Traumatic Brain Injury

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Contents

Components of the Brain.....3

Monro-Kellie Hypothesis.....4

Intracranial Pressure.....5

Definition of Traumatic Brain Injury.....6

Types of Traumatic Brain Injury.....8

Nursing management of the patient with traumatic brain injury.....10

Pharmacological agents used to treat intracranial hypertension.....16

Surgical treatments of increased intracranial pressure.....19

General nursing considerations.....20

References.....22

COMPONENTS OF THE BRAIN

The skull is a rigid structure with a fixed internal, non-compressible volume of 1400-1700mls, filled with 3 components:

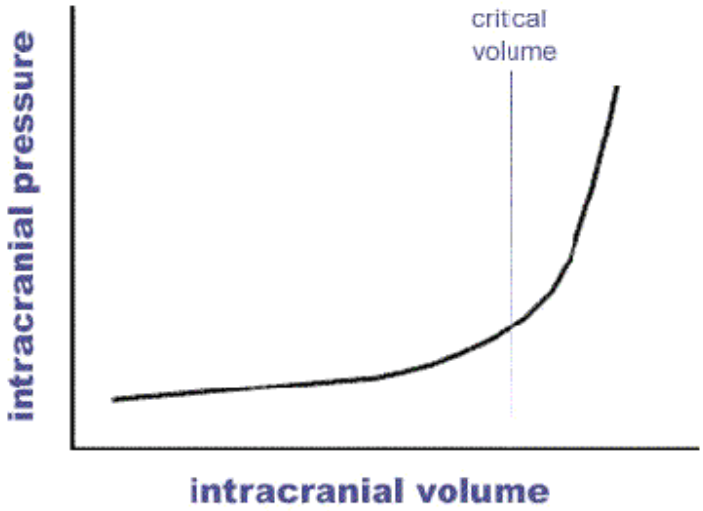
Brain: approximately 80% of intracranial volume, including brain tissue, interstitial and intracellular water

Intracranial blood volume: approximately 10% of intracranial volume

Cerebrospinal fluid (CSF): approximately 10% of intracranial volume

MONRO-KELLIE HYPOTHESIS

As the skulls contents are incompressible, if one of the three components increases in volume, it must be compensated for by a decrease in the volume of one or more of the remaining components; otherwise intracranial pressure will increase (the Monro-Kellie Doctrine) (Kellie, 1823; Monro, 1823; cited in May, 2009: 911). The relationship between volume and pressure is best illustrated through the volume pressure curve.



INTRACRANIAL PRESSURE

Intracranial pressure (ICP) is the pressure exerted by the brain tissue, blood volume and CSF.

Normal values: Adults and older children – 0-15mmHg
Young children – 3-7mmHg
Term infants – 1.5-6mmHg

20-30mmHg indicates **mild** intracranial hypertension and requires treatment

>40mmHg indicates **severe**, life threatening intracranial hypertension.

Increased ICP occurs when the volume of any of the three components in the cranial vault exceeds the brain's ability to accommodate. This may occur in patients with acute neurologic conditions such as tumour, haemorrhage, anoxic brain injury and in particular, traumatic brain injury (TBI).

These conditions alter the relationship between intracranial volume and pressure.

Current guidelines recommend maintaining ICP lass than 20mmHg in the traumatic brain injured patient (Ling & Marshall, 2008: 419).

DEFINITION OF TRAUMATIC BRAIN INJURY

Hickey (2003) defines TBI as:

"any injury to the scalp, skull or brain that is of significant magnitude to interfere with normal function and to require treatment".

