
Summary

Head injury is a common occurrence that often leads to raised intracranial pressure (ICP). While the best care for patients with head injury might be in specialist neuroscience units, limited places mean that nurses working outside these units might also encounter patients with raised ICP. This article describes the pathophysiology of raised ICP in relation to vital observations and nursing interventions.

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Pathophysiology of intracranial pressure

Compliance The skull is a rigid container enclosing different proportions of three incompressible volumes: brain tissue (80%), blood (10%) and cerebrospinal fluid (CSF) (10%) (Allan 2006). ICP is defined as the sum of the pressures that these three volumes exert in the skull (Allan 2006). In healthy adults, ICP is maintained nearly constant between 0mmHg and 15mmHg (Woodrow 2006). A mechanism known as compliance, or the modified Monro-Kellie hypothesis, compensates for small and brief increases of blood volume, CSF and brain tissue (Mokri 2001). Through this mechanism, when the content of the skull increases, the body responds by pushing blood into the venous sinuses in the brain, increasing the CSF reabsorption rate or moving CSF into the spinal column (Josephson 2004).

Therefore, in the normal range, ICP remains almost constant despite small increases in volume. However, at higher volumes, a decompensation point is reached, where for a smaller volume increase there is a larger increase in pressure (Andrews and Citerio 2004) (Figure 1). A level of ICP higher than 15mmHg is considered abnormal and is defined as raised ICP, or intracranial hypertension (Woodrow 2006).

Auto-regulation and cerebral perfusion pressure

Before discussing the pathophysiology and the clinical aspects of raised ICP, it is important to mention another important physiological mechanism that occurs in the brain: autoregulation of cerebral blood flow (CBF). While the brain contributes to only 2% of body weight, it accounts for 20% of the body’s consumption, at rest, of oxygen and glucose (Tortora and Grabowski 2002). Neurones in the brain produce energy almost exclusively by oxidising glucose.
Moreover, the brain does not store glucose. Therefore, a constant CBF is necessary to maintain a regular supply of oxygen and glucose (Tortora and Grabowski 2002). This is guaranteed by autoregulation, which is the ability of blood vessels in the brain to constrict or dilate to maintain a stable blood flow within the normal range of cerebral perfusion pressure (CPP), which is between 50-140mmHg (Dunn 2002) (Figure 2). CPP is closely related to ICP: it is defined as the difference between systemic mean arterial pressure (MAP) (usually varying from 50-150 mmHg) and ICP (Box 1).

According to this relationship, if ICP rises or MAP decreases, CPP decreases, and if MAP increases, CPP increases. If CPP decreases below 50mmHg it can lead to hypoxia (lack of adequate oxygen at tissue level) and ischaemia (lack of sufficient blood flow to the tissues). If CPP rises above 150mmHg it can cause cerebral oedema (abnormal accumulation of interstitial fluid) (Josephson 2004).

**Causes of raised ICP**

Causes of increased intracranial volume that raise ICP could involve (Dunn 2002):

- The brain – tumour, brain oedema or haemorrhage following a head injury.
- CSF – increased production, decreased absorption or impaired circulation of CSF.
- Blood – vasodilation induced by hypercapnia (an abnormally high level of carbon dioxide in blood), or venous failure as a result of cerebral venous thrombosis, superior vena cava obstruction or heart failure.

The causes of increased intracranial volume are summarised in Box 2.

**Subarachnoid haemorrhage**

A typical presentation in patients in acute clinical settings is a subarachnoid haemorrhage: a bleed in the subarachnoid space between the arachnoid mater and pia mater (two of the three meninges covering the brain and spinal cord), which normally contains only CSF. A subarachnoid haemorrhage can be caused by head injury, ruptured intracranial aneurysm or severe hypertension (Corwin 2006). The volume of blood accumulating in the subarachnoid space causes increased pressure on the surrounding brain tissue, therefore raising ICP.

**Herniation**

The cavity of the skull is divided into compartments by the infoldings (reflections) of the dura mater (Moore and Dalley 2005). An increase in ICP often establishes a difference in pressure between these compartments, forcing and compressing brain structures from a high-pressure to a low-pressure compartment. This process is called herniation (Allan 2006). Depending on which parts of the brain are herniated, different pathological effects can develop, such as non-reactive or dilated pupils, decreased conscious level or impairment of the gag reflex (Woodrow 2000, Allan 2006).

