Managing children with raised intracranial pressure: part two (brain tumours and intracranial bleeds)


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Abstract
This article is the second of two examining the causes of increased intracranial pressure in children. Key features and management associated with brain tumours and intracranial bleeds are highlighted. The conditions are accompanied by illustrative case studies to give an idea of what children’s nurses may encounter in a patient presenting with raised intracranial pressure. Part one, published in December 2013, focused on the signs and symptoms of raised intracranial pressure and meningitis.

Aims and intended learning outcomes
This article discusses the presentations, assessment, investigations and management of children with brain tumours or intracranial bleeds who present with signs of raised intracranial pressure. After reading this article and completing the time out activities, you should be able to:
- Outline the features of headaches and associated symptoms that may be associated with brain tumours.
- Describe the management of a child presenting with a brain tumour.
- Identify the range of head injuries that can cause raised intracranial pressure.
- Discuss the signs and symptoms of haemorrhagic stroke in children.
- Describe the management of a child presenting with haemorrhagic stroke.

Introduction
The second article of two on managing children with raised intracranial pressure (Paul et al 2013) discusses brain tumours and intracranial bleeds – two of the most important causes of acute or subacute increases in intracranial pressure – and their key clinical features and management strategies. The most common cause of raised intracranial pressure seen in clinical practice is likely to be a serious head injury and therefore it is vital that the symptoms are identified and escalated appropriately.

Now do time out 1.

Brain tumours
Intracranial tumours are a rare but important cause of raised intracranial pressure in children. Most are primary brain tumours, that is, they are derived from the types of cells, neurons and glial cells, that make up the brain, although other cancers, such as acute lymphoblastic leukaemia, may also involve the central nervous system.

It is estimated that ten children and young people in the UK are diagnosed with a brain tumour each week; however, due to lack of awareness about the condition, in the UK it may take up to three times longer to diagnose a brain tumour in children compared with other countries such as the United States (HeadSmart 2011, Wilne et al 2010). The HeadSmart campaign was launched to improve awareness among health professionals and the public.

The initial presentation of brain tumours in children frequently mimics more common and less serious childhood conditions and illnesses, resulting in diagnostic
Idiopathic intracranial hypertension, or pseudotumor cerebri, is characterised by increased intracranial pressure in the presence of normal cerebrospinal fluid studies. The patient is usually an adolescent girl who is overweight and presents with severe headache and visual disturbances that may become aggravated by change of position or Valsalva manoeuvre.

Normal neuroimaging and a neurological examination reveal no localising signs. However, papilloedema (blurred disc margins on fundoscopy) may be present. During lumbar puncture, raised cerebrospinal fluid opening pressure (above 21cm H₂O) is noted.

The aim of treatment is to minimise visual loss and relieve the headache. Among the treatment options are medical management (acetazolamide, steroids and serial lumbar puncture) or surgical management (optic nerve sheath decompression or diversion procedures, such as lumboperitoneal or ventriculoperitoneal shunts).

(Schexnayder and Chapman 2006)

difficulty and a prolonged time to diagnosis. Conditions with similar symptoms include new-onset seizures, unsteadiness on feet coincidentally after a viral illness, deterioration in school grades thought to be secondary to hearing or visual difficulties, or gradual loss of sensation in a limb (Wilne et al 2010). Hence, there is a potential for delay in detection.

Brain tumours are the second most common malignancy in children and young people in the UK after leukaemia aged under 14 years (Cancer Research UK 2010), and has an incidence of five per 100,000 (around 500 cases per year) in those aged up to 18 years (HeadSmart 2011). They are the most common cause of cancer-related death in childhood (about 100 deaths/year). A total of 70% of survivors have abnormal neurological function, 30% with pronounced disability (Pietilä et al 2012).

A delay in diagnosis is associated with a poor neurodevelopmental outcome. A positive trend has been noted due to the HeadSmart campaign in the UK, with time to diagnosis being reduced from 9.3 weeks to 7.5 weeks one year later, with a further reduction announced in June 2013 to 6.9 weeks (Paul and Walker 2013, Wilne et al 2013).

