography
  - Keep well oxygenated with PaO<sub>2</sub> > 90mmHg and SpO<sub>2</sub> >95%
- Early non contrast CT Brain
- Maintain CPP and prevent hypotension
  - All patients should have arterial and CVC (arterial pressure and ICP zero point: at external auditory meatus)
  - First, volume resuscitation to achieve euvolaemia→ then noradrenaline to maintain MAP 80mmHg when ICP is unknown
  - Once ICP monitoring present→ fluid and noradrenaline, to aim CPP 60mmHg
- Monitor ICP
  - Ventriculostomy and EVD preferred to allow for therapeutic CSF drainage

- Provide early nutrition via OGT/NGT
  - Exclude anterior base of skull fracture first before NGT insertion
  - Enteral feeding as soon as any urgent surgery completed
  - Aim to provide full caloric requirements by day 7 post injury
- Maximise venous drainage from head
  - Nurse 30 degrees head up unless haemodynamically unstable, or need large doses of noradrenaline, or prevented by unstable spinal or pelvic injuries
  - If unable to nurse 30 degrees head up, tilt bed feet down, patient supine
  - Secure ETT with brown tape to chin or lips, avoid neck compression
  - Remove cervical collars and use sandbags if necessary
- Clear the spine
  - ALL cervical/thoracic/lumbar spine imaging should be completed within 24h of injury
  - A full CT neck at the same time of first CT brain
  - Hard collars to be removed within 4h and either changed to a Philadelphia collar or preferably sandbags, if spine cannot be cleared.
  - If lateral cervical spine Xray and CT neck are reported normal, then all collars can be removed

- Fluid management
  - Isotonic crystalloid (generally 0.9% saline unless significantly hyperchloraemic, or Plasmalyte) to maintain serum
- Keep normothermic
  - Kept 37°C with forced air warming or cooling, and paracetamol. Intravascular cooling to be considered if other methods are ineffective.
- Prevent stress ulcers
- Assess for Anticonvulsant Requirement
  - Only those who already had seizure should be loaded with phenytoin 15mg/kg IV and prescribed 150mg phenytoin IV BD for 7 days.
- Prevent DVT
  - Sequential calf compressors to be used routinely
  - Add heparin 5000 units BD subcut if no intracerebral bleeding or > 48h after surgical haemostasis
- Control BSL
  - Maintain BSL below 10mmol/l with insulin infusion if necessary (start insulin if BSL >10, then maintain BSL between 8-10mmol/l)

# What if ICP goes up?

- Deepen sedation
  - Recheck basics including ventilation (including CO<sub>2</sub> and oxygenation) and monitoring system (including ICP waveform)
- Neuromuscular blockade
  - Bolus 10mg cisatracurium or equivalent. If ICP responds, then deepen sedation.
- CSF drainage
  - If ICP >20mmHg, drain CSF (drain set 5cm above auditory meatus) for 5minutes then monitor for 10 minutes. Repeat as necessary.
- Consider osmotherapy
  - Only used to buy time
  - Contraindicated if serum osmolality >315mosm/kg. Either 7.5% hypertonic saline (50-100ml boluses) or mannitol 20% (100ml) can be used.
  - Greater diuretic effect with mannitol but more prolonged effect with hypertonic saline
- Manual hyperventilation
  - Only used to buy time in life-threatening emergency, not routinely used for high ICP
- If ICP remains >20mmHg for 15minutes despite the above treatment:
  - Recheck monitoring system. Does the ICP monitor show a normal waveform?
  - Notify neurosurgeon and intensivist

# Second Line Treatment for Persistent Intracranial Hypertension

- Further increase sedation
  - Does a bolus of 250mcg of fentanyl make any difference
- Optimise fluids
  - If fluid balance > 2litres positive, consider frusemide IV. If serum Na < 150mmol/l give 7.5% hypertonic saline bolus 50-100ml
- Open EVD
  - To continuous drainage (still measure ICP regularly as well)
- Mild hypothermia
  - Cool to between 36.5-37, but don't allow temperature below 36.
- Neuromuscular blockade
  - Bolus as above, if required, infuse neat cisatracurium at 0.05-0.15mg/kg/h.
  - Aim train of Four  $\frac{1}{4}$  at the ulnar nerve at the wrist if relaxant infusion used
- Thiopentone 2mg/kg boluses
  - If necessary, move on to infusion 100mg/h and increase to achieve burst suppression on EEG.
  - Bedside EEG monitoring mandatory if thiopentone infusion used.
  - BIS acceptable substitute if we ignore number and look at waveform from the frontal EEG and the suppression ratio
  - Back off to minimum effective dose when burst suppression has been achieved for 20min, cease infusion when ICP controlled for 24h.
- Consider late decompressive craniectomy in selected cases

