Immediate Care of Traumatic Brain Injury & Subarachnoid Haemorrhage

Paul Harrison
Consultant Anaesthetist

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• Background information about TBI / SAH
• Basic principles of immediate management
• Neurophysiology
• Practical aspects of immediate management
• Further management of TBI / SAH
Traumatic Brain Injury

- Major cause of
  - Disability
  - Death
  - Economic cost
- Recent study in Scotland estimated an annual incidence of between 100 and 150 per 100,000 adults disabled a year after a head injury

- 47% of patients followed up for one year after discharge had survived with some form of restriction to lifestyle

- Proportion of patients experiencing the most serious sequelae did not vary according to the severity of the initial injury
• About 80% male

• About 15% elderly (≥ 65 years)

• About 40 – 50% children

• Alcohol may be involved in up to 65% of adult head injuries
• Common causes of minor head injury
  – Falls (22-43%)
  – Assaults (30-50%)
  – Road traffic accidents (~25%)

• RTA’s account for a far greater proportion of moderate to severe head injuries
Who should have a CT scan

- The patient is eye opening only to pain or does not converse (CGS 12/15 or less)
- A deteriorating level of consciousness or progressive focal neurological signs
- Confusion or drowsiness (CGS 13 or 14/15) followed by failure to improve within at most four hours of clinical observation
- Radiological/clinical evidence of a fracture, whatever the level of consciousness
- New focal neurological signs which are not getting worse
- Full consciousness (GCS 15/15) with no fracture but other features, e.g.:
  - Severe and persistent headache
  - Nausea and vomiting
  - Irritability or altered behaviour
  - A seizure.
• Types of injury
  – Impact injury
    • Contusions/lacerations
    • Diffuse axonal injury
  – Bleeding
    • Extradural
    • Subdural
    • Intracerebral (usually into contusions)
Once injury has occurred, damage cannot be altered

However, secondary brain damage starts to occur
  – Swelling
  – Ischaemia
  – (Infection)
• Once head injury has occurred, management is aimed at avoiding secondary injury

• GOOD SPECIFIC CARE IS ESSENTIAL.
• Outcome determined by
  – Severity of initial injury
  – Prevention / management of subsequent complications

• Outcome poorer with increasing age

• Young children have greatest capacity for recovery
Subarachnoid Haemorrhage

- Traumatic

- Spontaneous
  - Rupture of intracranial aneurysm (70 – 75%)
  - Bleeding from arterio-venous malformation (5%)
  - Others/undefined (20%)
Aneurysmal SAH

- Incidence 5 – 30 per 100,000 population per year
- Peak age 40 – 60 years (rare in childhood)
- Female:Male ratio 3:2
Aneurysmal SAH

• Predisposing factors
  – Hypertension
  – Smoking
  – Cocaine abuse
  – Alcohol
    • Especially binge drinking
  – Race
    • Black > white
    • High incidence in Finland & Japan, low in South/Central America
  – Other conditions
    • Autosomal dominant polycystic kidney disease
    • Sickle cell disease
    • Connective tissue disorders
• Blood supply is from
  – Internal carotid arteries
  – Basilar artery
• Anastomose to form Circle of Willis
• Divides to give anterior, middle and posterior circulations
Aneurysms are usually saccular and occur at the bifurcation of vessels.

- Site of aneurysm
  - Anterior cerebral artery 35 – 40%
  - Middle cerebral artery 20 – 25%
  - Posterior circulation 10%
  - Internal carotid 30%

- Approximately 30% have > 1 aneurysm

- At post-mortem 1% of population have aneurysm
Aneurysmal SAH

• Presentation

• Classic
  – Instantaneous severe headache
  – “Thunderclap headache” or “Blow to the head”
  – Nausea/vomiting
  – Neck stiffness/photophobia
  – Seizure
  – Reduced conscious level
  – Focal signs e.g. IIIrd nerve palsy

• Presentation may not fit classical picture
Aneurysmal SAH

• 1/3 patients will die before reaching hospital

• Remaining patients have 30 – 50% mortality

• 1/3 of survivors remain dependent
Aneurysmal SAH

- Prognosis depends on
  - Severity of initial bleed
  - Prevention of rebleeding
    - Success of procedure to secure aneurysm
  - Management/prevention of complications
    - Secondary injury
    - Hydrocephalus
    - Vasospasm
Aneurysmal SAH

- Prone to cardiac problems
- SAH associated with release of large amounts of catecholamines
- Can lead to
  - Cardiac arrhythmias / ECG changes
  - Myocardial infarction
  - Neurogenic pulmonary oedema
  - Cardiac arrest
• Cannot alter primary event

• Good medical care essential to prevent secondary damage
• Initial management should be based on ATLS/ALS
  – Airway
  – Breathing
  – Circulation

• Not specific enough for TBI/SAH
• Some animal evidence of efficacy

• No clinical evidence in humans
  – Large randomised control trial (CRASH trial) suggested corticosteroids worsen outcome

• No agents currently in use as neuroprotective agents
• Physiological neuroprotection
  – O\textsubscript{2}
  – CO\textsubscript{2}
  – Blood pressure
  – Intracranial pressure
  – Temperature
  – Blood glucose
• Need to know basic knowledge of neurophysiology!
• Brain weighs ~ 2% of body weight