It is important to recognise that idiopathic intracranial hypertension can be confused with a brain tumour (Box 1, also Case studies 1 and 2, and Figure 1, page 32).

Clinical presentation Headache is a common presenting feature and is seen in 30-60% of children with brain

Case study 1

A 12-year-old boy presented with a three-month history of frontal headaches and vomiting. His symptoms had worsened over time and he was now vomiting most days, particularly in the mornings when his headache was at its worst. He had missed two weeks of school this term due to illness. There had been no deterioration in his behaviour or school performance. Three weeks previously he had developed a squint, and was now complaining of double vision.

On examination, he had paralysis of the left abducens (sixth cranial nerve), meaning that he could not abduct his left eye. He had bilateral papilloedema. He was sent for an urgent computed tomography scan and, which showed an intracranial mass, following which he had a magnetic resonance imaging scan. The scan shows a mass in the medulla, which extends into and obstructs the fourth ventricle. This resulted in acute obstructive hydrocephalus – a neurosurgical emergency.

He was commenced on dexamethasone to reduce intracranial pressure by reducing cerebral oedema, and transferred to a neurosurgical unit for tumour resection. Once the diagnosis of brain tumour was given to the family, the nurse looking after the child explained what to expect in the neurosurgical unit, including that the boy was likely to be taken for urgent surgery. She also spoke to the ward in charge at the neurosurgical unit explaining how devastated the family was with the diagnosis as they had thought it would be something less serious, and the need for ongoing support.

Histology subsequently showed the tumour to be a grade 4 medulloblastoma, and the boy commenced chemotherapy and radiotherapy after his surgery. He was reported to be under treatment and stable at the time of writing.

Case study 2

A two-and-a-half-year-old girl attended the emergency department (ED) with a five-day history of frequent headaches, which sometimes woke her from sleep, and vomiting. Examination was unremarkable, and she was diagnosed with a viral illness and discharged.

Her symptoms persisted for a further week before she attended again, and was discharged once again. The next day, she had a complex partial seizure and was brought to the ED by ambulance with a decreased consciousness level. She was triaged and the paediatric team was requested to attend urgently.

Neurological observations were carried out every 15 minutes by the ED nurse. The girl was intubated for a computed tomography (CT) scan and her family was kept updated by the nurse who supported them while the news about a brain tumour was given to them.

The large frontotemporal mass was causing a midline shift, as is evident on the CT (Figure 1, page 32). The girl was transferred urgently to the local neurosurgical unit for debulking. Histology showed the mass to be a supratentorial primitive neuroectodermal tumour, and imaging revealed spinal metastases. She commenced chemotherapy and radiotherapy following tumour debulking. She was reported to be under treatment and stable at the time of writing.

tumours. It may result from either raised intracranial pressure due to mass effect and hydrocephalus, or infiltration of tissues such as the meninges that cover the brain (Barlow and Stewart 2007). Particular features of a child’s headache or associated signs and
symptoms will raise suspicion of a brain tumour as the cause (Box 2).

There is a wide variety of other presenting symptoms as outlined by the HeadSmart symptom card (Figure 2), occurring in isolation or in combination, and a large proportion of children younger than four may not have headache as a presenting feature at the time of diagnosis (Wilne et al 2006). Focal neurological signs, such as abnormal eye movements, focal seizures, loss of previously acquired developmental skills or sudden change in personality may all be presenting features (Wilne et al 2006, Paul and Walker 2013).

Some brain tumours are also associated with endocrine disorders, such as precocious puberty (Wilne et al 2010, Stephen et al 2011), and boys with the condition should always have neuroimaging; this is because a hormonal imbalance due to a brain tumour can trigger early puberty and diabetes insipidus (Wilne et al 2010, Bin-Abbas et al 2001). Although some children with brain tumours present with sudden acute onset of symptoms, in most cases they develop insidiously and therefore can be more difficult to diagnose (Wilne et al 2006, Paul and Walker 2013). Now do time out 2.