# Management of aneurysmal SAH

- Guidelines here apply to NON-TRAUMA patients with spontaneous SAH bleed on CT, or with other evidence of SAH (eg xanthochromia on LP)
- Patients with traumatic SAH → Managed according to severe TBI guidelines
- Management different depending on whether aneurysm is secured
  - Risk of catastrophic re-bleed ~8% in the first 48h and then 1% per day thereafter, so usual plan is to secure aneurysm within 28h of presentation.
  - Reasonable to defer surgery in poor grade patients until there is evidence of neurological improvement, and in patients who present late with established vasospasm until the spasm has resolved
- Vasospasm causing delayed cerebral ischaemia and hydrocephalus are major complications after aneurysms secured
  - Risk of vasospasm (low, moderate or high risk) relate to:
    - The presenting grade
    - The overall blood load (Fisher score), and
    - The anatomy of the bleed in patient

# ICU Admission and Discharge Criteria

- Under usual circumstances, all acute SAH patients admitted to ICU
- Occasionally reasonable to admit low risk Grade 1 and Grade 2 SAH patient, and those with an unclear diagnosis, to neurosurgical ward after discussion with consultant neurosurgeon.
- All patients remain in ICU for at least 48h after securing aneurysm
- High risk patients remain in ICU until the peak incidence of vasospasm has passed (7 days) and the patient is stable.
- On admission, clearly document:
  - Grade (Hunt and Hess or WFNS)
  - Fisher score
  - Vasospasm score
- Discharge criteria:
  - At least 48h post securing aneurysm and no further planned procedure
  - No significant neurological changes for at least 24h
  - Past peak incidence of vasospasm in high risk patients
  - Stable off ventilation or catecholamines for at least 24h
  - Not dependent on CNS drainage if a ventriculostomy is present

# Initial Investigation and Imaging

- All non-trauma patients with free subarachnoid blood on initial CT, should have CT angiogram circle of Willis study done at same time
- Most patients will require DSA, generally within 24h of presentation, to guide subsequent therapy, unless CT Angio sufficiently clear for surgical decision-making
- In poor grade patients who present deeply unconscious → reasonable to defer angiogram while awaiting evidence of neurological improvement.
  - Generally need early ventriculostomy to prevent development of obstructive hydrocephalus and brainstem herniation
- All SAH should have daily transcranial dopplers during weekdays
  - Large artery vasospasm often present if mean blood flow velocities are  $> 200\text{cm/s}$  and/or MCA/ICA ratio  $> 6$

# Basic Resus and ICU management of Unconscious SAH patient

- Perform a Primary Survey and Protect airway
- Monitor ETCO<sub>2</sub>
- Obtain brain imaging
- Monitor ICP if necessary
- Maintain euvolaemia and adequate BP
  - Fluid management: isotonic crystalloid (0.9% saline or Plasmalyte) is used to maintain clinical euvolaemia (generally 3L over 24h)
  - Modest BP elevations (MAP <110mmHg and systolic BP <160mmHg) not thought to be associated with re-bleeding and do not need therapy.
  - Hypotension need treatment (pre-morbid BP should be used to determine on target)
  - CPP targets have not been validated after SAH and should not be extrapolated from traumatic brain injury literature.
  - INTERACT-2: target sys BP <140mmHg prior to securing aneurysm
- Early nutrition
- Maximise venous drainage from head
- Keep normothermia
- Prevent bleeding from stress ulcers
- Prevent DVT
- Control BSL
- Maintain Hb above 80-100g/L
  - Threshold for pRBC is 80-100g/L, rather than standard 70g/L based on TRICC trial

# Neurogenic pulmonary oedema

- Any respiratory compromise that accompanies an acute neurologic insult that cannot be explained by co-existing cardiac or pulmonary derangement.
- Up to 23% of SAH patients get some sort of pulmonary oedema with 2-8% attributable to neurogenic pulmonary oedema (mortality rate up to 50%)
- Early onset (minutes to hours) vs delayed onset (by 12-24h post-bleed)
- Pathophysiology: sympathetic surge → pulmonary venoconstriction + rise in cytokine-driven capillary permeability → extravascular lung water and impaired oxygenation
- Treatment:
  - Supportive
  - Reduce ICP
  - Maintain euvolaemia
  - Lung protective measures: Low tidal volume, PEEP, titration of Fio2 to maintain SaO2

# Stress Cardiomyopathy

- ECG changes (ST and T wave changes, QT prolongation and U waves)
- Ventricular and supraventricular arrhythmias
- Troponin elevation
- Myocardial dysfunction in absence of coronary vasospasm
- Incidence of arrhythmia 35% (~5% life threatening)
- Incidence of left ventricular wall motion abnormalities 22% with troponin elevation in 68% of patients post-bleed.
- Tako-tsubo cardiomyopathy (transient wall motion abnormality of LV-- apex of LV balloons outward with relative sparing of basal segments)
- Treatment:
  - Minimise myocardial oxygen demand (treat arrhythmias promptly)
  - Aspirin, nitrates, statins, beta-blocks, calcium channel blockers, ACEI
  - Intra-aortic Balloon pump