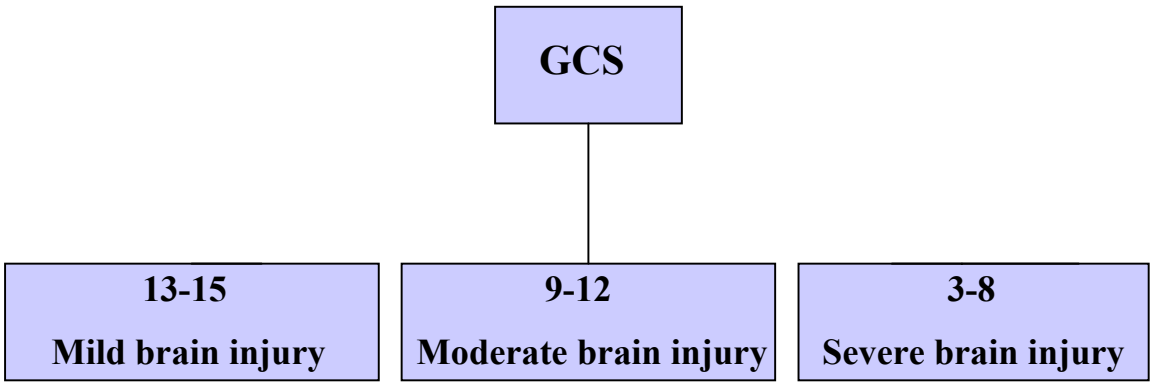
TBI can be further classified as primary or secondary injury.

Primary injury comes through direct contact to the head and brain as an immediate result of the initial insult at the moment of injury. Typically, the damage that occurs from primary injury is often complete by the time medical care can be instituted (Hickey, 2003).

Secondary injury occurs in the aftermath of trauma and is caused by a blood flow – metabolism mismatch and results in ischaemia that can lead to infarction. Secondary injury influences the extent of brain damage and patient outcome significantly by compromising the oxygen and nutrient supply to the brain (Saiki, 2009: 550).

Hypoxia and hypoperfusion are recognised as leading contributing factors to secondary brain injury.

TBI severity is classified as mild, moderate or severe depending on the patient's GCS on admission.

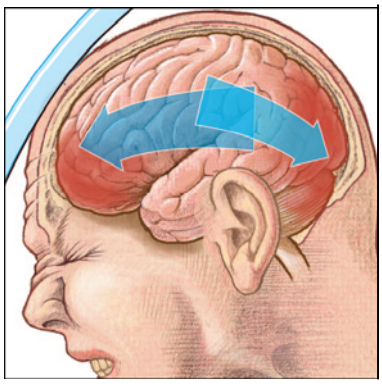


Mild brain injury is frequently referred to as 'concussion'. These patients have experienced a brief loss of consciousness, and present may with headache, confusion and amnesia. Following the acute period, postconcussion syndrome (headache, dizziness, anxiety, difficulty concentrating) may develop and can last up to one year (Ling & Marshall, 2008: 414)

Moderate brain injury is often associated with prolonged loss of consciousness or neurological deficit. These patients require hospitalisation, have a greater likelihood of abnormal findings on craniocerebral imaging, and may need neurosurgery (Ling & Marshall, 2008: 414).

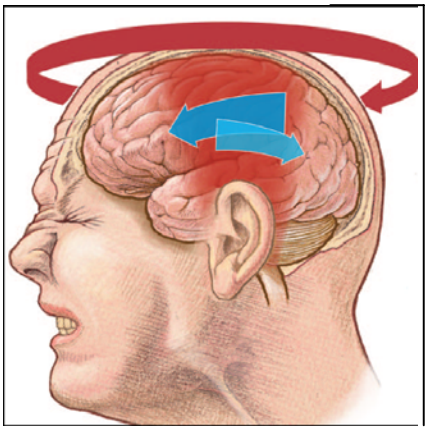
Severe brain injury is significant neurological injury. Typically, these patients have abnormal neuroimaging and require ICU admission and neurosurgical intervention. Recovery is prolonged and usually incomplete, with a significant percentage of severe TBI patients not surviving to 1 year (Ling & Marshall, 2008: 414).