2 Triage

You are triaging an unwell child with headache and vomiting. In what circumstances would you request an urgent involvement of the anaesthetic team?

Management The initial management of a child presenting acutely with a brain tumour follows, as with all cases, an ABCD approach (airway, breathing, circulation and disability). All children with decreased consciousness levels should receive high-flow oxygen via an appropriate apparatus and some may need airway protection (Advanced Life Support Group (ALSG) 2011).

Children who present acutely with seizures or with a decreased consciousness level may require intubation for airway protection. Even children who are maintaining their airway but have decreased consciousness may need to be considered for elective intubation, particularly if they need transfer to another unit. An experienced anaesthetist should always be involved in managing a child with a Glasgow Coma Scale score below eight (Table 1, page 36), as they may suddenly lose airway control.

The centres responsible for controlling breathing and circulation, and many other functions, via the autonomic nervous system, are in the brainstem. Injury to this region, from direct tumour infiltration or as the result of bleeding or compression, can therefore lead to severe respiratory and cardiovascular compromise. Particular combinations of abnormal observations, such as irregular respiration, bradycardia and hypertension (Cushing's triad), can indicate raised intracranial pressure and high risk of imminent brainstem herniation (Marcoux 2005). Cushing's triad occurs as a result of cerebral ischaemia, causing peripheral vasoconstriction (Marcoux 2005).

Once the child's airway, breathing and circulation are stable, it is important to assess neurological function at 15-minute intervals until the condition stabilises. Children with abnormal consciousness should have a Glasgow Coma Scale score assessment.
at presentation, and every 15 minutes while the score remains abnormal. Capillary blood glucose should be measured at presentation. Seizures should be treated promptly according to the Advanced Paediatric Life Support protocol, protecting the child from injury should further seizures occur (ALSG 2011). Once stable, urgent neuroimaging should be arranged.

In addition to supporting the therapeutic interventions, such as helping to prepare and administer medicine and fluids, nurses should keep the parents updated about their child’s condition because medical teams are often occupied stabilising the child and arranging for a transfer.

Evidence of raised intracranial pressure, clinical or radiological, should prompt measures to reduce this. The higher the intracranial pressure, the lower the cerebral perfusion pressure, resulting in hypoperfusion of the brain and worse neurological outcome.

Patients should be nursed with a head-up tilt of 30°. Intravenous mannitol or hypertonic saline can be used to reduce intracranial pressure, with evidence suggesting that 3% saline is safer and of equal efficacy to mannitol (Upadhyay et al. 2010), although it is less widely available. Practitioners may prefer to consult the regional specialists before administration of 3% saline. Intubated patients should have a target partial pressure of CO₂ in the blood (PaCO₂) at the lower end of the normal range (about 4.5kPa); this is useful in reducing intracranial pressure in the first 24 hours (Kim et al. 2006).

In cases where there is swelling of the brain tissue, such as oedema around a tumour, there may be a benefit in giving dexamethasone for urgent reduction of raised intracranial pressure (French and Galicich 1964, Chen 2012). This should be done with the advice of the neurosurgical or paediatric oncology team. Dexamethasone reduces vasogenic oedema around the tumour and has anti-inflammatory properties.

These children will then require transfer to a tertiary centre or local specialist unit for definitive management. This may involve a combination of neurosurgery, radiotherapy and chemotherapy. Treatment details and prognosis will depend on tumour site and type, which is beyond the scope of this article. Rapid stabilisation, facilitation of prompt transport to specialist care, and support for children and families should be the priorities for nursing and medical staff. These require careful planning, communication and involvement of appropriate personnel such as retrieval teams.

Although during the initial phase, the aim is to facilitate rapid transfer to a neurosurgical unit, the paediatric early warning score (PEWS) can also be used in a haematology/oncology unit with an associated multidisciplinary action algorithm. Using PEWS will reduce the problems of inter-observer variation by providing objective evidence when discussing a case with other specialists such as anaesthetists and neurosurgeons, and ensure faster referral of children who are clinically deteriorating and require urgent medical attention.

