Management of Traumatic Brain Injury

What happens, why & what can we do...
Levels of Injury

MILD

MODERATE

SEVERE
The Extent of the Problem

1,000,000
150,000
12,000
17,000
5,000
120,000
Who is Most at Risk?

- The risk of TBI in men is twice that of women.
- The risk is higher in adolescents, young adults and people over 75 years.
- More than 50% of persons with brain injury were intoxicated at the time of injury.
- Most TBI’s occur mid afternoon to early evening
Phases of Injury

- **First/Primary**
  - at time of incident
  - closed, open or crushing
  - not treatable

- **Second**
  - events or injuries worsen original injury

- **Third**
  - occurs days to weeks following first injury
  - causes extreme pressure and can be fatal
What causes brain damage?

Region at risk

Blood flow reduced by brain swelling

Injured Brain

Lesion core

“Primary Injury”
Effects of Injury

- Impaired airway reflexes.
- Abnormal respiratory pattern.
- Neurogenic pulmonary oedema.
- Hypertension.
- Cardiac dysrhythmias & ECG abnormalities.
- Coagulation disturbances.
- Endocrine & electrolyte disturbances
- Stress response
Key Concepts

- Monro-Kellie Hypothesis
- Brain compliance
- Managing intracranial pressure
- Cerebral Blood Flow & Autoregulation
- Cerebral Metabolism
- Prevention of secondary brain injury
Cranial compartments

- Dural folds divide up the cranium into compartments:
  - Falx cerebri
  - Tentorium cerebelli
  - Falx cerebelli

- Ventricular system distributes pressure evenly
Monro-Kellie Doctrine

“The skull is a rigid compartment, filled to capacity with non-compressible material. Any increase in the volume of one component must be matched by a corresponding decrease in one or both of the others, or intracranial pressure will rise”
Monro-Kellie Doctrine

- 3 components in a rigid compartment.
- Need to measure pressure effects of expanding intracranial mass seen after TBI.
- ICP monitoring first described in 1951 by Guillaume & Janny.
Pressure & Volume

Compensation mechanisms protect the brain from increases in ICP.

Decompensation – Small increases in volume result in progressively larger increases in ICP.
Compensation

- Decrease in CSF volume:
  - Displacement to lumbar theca

- Decrease in cerebral blood volume:
  - Compression of the venous sinuses

- Decrease in CSF production

- Decrease in arterial blood supply
Compliance

- Change in pressure resulting from a change in volume

\[
\text{Compliance} = \frac{\text{change in volume}}{\text{change in pressure}}
\]

Low compliance – (stiff brain)

High compliance – (slack brain)
Factors Increasing ICP

Hypercapnoea

Decreased Venous Drainage

Hypo-osmolar States

Hypertension
Traditional Therapies

Hyperventilation

Head Elevation

Dehydration

Barbiturates
Physiology of Cerebral Blood Flow

- Cerebral blood flow (CBF) main determinant of oxygen and energy supply.
- Normal CBF = 50ml/100g/min.
- CBF <30ml/100g/min leads to symptoms of failing oxygen supply.
- Ischaemic threshold <20ml/100g/min.
- Effects time dependent and may be reversible.
The Ischaemic Penumbra

Diagram showing the Ischemic Penumbra with layers of metabolic failure, electrophysiological failure, and normal tissue.
CBF Following Head Injury
Manipulating CBF

**Response to Oxygen**

- Cerebral Blood Flow (ml/100g/min)
- Arterial PO2 (kpa)

**Response to CO2**

- Cerebral Blood Flow (ml/100g/min)
- Arterial PCO2 (kpa)
Autoregulation

- Cerebral blood flow: 50ml/100g/min
  - Grey matter: 100ml/100g/min
  - White matter: 20ml/100g/min
Autoregulation

![Graph showing autoregulation of CBF (Cerebral Blood Flow) with C.P.P (Cerebral Perfusion Pressure) ranging from 50mmHg to 150mmHg.](image)
Blood Pressure Autoregulation

- CBF maintained constant when MABP remains within range.
- CBF heterogenic.
- Autoregulation due to the myogenic response of smooth muscle.
- With a drop in MABP, CBF decreases and oxygen extraction increases.
- Impaired autoregulation can increase regional CBF leading to BBB damage and cerebral oedema.

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**Autoregulation**

![Autoregulation Diagram](image)

- Mean arterial pressure (mmHg)
Cerebral Metabolism

- Need to control metabolism
  - high dose barbiturates
  - neuromuscular blockade
  - Hypothermia

- Need to understand relationship between metabolism and cerebral blood flow
Therapeutic Hypothermia

- Slows certain secondary processes, e.g. ischaemic rises & brain acidosis
- No definitive level of hypothermia determined
- Mild to moderate reduces release of excitatory amino acids
- Therapy has major implications for nursing care
Changes in Cerebral Blood Flow

Fig 1. Relationship of cerebral blood flow and metabolism in the presence of ischaemia and hyperaemia. Modified from Menon 2000.
Primary Injury

Inflammatory Response

Increased vascular permeability & vasodilatation

Vasogenic Oedema

Cerebral Ischaemia & Impaired Autoregulation

Cytotoxic Oedema

Decreased ATP Production

Increased Lactic Acidosis

Increased Intracellular Influx of Sodium, Chloride, Calcium & Water
Physiological Insults Following Head Injury-Relation to Outcome

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mortality</th>
<th>GOS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of hypotension (SBP ≤ 90 mmHg)</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Duration of hypoxia (SpO₂ ≤ 90%)</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>Duration of pyrexia (T_{core} ≥ 38°C)</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>Intracranial hypertension (ICP &gt; 30 mmHg)</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>Cerebral perfusion pressure (CPP &lt; 50 mmHg)</td>
<td>yes</td>
<td>no</td>
</tr>
</tbody>
</table>
Initial Management

- Be aware of the effects of a severe brain injury-
  - GCS <8 will need airway protection for transfer

- Maintain effective ventilation-
  - Hyperoxygenate
  - Mild hyperventilation only for first 24 hours

- Ensure adequate resuscitation-
  - Assume ICP of around 30mmHg.
Cerebral Oedema

Vasogenic - Protein rich fluid passes through damaged blood vessel walls.

Cytotoxic – Fluid accumulates within the cells.

Interstitial – CSF forced under pressure into the extracellular space.
Jugular Bulb Monitoring

- Jugular Bulb
- Common Facial Vein
- Jugular venous cannula
- Internal Jugular Vein
Tissue Oxygen Measurement

Figure 5.5.1 Diagrammatic representation of a cross section of the Neurotrend™ sensor. (Reproduced with permission from Codman UK.)
Principles of Microdialysis

- Microdialysate collected via outlet tube
- Isotonic fluid perfused via inlet tube
- Molecules in ECF equilibrate across MD membrane
- Microdialysis catheter
- Semi-permeable membrane
- Brain tissue interstitium
Clinical Application

Markers of Ischaemia
- Fall in brain glucose
- Increase in lactate
- Fall in pyruvate
- Increase in LP ratio
- Increase in glutamate
- Increase in glycerol