# Prophylaxis against Cerebral Vasospasm

- All SAH patients admitted to ICU (pre- or post-clipping) require prophylaxis against vasospasm:
- Maintain Euvolaemia and watch for diuresis
  - Keep well hydrated and euvolaemic. ~3L/day (1.7ml/kg/h) of 0.9% saline or Plasmalyte initially prescribed and adjusted according to clinical response.
  - Osmotic diuresis common after angiography → watch for excessive hypernatremia (sNa above 145mmol/l) and treat with 5% dextrose as required.
- Give nimodipine
  - Given to all aneurysmal SAH patients for a total of 21 days.
  - No evidence that there are any outcome differences with oral/enteral nimodipine compared with IV
- Maintain “high normal” serum magnesium 1-1.2mmol/l
- Maintain “high normal” serum sodium
  - Fluid restriction must not be used to treat hyponatremia even if SIADH thought to be main problem; correct hyponatremia with 0.9% saline or 3% saline after consultation.
- Continue statin therapy
- No prophylactic Hypervolaemia Haemodilution Hypertension therapy

# Treatment of Vasospasm

- Watch for new neurological deficits
- Vasospasm most frequently seen between day 3 and 14 after SAH, and rarely before 3 days or after 18 days.
- Most common presentations:
- Transcranial Doppler can provide early warning

# Early Reperfusion Therapy

- IV thrombolytic agents
- Intra-arterial thrombolysis
- Mechanical thromboembolectomy
- Ultrasound enhanced thrombolysis

## Alternatives:

- Enhanced oxygen delivery
- Haemodilution
- Systemic central haemodynamic augmentation treatment

# IV Thrombolysis

- Clear benefit in survival and in neurological outcomes within 3h of acute ischaemic stroke
- Still beneficial within 4.5h of ischaemic stroke in non-diabetic patients younger than 80 years
- Improved survival and neurological outcomes at 3-6 months when given within 6h of ischaemic stroke in Cochrane review of 27 controlled trials
  - Although increased early death and increased rates of intracerebral haemorrhage

# Risks

- Failure to reperfuse
- Intracerebral haemorrhage
- Ischaemic reperfusion injury
- Catheterisation complications

# Intra-arterial Strategies

- Best to pick patients who demonstrate mismatch between hypoperfused but salvageable brain tissue (on brain imaging) and tissue that has or is predicted to infarct
- After 4.5h of acute ischaemic stroke, or if IV thrombolysis contraindicated
- In TPA-eligible patients, results are mixed when intra-arterial strategies are used either as an alternative or as a supplement to IV thrombolysis

# Prevention of Secondary Insults

- Early removal of large intracerebral haematomas
  - Evidence of improved outcomes lacking, except in epidural haematoma management as shown in a prospective cohort study
- What about decompressive craniectomy in patients who still have raised ICP despite first-tier ICU and neurosurgical management?
  - Poor outcomes at 6 months of injury by Extended Glasgow Outcome Scale
- Early detection and management of:
  - Hypoxia
  - Hypotension → Hypertonic saline not better
  - Raised ICP
  - Reduced cerebral perfusion
  - Seizures
- Pre-hospital intubation is good (as evaluated by Extended Glasgow Outcome Scale)

# Autoregulation and Neuroprotection

- Cerebrovascular autoregulation= ability to maintain constant cerebral blood flow through a range of cerebral perfusion pressure
- Several dynamic pressure reactivity indices can be used (by calculating correlation between arterial blood pressure and continuous cerebral blood flow/ cerebral blood volume)
  - ICP
  - Transcranial dopplers
  - Brain tissue oxygenation
  - Spectroscopy

# Transfusion threshold?

- For closed head injury (Transfusion threshold 70 vs 100) → No neuro outcome differences at 6 months, but higher adverse events with higher threshold
- For SAH with high risk for vasospasm (Liberal transfusion strategy aim Hb >115 vs conservative >100): no difference in safety endpoints; more cortical infarcts in conservative group.
- In SAH patients who were transfused → higher risk of PE, thrombosis and poor outcome
- Transfuse only if anaemic AND brain tissue oxygen tension (PbtO<sub>2</sub>), and not anaemia alone in TBI patients (due to poor 1 month outcomes)

# Hepatic Encephalopathy

- Increases in cellular glutamine/glutamate levels → increased circulating ammonia → altered neurotransmitter signalling → astrocyte swelling + microglial and mitochondrial dysfunction
- For acute liver failure:
  - Renal replacement for ammonia
  - Temperature control
  - Control inflammatory phenotype
  - Hyponatremia
  - Liver transplant
- For chronic cirrhosis:
  - Agents to drop arterial ammonia
  - Liver transplant

# Temperature

- Brain temp > core temperature by 1-2 degree Celsius
- Aim temp not more than 37.5
- For Ischaemia-reperfusion injury:
  - begins within minutes to hours of injury
  - Effects last up to 72h after initial ischaemic insult
- For brain oedema: Treat for as long as it persists
- For TBI, acute ischaemic CVA with brain oedema, and acute hepatic encephalopathy:
  - Therapeutic hypothermia lowers ICP, BUT this does not lead to better outcomes
- Prolonged (4-5 days) therapeutic hypothermia has better results than TH for shorter periods associated with rebound ICP when rewarmed
- For out-of-hospital cardiac arrest: very mild hypothermia (36°C) as protective as moderate hypothermia (33°C)