Figure 2 HeadSmart card for detecting signs of brain tumour

Reproduced with permission from HeadSmart
presentations can cause a raised intracranial pressure and any or all the symptoms described in part one, may be observed (Paul et al 2013).

"Head injury" National Institute for Health and Care Excellence (NICE) (2007) guidelines define head injury as any trauma to the head other than superficial injuries to the face, and it is the most common type of traumatic injury seen in children (Paul et al 2011). About half a million patients aged 16 years or younger present with head injuries to emergency departments (EDs) in the UK; about 50,000 children a year are admitted to hospital, which is about 10% of all childhood admissions (Reed 2005, Paul et al 2011).

It is important to check for and detect the signs of raised intracranial pressure, outlined in part one (Paul et al 2013), as there is a risk of long-term neurological problems or even death. Head injury with intracranial bleeding is the main cause of trauma-related death in children. In a study of 43,904 children aged under 18 years with blunt head trauma, computed tomography (CT) scans showed intracranial haemorrhage in 4.4% of previously healthy children (n=665) without any bleeding disorder (Lee et al 2011). It may be noted that 25% of children with skull fractures do not lose consciousness (Paul et al 2011).

Continuing professional development

Immediate help, also enhancing communication between multidisciplinary teams (Demmel et al 2010). Now do time out 3.

Intracranial bleeds

Intracranial bleeds can be separated into trauma after a head injury or non-traumatic causes. Both presentations can cause a raised intracranial pressure and any or all the symptoms described in part one, may be observed (Paul et al 2013).

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Now do time out 3.

Patient management

What supportive measures do you consider useful in a child with raised intracranial pressure and intracranial haemorrhage? Please list a few from your experience in managing such patients or from the discussion in the previous section.

It is important that health professionals remain aware of the range of injuries that cause intracranial bleed/head injury leading to raised intracranial pressure. While skull fractures in children provide a high index of suspicion, it is necessary to remain aware of the range of injuries that could be the cause, as described below (Khilnani 2004).

"Extradural haematoma" This occurs mainly at the convexity or posterior fossa of the brain, but could occur due to a middle meningeal arterial bleed or tearing of bridging veins. It arises following a compression/decompression injury, acceleration/deceleration injury or direct trauma. Extradural haematoma usually present initially with a normal childhood Glasgow Coma Scale score, followed by progressive deterioration in consciousness, followed by recovery (lucid interval), and subsequent further loss of consciousness and continuing progressive neurological deterioration (Khilnani 2004).

"Subdural haematoma" This can be an acute, subacute or chronic presentation. Acute subdural haematoma presents clinically with severe depression of consciousness level with focal deficit and cerebral injury, and is associated with poor outcome (Khilnani 2004). Subacute subdural haematoma presents as progressive deterioration of consciousness and/or neurological function; timely detection and neurosurgical intervention can be associated with a good outcome (Khilnani 2004).

Chronic subdural haematoma is relatively common in paediatric traumatology (it is more common in children under two years of age) and may also be seen in babies with non-accidental injury (shaken baby syndrome); it is amenable to surgical treatment (Khilnani 2004, Paul et al 2011).

"Intracerebral haematoma and contusions" These can have variable and multiple presentations ranging from normal Glasgow Coma Scale score and mildly depressed level of consciousness to severe neurological deficit, seizures and coma. These injuries may require surgical intervention, or may be managed conservatively depending on the extent and the nature of damage. Outcome is variable (Khilnani 2004).

"Diffuse brain injury" Head injury can lead to minor concussion or diffuse axonal injury, depending on the nature of the impact. A concussion describes temporary loss of neurological function immediately after trauma with no radiological findings; it is associated with complete recovery. However, diffuse axonal injury is usually caused by shearing movements of the brain during acceleration/deceleration injuries; it is almost always associated with a poor outcome (Khilnani 2004).