TYPES OF TRAUMATIC BRAIN INJURY

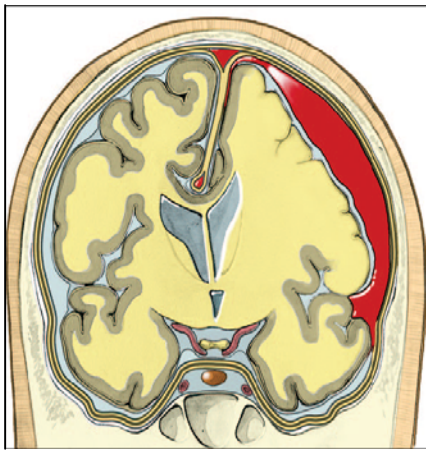


Contusions are bruises along the surface of the brain as the result of the brain moving within the skull, causing disruption of the tissue and vascular components of the brain.

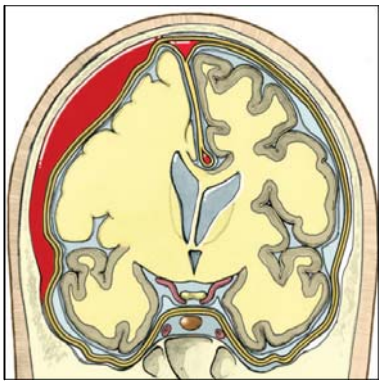
Cerebral contusions may be at the site of impact **(coup)** and/or directly opposite point of impact (acceleration-deceleration) **(contrecoup).** These types of injuries are commonly seen child abuse involving shaking of the victim.



Diffuse Axonal Injury (DAI) commonly occurs as a result of rotational acceleration-deceleration injury. In DAI, axons and small vessels become torn as a result of shearing force, which results in widespread damage to neurons and often small contusions throughout the parenchyma within the deep white matter and brain stem.

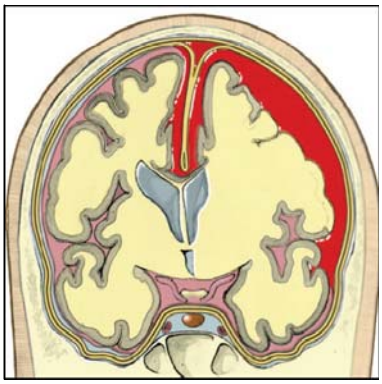


Subdural Haematoma (SDH) is a collection of blood between the dura mater and the arachnoid meningeal layer, usually caused by damage to bridging veins that course through the subdural space. As venous blood accumulates more slowly than arterial blood, signs & symptoms of ↑ ICP may develop less rapidly.



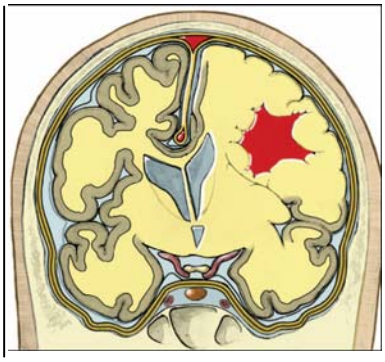
Extradural Haematoma (EDH) is a collection of blood between the first protective covering of the brain (dura mater) & the inner table of the skull (inner periosteum). Most EDH's are arterial origin, occurring from a tear of the middle meningeal artery, therefore are mainly found in the temporal- parietal region.

In most cases EDH is a neurosurgical emergency, requiring evacuation of the clot to relieve brain compression and prevent herniation (Zink & McQuillan, 2005: 37).



Traumatic Subarachnoid Haemorrhage (SAH) is a collection of blood that spreads diffusely throughout the subarachnoid space. A degree of SAH is invariably present with serious brain injury caused by surface contusions.

Intracerebral Haemorrhage (ICH) is bleeding within the brain tissue caused by a deep contusion or tear in the blood vessels. ICH's result from significant blows to the head and may be associated with other brain injuries, particularly contusions or depressive skull fractures.



(Zink & McQuillan, 2005: 37-40)

NURSING MANAGEMENT OF THE PATIENT WITH TBI

Oxygenation

Hypoxia and hypercapnia can ↑ ICP dramatically; therefore optimal respiratory management is crucial in TBI patients.