The physiological changes described above have a direct effect on clinical nursing observations and interventions, which are now discussed.
Vital observations

The initial aim of a nurse caring for a patient with confirmed or suspected ICP is to carry out a thorough clinical assessment. This is essential in guiding interventions to prevent further deterioration of the patient’s condition (Arbour 2004). The minimum documented neurological observations should include vital observations, level of consciousness, pupillary activity and limb movements (NICE 2007). This article focuses on vital observations.

Vital observations include blood pressure, pulse, respiration and temperature monitoring (Pemberton and Waterhouse 2006). Assessment of vital signs is fundamental for all patients. For individuals with raised ICP, changes in vital signs indicate brainstem pressure (Arbour 2004). These changes are explained below.

**Blood pressure** Several feedback systems regulate blood pressure in the body. One of these systems is controlled by the vasomotor area of the cardiovascular centre. This is a group of nerve cells in the medulla oblongata, located in the inferior part of the brainstem (Tortora and Grabowski 2002). The vasomotor centre controls the constriction of vasoconstriction and a consequent perfusion, which activates the ischaemic reflex. Previously discussed. This decreases medullary later stages, increased ICP reduces CPP, as blood pressure is relatively stable. However, in (Ganong 2003). In the early stages of rising ICP, medulla oblongata (Cushing or ischaemic reflex) controls the constriction of vasoconstriction in the inferior part of the brainstem (Tortora and Grabowski 2002). The vasomotor centre activates the ischaemic reflex. The result is vasoconstriction and a consequent rise in arterial pressure (Hickey 2002a).

**Pulse** The pulse rate is relatively stable in the early stages of increasing ICP. In later stages, however, bradycardia is often observed. Hickey (2002a) suggested that this probably results from compression of the vagal control mechanism in the medulla oblongata. However, Ganong (2003) proposed another mechanism for the appearance of bradycardia. In response to raised ICP, the vasomotor centre causes hypertension. Arterial baroreceptors (pressure-sensitive receptors) react to it by sending nerve impulses to the cardiovascular centre. This, in turn, increases vagal stimulation of the heart to decrease heart rate in an effort to reduce blood pressure. This is why the combination of hypertension and bradycardia rather than tachycardia is characteristically seen in patients with raised ICP (Ganong 2003).

**Respiration** Of the four vital observations, respiratory patterns provide the clearest indication of brain dysfunction (Russell 2008). The autonomic respiratory control centres are located in the lower pons and upper medulla of the brainstem (Guyton and Hall 2006). Increased ICP causes pressure on these areas, giving rise to respiratory changes. These include breathing patterns such as Cheyne-Stokes respiration (alternation of rapid rhythmic breathing with periods of apnoea) and cluster breathing (irregular respirations alternating with periods of apnoea) (Russell 2008). However, if a patient is ventilated, the ventilator might mask these abnormal patterns, making it impossible for the nurse to assess respiration (Hickey 2002a). The combination of hypertension, bradycardia and abnormal respiration is called Cushing’s triad (Hickey 2002a). It is a late sign of brainstem dysfunction, often accompanied by cerebral herniation (Hickey 2002a).

**Body temperature** The temperature of the body is controlled by the hypothalamus, which is located between the brainstem and the cerebrum (Tortora and Grabowski 2002). Major fluctuations in body temperature might indicate damage to the hypothalamus caused by raised ICP (Russell 2008). If ICP is raised, body temperature is generally high. However, it is important that the nurse is able to distinguish between hyperthermia caused by raised ICP and elevated temperature caused by infection (Hickey 2002a). This can be achieved using a diagnosis of exclusion; that is, by excluding sepsis by, for example, looking for evidence of pneumonia or by carrying out a blood culture (Agrawal et al 2007).

Independent nursing activities

In addition to monitoring, the nurse’s therapeutic goals when ICP is raised are to treat the underlying causes, manage factors known to increase ICP, monitor and manage ICP, support body systems and prevent secondary injuries to the brain and complications (Hickey 2002a).