The latter should be suspected in a patient whose poor clinical neurological status does not correlate with CT scan findings. Magnetic resonance imaging is advisable in these cases and will demonstrate lesions in the corpus callosum, the rostral part of the pons or in the internal capsule (Khilnani 2004).

Now do time out 4.
Cerebral oedema This can occur after a traumatic brain injury. It leads to an expansion of brain volume, causing raised intracranial pressure; this impairs cerebral perfusion and oxygenation, and contributes to additional ischaemic injuries (Khilnani 2004).

Two major types of traumatic brain oedema are classically described: vasogenic due to blood-brain barrier disruption resulting in extracellular water accumulation, and cytotoxic/cellular due to sustained intracellular water collection (Untenberg et al 2004).

A third type, osmotic brain oedema, has also been described and is caused by osmotic imbalances between blood and tissue (Untenberg et al 2004). A hydrocephalic oedema/interstitial brain oedema may rarely occur after traumatic brain injury and is related to an obstruction of cerebrospinal fluid outflow (Untenberg et al 2004).

Haemorrhagic stroke Haemorrhagic stroke includes spontaneous intracerebral haemorrhage, parenchymal or intraventricular haemorrhage and non-traumatic subarachnoid haemorrhage. Of all causes identified, the most common are cerebral vascular abnormalities. They occur in 40-90% of children experiencing haemorrhagic stroke, and include arteriovenous malformations, cavernous malformations and aneurysms (Jordan and Hills 2011).

Other causes of haemorrhagic stroke that are less commonly seen include bleeding disorders, thrombocytopenia and brain tumours. Stroke, which has an incidence of 2-3 per 100,000 children, is among the top ten causes of death in childhood and is almost as common as brain tumours (Jordan and Hills 2007). Haemorrhagic stroke accounts for about 50% of stroke in childhood (Jordan and Hills 2011). Overall mortality is about 25% in children (higher than ischaemic stroke) and significant disability is present in 42% of those who at first survive (Jordan and Hills 2011). To avoid potentially devastating neurological sequelae, prompt diagnosis and management are essential (Case study 3).

Clinical presentation The onset of symptoms in children with haemorrhagic stroke is often rapid and dramatic, developing over minutes to hours. At times, however, symptoms of this condition can be insidious, developing over several hours to days. Stroke in children is rare and often under-recognised by healthcare providers (Jordan and Hills 2011).

Intraparenchymal haemorrhage Frequent presents with signs of raised intracranial pressure, such as headache and vomiting. Seizures occur in up to one third of cases. Focal neurological signs may be absent and symptoms can be non-specific in infants (Jordan and Hills 2011).

Subarachnoid haemorrhage Children can present with symptoms and signs of meningeal irritation, including headache of sudden onset, neck stiffness and photophobia (Jordan and Hills 2011). Again, these conditions may not be apparent in young infants, in whom signs of raised intracranial pressure or meningeal irritation are much less specific. Autonomic dysfunction may be a feature.

Seizures occur in about 20% of cases at presentation (Jordan and Hills 2011). In a study by de Ribaupierre et al (2008), acute headache was the presenting feature in 77% of patients – 23% had no neurological signs at time of onset, 27% had moderate and 50% had severe symptoms. A total of 14% presented with new-onset seizures, however, 41% had a normal Glasgow Coma Scale score.

Subdural haemorrhage The symptoms and signs are described earlier. While most cases are related to trauma, some may occur spontaneously, particularly in children with coagulopathy (Khilnani 2004).

Management The initial aim should be to stabilise the child with raised intracranial pressure, whether the cause is haemorrhagic stroke or intracranial bleed after head injury, using an ABCD approach; this includes a quick assessment of neurological status with the AVPU scale (alert, voice, pain, unresponsive) while assessing disability (‘D’) (ALSG 2011). The steps described for managing an unwell child with a brain tumour will be the same where the child has signs of raised intracranial pressure.