Approx 36% of TBI patients present with hypoxia & respiratory dysfunction requiring mechanical ventilation on admission (Rangel-Castillo, Gopinath & Robertson, 2008: 529). Zink & McQuillan (2005: 37) recommend that any head-injured patient with a GCS of 9 or less (severe TBI) should be intubated and mechanically ventilated, as altered mental status, impaired airway patency and inadequate ventilation can cause hypoxia and hypercapnia.

Current recommendations define the threshold for hypoxia as PaO^2 <60mmHg (Hickey, 2003: 302). Oxygenation should therefore be monitored & hypoxia avoided. NICE guidelines (2007) (cited in Christie & McLernon, 2008: 555) state the need to **aim for a PaO^2 >98mmHg during the acute phase of TBI**. Continual pulse oximetry (SaO^2) and regular arterial blood gases should therefore be attended, with FiO^2 titrated accordingly.

Mechanical ventilation can however, have adverse effects on ICP. PEEP can increase ICP by impeding venous return & increasing cerebral venous pressure. Acute lung injury and ventilator associated pneumonia are also potential complications of mechanical ventilation.

CO^2 Control

The control of $PaCO^2$ is fundamental in the management of TBI patients.

Hypercapnia ($PaCO^2$ >45mmHg) is a potent vasodilator & so will induce hyperaemia, whereby cerebral blood volume (CBV) increases, requiring a reciprocal decrease in either brain tissue of CSF. In severe TBI, the limitations of the compensatory mechanism are often exhausted, therefore leading to ↑ICP.

Hypocapnia ($PaCO^2$ <35mmHg) is a potent vasoconstrictor, reducing cerebral blood flow (CBF) & CBV, therefore inducing a sharp decrease in perfusion of blood to the brain. Failure to maintain adequate CBF increases the risk of secondary injury; therefore the aim is to maintain normocapnia in TBI patients.

Controlled ventilation can maintain a normal PaO^2 & $PaCO^2$, therefore eliminating this as a cause of ↑ICP.

Cerebral Perfusion Pressure

CPP represents the blood pressure gradient across the brain, and in its simplified form can be calculated as:

$$CPP = MAP - ICP$$

CPP is used as an indicator of CBF and therefore, oxygen delivery to the brain. In severe TBI, cerebral autoregulation is lost & CPP becomes dependent on systemic BP.

Maintaining a CPP of 60-70mmHg is most effective in adequately perfusing traumatised cerebral tissue without oversupplying CBF & creating an increase in ICP (Ladanyi & Elliot, 2008: 144).

If CPP cannot be maintained with intravenous fluids alone, inotropic support, such as noradrenaline, may be required.

A CPP persistently less than 60mmHg can result in cerebral ischaemia and infarction (Ladanyi & Elliot, 2008: 144)**.**

Blood Pressure Management

Hypotension is the predominant contributing factor in secondary brain injury & has the highest correlation with morbidity & mortality (Christie & McLernon, 2008: 556).

The Brain Trauma Foundation (BTF) defines hypotension as systolic blood pressure (SBP) <90mmHg. However in the acute phase of TBI, it is recommended that the MAP be supported in order to maintain a CPP 60-70mmHg (Ladanyi & Elliot, 2008: 144). Fluid resuscitation and/or inotropes should be administered to the patient to ensure these guidelines are met.

Continuous arterial BP monitoring is required in the TBI patient to enable estimation of cerebral perfusion pressure.

Temperature Control

Loss of thermal autoregulation as a result of TBI can present additional challenges in management. In response to a temperature increase of 1°, CBF & metabolic rate ↑ 5-7% with a 10% rise in oxygen consumption (Ladanyi & Elliot, 2008: 147).