**BOX 2**

<table>
<thead>
<tr>
<th>Causes of increased intracranial volume</th>
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<tbody>
<tr>
<td><strong>Brain</strong></td>
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<td>- Tumour</td>
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<td>- Oedema</td>
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<td>- Haemorrhage</td>
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<tr>
<td><strong>Cerebrospinal fluid</strong></td>
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<tr>
<td>- Increased production</td>
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<td>- Decreased absorption</td>
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<td>- Impaired circulation</td>
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<td><strong>Blood</strong></td>
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<tr>
<td>- Vasodilatation</td>
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<td>- Cerebral venous thrombosis</td>
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<tr>
<td>- Superior vena cava obstruction</td>
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<tr>
<td>- Heart failure</td>
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</table>

(Dunn 2002)

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Mestecky 2007). This discussion will focus on the independent nursing activities necessary to minimise the risk of further increases in ICP.

**Bed elevation** Patients with raised ICP are routinely cared for with their head elevated to 30° (Hickey 2002a, Cree 2003, Mestecky 2007). There are indications that this method decreases ICP by increasing the drainage of venous blood from the brain (Hickey 2002a). Bed elevation might also move CSF from the cranial to the spinal subarachnoid space (Fan 2004). However, mechanisms of change in cerebral venous drainage between the supine and elevated position are complex and vary between patients (San Millán Ruiz et al 2002, Schreiber et al 2003, Doepp et al 2004). Also, head elevation might reduce CPP to a level that can prevent autoregulation.

A systematic review by Fan (2004) recommends the use of 30° elevation to reduce ICP and monitor the effects on CPP in patients with a brain injury. However, the 11 studies included in this review were limited by a small sample size and low statistical significance. In an original study with a larger sample size, Ng et al (2004) found significantly lower ICP and constant CPP when 30° elevation of the head was used. Based on the available evidence, the effect of head elevation should be considered as a therapeutic intervention and its effect on ICP and CCP should be established in each patient.

**Patient movement** Nurses commonly reposition and turn patients. Changing a patient’s position might precipitate the Valsalva manoeuvre, which involves forced expiration against a closed glottis (Ganong 2003). This can increase pressure in the abdomen and in the chest, obstructing the drainage of venous blood from the brain. Hickey (2002a) suggested maintaining alignment of body parts during movement to avoid stimulating the Valsalva manoeuvre. In particular, maintaining nose and sternal alignment prevents jugular vein occlusion. However, this ‘log rolling’ technique requires five to six nurses, which increases cost and has not been researched fully (McGuinness 2007). Johnson (1999) suggested that, alternatively, one nurse can maintain head alignment while two nurses reposition the patient. Rising (1993) showed that although turning patients was associated with an increase in ICP, this increase was short-lived. To decrease the risk of increasing ICP, McGuinness (2007) encouraged the use of bolus sedation to coincide with repositioning.

This involved injecting the patient with a short-acting sedative to achieve a short and deep sedation. The nurse can assist in this by discussing the patient’s specific requirements with medical colleagues.

**Endotracheal suctioning** Nurses commonly perform endotracheal suctioning on ventilated patients with brain injuries, and this procedure is known to increase ICP (Kerr et al 1999, Gemma et al 2002). Therefore, suctioning has to be undertaken with care, balancing risks with therapeutic goals (Hickey 2002a). In a review of the literature, Pedersen et al (2009) collated a list of evidence-based recommendations for safe patient suctioning and avoidance of complications (Box 3).

The main recommendations include: suctioning only when necessary – the need for suctioning can be determined by observing the patient’s colour, presence of secretions in the upper respiratory tract, chest and abdominal movement, noisy and gurgling respirations, and chest auscultation (Hickey 2002a, Pemberton and Waterhouse 2006); using a suction catheter that occludes less than half the lumen of the endotracheal tube; using the lowest possible suction pressure; inserting the catheter no further than the carina, which is the keel-like cartilaginous ridge in the area where the trachea divides into two bronchi (Moore and Dalley 2005); suctioning for no longer than 15 seconds; performing continuous rather than intermittent suctioning technique; and avoiding 0.9% sodium chloride instillation.