The Paediatric Glasgow Coma Scale (Table 1, page 36) is an important tool in the initial assessment and monitoring (in combination with PEWS) for deterioration (Worrall 2004). However, it should not be used in isolation but in conjunction with other parameters, such as pupil size and reactivity, limb movements, respiratory rate and oxygen saturation, heart rate, blood pressure, temperature and unusual behaviour (Scottish Intercollegiate Guidelines Network (SIGN) 2009).

Case study 3

A 12-year-old normally fit and well girl presented with a sudden-onset posterior headache following a physical education lesson at school. She was practising a headstand and had no history of injury. After onset of the headache, she was hypertensive (170/82mmHg), had severe neck pain, vomited six times and had a fluctuating Glasgow Coma Scale score of between 14 and 15.

Because of these signs of raised intracranial pressure, she had a computed tomography scan, which found a subarachnoid haemorrhage. Her blood count and clotting profile were reported as normal. She was transferred to a neurosurgical unit for further treatment. She had surgical evacuation of the bleed and recovered well.
It is important to speak to the family to find out what the child’s behaviour is usually like before deciding that it has changed after the injury (SIGN 2009).

Another neurological assessment scale that can be used in children with head injury is the Adelaide Coma Scale. This was found to be a more appropriate tool in a descriptive multiple-case study for children younger than five years because it considered the child’s age, hence taking neurological immaturity into account (Westbrook 1997).

Neuroimaging should be arranged within the first 24 hours of presentation because the CT scan is positive for intracranial bleeding in 95% of children with haemorrhagic stroke (Beck et al 2006). However, the chance of detection of intracranial bleeding falls to 50% of cases if the scan is carried out after a week, with the consequence that many children fail to be diagnosed with haemorrhagic stroke (Beck et al 2006).

Lumbar puncture may be done once the child is clinically and neurologically stable, and cerebrospinal fluid (CSF) should be examined for the presence of xanthochromia, which is yellow discoloration indicating the presence of bilirubin. This can be used to differentiate in vivo haemorrhage from a traumatic lumbar puncture (Bederson et al 2009). After an intracranial bleed, lumbar puncture will show blood in the CSF, which will also happen in the case of a traumatic lumbar puncture, that is, if the operator has hit blood vessels while inserting the needle. Raised bilirubin levels indicate an intracranial bleed (Bederson et al 2009).

NICE (2007) guidelines should be followed while ordering a CT scan for traumatic head injuries to prevent the potential harm posed by unnecessary exposure to radiation.

Nurses play an important role in managing children where an intracranial haemorrhage has been identified. Children admitted with a head injury or with signs of raised intracranial pressure should have neurological observations carried out at the following intervals, unless otherwise indicated (NICE 2007, SIGN 2009):

- Half-hourly for two hours.
- Hourly for four hours.
- Two-hourly for six hours.
- Four-hourly thereafter until intracranial pressure has been normalised.

Neurological observations will need to revert to half hourly if the child’s Glasgow Coma Scale score drops at any time.

Supportive measures include avoiding hyperthermia, monitoring blood glucose (avoid hyperglycaemia), monitoring blood pressure, the appropriate use of anticonvulsants and management of intracranial pressure (NICE 2007, SIGN 2009). Attempts to treat raised blood pressure aggressively with pharmacological agents should be avoided because reducing systemic blood pressure in the face of raised intracranial pressure will decrease the cerebral perfusion pressure and risk further cerebral ischaemia (Bederson et al 2009). If there is any concern about circulatory fluid volume, cautious administration of fluid boluses in aliquots of 10mL/kg is necessary (ALSG 2011).

Any ongoing intracerebral haemorrhage may be accompanied by an increase in body temperature and this is usually resistant to antipyretic agents (Bederson et al 2009). Attempts should be made to maintain a normal body temperature, unless therapeutic hypothermia is specifically requested by the paediatrician. Treatment of an intracerebral haemorrhage requires a multidisciplinary approach. If there is an underlying vascular malformation, an interventional neuroradiologist or neurosurgeon will need to be involved. The presence of life-threatening mass effect necessitates immediate surgical treatment.

The child with severe head injury often needs transfer to the radiology department for neuroimaging.