• Receives ~ 15% of cardiac output

• 20% of basal $O_2$ consumption

• CBF=50ml/100g/min
• Blood flow affected by several factors
• Local metabolites
  – H⁺, K⁺, adenosine, NO
• Other factors
  – CO₂ & O₂
  – Cerebral perfusion pressure
  – Drugs
  – Temperature
  – Haematocrit
• Why does a small amount of swelling / bleeding in the brain cause such problems?
• Monro-Kellie Doctrine

  – The skull is a rigid closed container

  – Contents are incompressible (brain, blood, CSF)

  – Any increase in the volume of one constituent results in a corresponding increase in ICP
Theoretical intracranial pressure-volume relationship
Actual intracranial pressure-volume relationship
• Some compensation occurs
• Veins, especially dural sinuses act as capacitance vessels
• Blood shunted to central circulation
• Also some movement of CSF
Rising ICP

- If cerebral blood flow \(\uparrow\), cerebral blood volume \(\uparrow\)

- If no compensation can occur this results in \(\uparrow\) ICP
- Brain ‘divided’ into two by a fold of dura, the tentorium cerebelli
- Separates occipital lobe from cerebellum
- As pressure builds up, brain herniates through tentorium, compressing the brainstem
- If unrelieved, will eventually result in brain stem death
• As pressure builds up, the oculomotor nerve is compressed, resulting in a fixed dilated pupil
Autoregulation

- Cerebral Blood Flow (CBF) normally constant despite changes in MAP
Autoregulation

![Graph showing CBF vs MAP (mmHg)]
• Autoregulation maintains CBF constant despite changes in cerebral perfusion pressure

$$\text{CPP} = \text{MAP} - \text{ICP}$$
Autoregulation

- Occurs due to changes in cerebrovascular resistance
- Curve can be shifted to left or right e.g. hypertension, chronically raised ICP
- AUTOREGULATION IS IMPAIRED BY CNS PATHOLOGY e.g. Traumatic Brain Injury
Effect of $O_2$
Effect of O$_2$

- Little effect on CBF until pO$_2$ falls to about 7 – 8 kPa
- Hyperoxia (>60 kPa) causes mild vasoconstriction
Effect of CO$_2$
Effect of CO$_2$

- Four fold increase in CBF between 2.7 kPa and 10.7 kPa
- Almost linear relationship in clinical range
Effect of CO₂

- **Moderate** hyperventilation may be useful to reduce ICP in the *short term*

- After about 24 – 48 hours cerebral blood flow returns to normal

- Return to normocapnia after hyperventilation results in overshoot in CBF
• $O_2$ delivery is a compromise between
  – oxygen carrying capacity
  – flow characteristics of blood

• Haemodilution *may* improve cerebral blood flow in vasospasm associated with SAH
Temperature

- Animal evidence that reducing body temperature after brain injury may improve outcome
- Evidence in humans that patients with normothermia after TBI may have a better outcome than those with hyperthermia
- No evidence that ‘therapeutic’ hypothermia improves outcome
Temperature

- Need to treat hyperthermia aggressively

- Hypothermia associated with complications
  - \( \uparrow \) Systemic vascular resistance / myocardial work
  - Coagulopathy

- Aim for normal body temperature (36 – 37°C)
• Brain is almost totally dependent on exogenous glucose for its cellular energy requirements

• Prolonged hypoglycaemia causes neuronal damage

• Hyperglycaemia is associated with poorer outcome in TBI & SAH
Glucose

- Mechanism unclear
  - In glycolysis, Pyruvate $\rightarrow$ Lactate
  - Lactate is neurotoxic

- In areas where partial ischaemia exists, high glucose levels may allow lactate to build up to very high levels

- No evidence that treating hyperglycaemia improves outcome, but seems sensible
• Maintain normality!
• Ensure optimal gas exchange
  – May require intubation / ventilation
  – Avoid hypoxia, \((PaO_2 > 13kPa)\)
  – Keep CO\(_2\) normal \((PaCO_2 4.5 – 5.0kPa)\)
• Avoid hypotension
  – Treat hypotension promptly
    • Fluid resuscitation
    • Vasopressors/inotropes
    • Treat other sources of bleeding promptly e.g. ruptured spleen
    • Aim MAP > 80mmHg
Management

- Reduce ICP
  - Head up tilt
  - Avoid compression of neck veins
  - If intubated, keep sedated & paralysed
  - Consider mannitol / hypertonic saline

- Avoid hyperthermia

- Avoid hyperglycaemia
Management

- Mannitol
  - ↓Viscosity causes ↑flow resulting in ↓cerebral blood volume causing ↓ICP
  - Osmotic effect – decreased brain water

- Useful to ‘buy time’ until definitive treatment
Further Management

• Traumatic Brain Injury
  – Evacuate haematomas causing increased ICP
  – Consider evacuation of contusions
  – If swelling likely, consider measuring ICP

• If ICP high with no surgical lesions
  – Medical management
    • Sedation (↓CMRO2, ↓CBF, ↓ICP), muscle relaxation, ventilation
  – Decompressive craniotomy
Further Management

- Subarachnoid haemorrhage
  - Prevent rebleeding
    - Endovascular coiling
    - Craniotomy & clipping
  - Prevent/treat vasospasm
    - Nimodipine 4 hourly for 21 days
    - Hypertension, hypervolaemia & haemodilution
  - Treat hydrocephalus
    - External ventricular drain
    - Ventriculo-peritoneal shunt
    - Endoscopic IIIrd ventriculostomy
References / Further Reading