Nursing care focuses on maintaining **normothermia**. External cooling strategies including cooling blankets or ice packs should be employed. If a patient shivers as a result of active cooling, oxygen consumption and ICP will increase as a result of an elevated cerebral metabolic rate. Care should therefore be taken not to cool a patient too quickly.

While fever is often due to the head injury itself, effort should be made to identify and eradicate sources of infection.

Fluid management

Dehydration and hypotension are dangerous in the TBI patient and can result in ↓CPP and ischaemia, therefore the goal is euvolaemia.

Hypotonic IV fluids such as **5% dextrose** and **0.45% saline** should be **avoided** in the patient with ↑ICP (Ling & Marshall, 2008: 420). Excess free water from these solutions lowers plasma osmolality and drives water across the blood-brain barrier, therefore increasing cerebral water content and ICP. Normal saline and Hartmann's solutions are the recommended IV fluids for TBI fluids.

Hourly measurement and recording of fluid balance, and regular assessment of central venous pressure (CVP) should be attended to give indication of volume status.

Position

The patient's head of the bed should be **elevated to 30°** and maintained in a **neutral position**. This helps to improve ICP by improving cerebral venous outflow & movement of CSF to the spinal canal and without impairing cerebral perfusion or oxygen delivery (Luks, 2009: 46)

The following positions should be avoided:

- Lateral flexion of the neck
- Prone position
- Extreme hip flexion

Seizure Management

Seizures increase cerebral metabolic rate & ICP, which may lead to secondary injury, therefore, should be treated aggressively.

Current literature demonstrates that prophylactic antiepileptic drugs, eg Phenytoin, are effective in preventing early post-traumatic seizures, however much less evidence exists in favour of prophylactic treatment of patients for more than 1 week after TBI.

PHARMACOLOGICAL AGENTS USED TO TREAT INTRACRANIAL HYPERTENSION

Sedation & Analgesia

As agitation raises ICP, care should be taken to ensure adequate sedation and analgesia is administered to the patient. With severely elevated ICP's sedation is necessary to decrease cerebral metabolic activity and oxygen demand.

Benzodiazepines are the most commonly used sedatives and do not affect the cerebral metabolic rate of oxygen consumption ($CMRO^2$), CBF or ICP (Hickey, 2003). **Lorazepam** & **Midazolam** are commonly used.

Propofol is an ultra-short acting **sedative-hypnotic** that decreases $CMRO^2$ (Saiki, 2009: 555). Hypotension, leading to decreased CPP, and 'propofol infusion syndrome' are common complications of Propofol.

It is unwise to assume that while a patient is sedated and/or unresponsive that pain will have no physiological effect. Therefore appropriate **analgesia** should be administered in conjunction with sedation, for example **Morphine** or **Fentanyl**.

Hyperosmolar therapy

Mannitol and hypertonic saline are the most frequent hyperosmolar therapies used in the treatment of ↑ICP associated with TBI.

Hypertonic saline (HTS), given in concentrations ranging from 3% - 23.4%, pulls water from the oedematous brain into the intravascular compartment, therefore reducing intracranial volume and ICP (Rangel-Castillo, Gopinath & Robertson, 2008: 533). Serum sodium and osmolarity must be closely monitored.

Side effects of HTS include haematologic and electrolyte abnormalities, hypernatraemia and hypokalaemia.

Mannitol exerts rheological effects that lead to expansion of plasma with resulting reduction in haematocrit and blood viscosity which inturn ↑CBF and oxygen delivery (Rangel-Castillo, Gopinath & Robertson, 2008: 533). Mannitol also produces an osmotic effect which draws fluid out of the brain into the intravascular space, therefore reducing cerebral oedema.

Side effects of mannitol include hypotension & hypovolaemia (2° to its diuretic effects), hyperosmolarity & renal failure.

Paralysis

Neuromuscular blocking agents work to ↓ICP by depressing the systemic response to internal or external noxious stimuli by decreasing muscle activity. The cessation of movement decreases systemic metabolism by reducing oxygen consumption and carbon dioxide production.

The most commonly used neuromuscular blocking agent is **Vecuronium**.