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### Table 1 Paediatric Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Area assessed</th>
<th>Score</th>
<th>0-23 months</th>
<th>2-5 years</th>
<th>&gt;5 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Best verbal response</td>
<td></td>
<td>Response &lt;1 year</td>
<td>Response &gt;1 year</td>
<td></td>
</tr>
<tr>
<td>Eye opening</td>
<td>4</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>To shout</td>
<td>To speech</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>To pain</td>
<td>To pain</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>None</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Best motor response</td>
<td>6</td>
<td>Normal movement</td>
<td>Obey command</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>Localises pain</td>
<td>Localises pain</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>Flexion withdrawal</td>
<td>Flexion withdrawal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>Flexion – abnormal (decorticate)</td>
<td>Flexion – abnormal (decorticate)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Extension (decerebrate)</td>
<td>Extension (decerebrate)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>None</td>
<td>None</td>
<td></td>
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</tbody>
</table>

It is important to speak to the family to find out what the child’s behaviour is usually like before deciding that it has changed after the injury (SIGN 2009). Another neurological assessment scale that can be used in children with head injury is the Adelaide Coma Scale. This was found to be a more appropriate tool in a descriptive multiple-case study for children younger than five years because it considered the child’s age, hence taking neurological immaturity into account (Westbrook 1997). Neuroimaging should be arranged within the first 24 hours of presentation because the CT scan is positive for intracranial bleeding in 95% of children with haemorrhagic stroke (Beck et al 2006). However, the chance of detection of intracranial bleeding falls to 50% of cases if the scan is carried out after a week, with the consequence that many children fail to be diagnosed with haemorrhagic stroke (Beck et al 2006). Lumbar puncture may be done once the child is clinically and neurologically stable, and cerebrospinal fluid (CSF) should be examined for the presence of xanthochromia, which is yellow discoloration indicating the presence of bilirubin. This can be used to differentiate in vivo haemorrhage from a traumatic lumbar puncture (Bederson et al 2009). After an intracranial bleed, lumbar puncture will show blood in the CSF, which will also happen in the case of a traumatic lumbar puncture, that is, if the operator has hit blood vessels while inserting the needle. Raised bilirubin levels indicate an intracranial bleed (Bederson et al 2009).

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The child with severe head injury often needs transfer to the radiology department for neuroimaging.
and sometimes to specialist services located in tertiary centres. In a retrospective study of bedside PEWS used during the transfer of children between, for example, the ED and the radiology department or specialist neurosurgical centre, it was found to be a useful tool, indicating the need for significant intervention in situations where the score was high (9.7 versus 3.44) (Page-Goertz and Meyer 2012).

**Prognosis**
Mortality of haemorrhagic strokes in children remains high, between 6% and 38%, depending on the primary lesions. Poor prognostic factors include reduced Glasgow Coma Scale score, infratentorial haemangiomas, the presence of intraventricular haemorrhage and younger age (Jordan and Hills 2007). The risk of having another intracranial bleed in survivors of subarachnoid haemorrhage (SAH) remains high – up to 30% in the first four weeks after the initial SAH (Beck et al 2006).

**Conclusion**
Children presenting with raised intracranial pressure can pose a diagnostic as well as management dilemma, especially where younger children present with non-classical symptoms. It is important to make a careful assessment of neurological status, keeping in mind the signs and symptoms of raised intracranial pressure. If there is a suspicion of raised intracranial pressure, urgent neuroimaging is necessary. Most children will need admission to hospital and some will need to be transferred to neurosurgical units.

It is hoped that the use of case studies to illustrate these clinical conditions will help nurses managing children with brain tumours and intracranial bleeds to carry out a thorough assessment and enable appropriate management along with recognition of early deterioration. An early diagnosis and appropriate treatment is associated with better outcomes.

Now do time out 5.

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**References**


**Self-assessment questionnaire**

Now that you have completed the article you might like to complete the questionnaire on page 38. To write a practice profile go to ncpyp.rcnpublishing.com