**Bowel management** Management of the bowel is important in patients with raised ICP. Constipation increases intra-abdominal pressure and causes straining when defecating, therefore raising ICP (Hickey 2002a, Allan 2006). The patient’s specific condition and treatment can be risk factors for constipation. These include certain analgesics – for example opioids – fluid restriction, nil-by-mouth status, decreased bulk in the patient’s diet and prolonged immobility (Hickey 2002a). To prevent constipation, a specific bowel programme should be initiated on

### BOX 3

**Recommendations for safe endotracheal suctioning**

- Suction only when necessary.
- The suction catheter should occlude less than half the lumen of the endotracheal tube.
- Use low suction pressure.
- Insert the catheter no further than the carina.
- Suction for no longer than 15 seconds.
- Avoid intermittent suctioning.
- Avoid instillation of 0.9% sodium chloride.

(Pedersen et al 2009)
admission. This might involve increasing fluid intake, if not contraindicated, and administering stool softeners, mild laxatives, suppositories or enemas (Hickey 2002a).

**Clustering of activities** Many nursing interventions, such as the promotion of personal care, repositioning, linen changing and suctioning, can increase ICP. When carried out in sequence, they can have a cumulative effect on ICP (Mestecky 2007). Clustering of nursing activities should therefore be avoided. However, McGuinness (2007) suggested timing patient repositioning to coincide with changing bed linen, to minimise patient rolling, and ensuring that everything is ready before repositioning commences. Moreover, response to nursing activities is variable among patients (Mestecky 2007). If ICP monitoring is in place, the nurse can establish the effect of each activity and adjust the patient’s care routine accordingly (Hickey 2002a).

**Pain management** Excessive stimulation, such as pain, agitation and other unpleasant stimuli, lead to an arousal response. This increases cerebral blood flow and ICP because of the increased metabolic requirement of the brain (Arbour 1998, Cook 2008). Analgesics and sedation can control pain and agitation. Narcotics, such as morphine sulphate or fentanyl, are effective in controlling pain and reducing agitation (Hickey 2002a). These drugs can be particularly effective when administered in small and frequent doses (Arbour 1998). Benzodiazepines, such as midazolam, can also reduce patient anxiety. However, narcotics and benzodiazepines can accumulate in the body, resulting in an intrinsic narcotic effect, which can last long after drug administration has stopped, making neurological assessment difficult (Arbour 1998). To avoid this limitation, a short-acting narcotic such as remifentanil or a short-acting non-benzodiazepine sedative such as propofol is commonly used. Propofol has the added advantage of reducing cerebral metabolic rate and ICP (Marik et al 2002, Marik 2004).

Care should also be taken to limit painful procedures. For example, blood should be taken from an existing vascular access device, if possible (Arbour 1998).

**Environment control** In intensive care units, nurses have the resources to provide the best possible control of ventilation and blood pressure, which are not available to ward nurses. However, intensive care units are busy, noisy and bright, and this environment that is less than optimal for patients with raised ICP. Nurses can limit the effects of emotionally upsetting and unpleasant stimuli by controlling the

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Kerr ME, Weber BB, Sereika SM, Darby J, Marion DW, Ondoff PA (1999) Effect of endotracheal suctioning...
environment around patients, limiting their sensory stimulation and providing dimmed lighting and quiet surroundings (Woodrow 2000). Nurses should also limit loud noises and sudden shaking of the bed (Hickey 2002a).

Studies have shown that conversations that might be emotionally upsetting to patients, such as discussions on their prognosis, might increase ICP (Hickey 2002a). The nurse should ensure that upsetting conversations cannot be overheard. Even if a patient is comatose, the nurse and family members should always assume that the patient can hear and understand what is being said.

Gentle therapeutic touch, speaking in a soothing voice, pleasant conversations and even hand, back or foot massage can be useful. These interventions could be incorporated into the patient’s care plan if vital signs or ICP show a positive effect when monitored (Arbour 1998). Hickey (2002a) states that the value of therapeutic touch cannot be overemphasised, particularly when it is provided at the same time as uncomfortable but necessary procedures.

**Conclusion**

Raised ICP is a relatively common event in specialist and non-specialist acute care units. Knowledge of the current theories of the pathophysiology of raised ICP is essential so that nurses understand the mechanisms by which blood flow and pressure are maintained in the brain. This, in turn, enables the nurse to link variations in vital observations to changes in ICP and to initiate appropriate interventions. These should be based on current clinical evidence to provide safe and effective nursing management.

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**References**