Barbiturates

Barbiturates should only be used as a last resort to treat intracranial hypertension resistant to other standard therapies. The underlying therapeutic of the drug is a vasoconstriction effect and a rapid decrease in cerebral metabolism & CBF, which leads to a decrease in cerebral blood volume & ICP (Hickey, 2003: 306). **Thiopentone** and **Pentobarbital** are the two commonly used barbiturates in ICP management.

The most common complication of barbiturates is systemic hypotension. In addition, barbiturates completely obliterate neurologic examination except for pupil examination; therefore the clinician relies solely on ICP monitoring (Saiki, 2009: 555). Other complications include hypokalaemia, hepatic and renal failure.

SURGICAL TREATMENTS OF INCREASED INTRACRANIAL PRESSURE

CSF drainage

Some patients with severe TBI may require an **external ventricular drain (EVD)** to be surgically inserted for periodic drainage of CSF in an effort to decrease the total volume of the intracranial compartment & restore equilibrium (Monro-Kellie hypothesis).

The EVD is positioned to regulate drainage at the level of tragus (external auditory meatus and reference point for levelling at the lateral ventricles) or above when the three way tap is opened. The catheter is generally not opened to drainage until ICP is sustained >20mmHg for prolonged periods, although sometimes free drainage is ordered (Ladanyi & Elliot, 2008: 146).

The EVD may incorporate an ICP monitoring device, or there may be separate devices.

Decompressive Craniectomy

When patients fail to respond to the above aggressive medical interventions, decompressive craniectomy is then considered. Decompressive craniectomies involve the surgical removal of a bone flap from the skull to allow brain swelling occur without the lethal side effect of intracranial hypertension. The excised portion of the patient's skull is stored in their abdomen or in a tissue bank until the patient is stable. After intracranial hypertension has subsided, the bone flap can be reinserted.

GENERAL NURSING CONSIDERATIONS

DVT Prophylaxis

The incidence of venous thromboembolic events in patients who have suffered TBI is high (Saiki, 2009: 556). The precise methods for preventing DVT in TBI patients remain controversial, however the Brain Trauma Foundation (2007) recommend the use of sequential compression devices and compression stockings, unless lower extremity injury prevent their use.

The use of low weight molecular heparin is associated with an increased risk of expansion of intracranial haemorrhage (Brain Trauma Foundation, 2007).

Nutritional Support

It is well documented that, despite their often comatose state, TBI patients are hypermetabolic. It is therefore recommended that 140% of the resting metabolism be replaced in TBI patients (Zink & McQuillan, 2005: 42). Nurses have a pivotal role in ensuring that patients suffering TBI achieve and maintain adequate nutrition during their hospitalisation. Nurses should advocate for early placement of feeding tubes and the initiation of nutrition.

Efforts to maintain normogylcaemia should be taken as hypoglycaemia and hyperglycaemia can both exacerbate secondary injury (Saiki, 2009: 556).

Early commencement of enteral feeding is a key component in caring for critically ill patients as it increases mesenteric blood flow, maintaining integrity of the gut mucosa, and promotes gastrointestinal mobility and peristalsis (Ladanyi & Elliot, 2008: 147). Stress ulcer prophylaxis should also be commenced early.

Bowel Management

Promotion of healthy bowel function in TBI patients helps to prevent increases in intra-abdominal pressure and any consequent rise in intra-thoracic pressure and ICP (Ladanyi & Elliot, 2008: 148). Apperients should be administered once the patient is stabilised and can tolerate intake through a feeding tube.

Personal Hygiene

Regular eye and mouth care should be attended to avoid infection and maintain mucosal integrity. The endotracheal tube should be carefully positioned and frequently moved to alternate sides of the mouth to help prevent pressure sores of the lips.

In the acute phase of TBI, washing the patient could be done as required rather than routinely if ICP's are unstable.

Pressure area care and repositioning should be attended third hourly or as tolerated by the patient.

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