Neurological observations in infants, children and young people

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Nursing Children and Young People

Neurological Observations in Infants, Children and Young People - Part 1

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Abstract:	Caring for infants, children and young people with an acquired head injury can be challenging due to their developing brain and reliance on families/carers. In such a diverse society and faced with exposure to different development stages, consistency of neurological observations is a complex challenge for children's nurses Inconsistency in neurological observations can have huge impacts on the management of patients with an acquired brain injury. In this two-part series, we will explore why performing neurological observations on infants, children and young people can be challenging, and how we can encourage consistency to optimise the management of patients with neurological deficit. In addition, we will be offering advice on how overcome diverse challenges that increases the accuracy of neurological observations in children and young people.
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Aims and Learning Outcomes

In this two-part series we will be exploring the threat posed both by acquired brain injuries (ABI) and outlining the important assessment measures that need to be completed. Part one of the Neurological Observations in Infants, Children and Young People series aims to raise awareness of why infants, children and young people are vulnerable to acquired brain injuries, including injuries not related to brain/spinal injury causes. It will explore the pathophysiology of acquired brain injuries and the impact on a child and young person. Part 2 of the series will then look at the neurological observations in detail, relating back to physiology and how we can optimise neurological observations to increase accuracy and consistency.

After reading Part 1 of the series and completing the necessary time-out activities, you should be able to:

- Discuss of the vulnerability of infants, children and young people being diagnosed with an ABI.
- Explain different types of ABI and mechanisms of injuries.
- Explore the pathophysiology of ABI – including increasing intracranial pressure and the Monro-Kellie Doctrine.
- Discuss impacts of increasing intracranial pressure on children and young people, including brain herniation.

Time-out activity 1:
It is beyond the scope of this article to reiterate complete anatomy and physiology of the neurological system. However, to refresh your memory visit https://nurselabs.com/nervous-system/. It is important to have adequate knowledge about the nervous system to understand the pathophysiological mechanisms that occur resulting from an acquired brain injury.

Introduction

Headway (2023) defines ABI as any situation that causes an injury to the brain since the point of birth. These injuries can be caused by traumatic or non-traumatic reasons and does not include injuries to brain that occur during foetal development.

Traumatic brain injuries (TBI) are the most common type of ABI and is defined as any alteration to brain function or pathology because of an external force. Common causes include:

- Road traffic collisions
- Falls
- Sport related injuries
- Assault – including child abuse and shaken baby syndrome.

ABI are not just caused by external forces but can be caused by internal pathological changes altering neurology. These can be a combination of metabolic changes to internal lesions that develop over a period. Common causes include:

- Infections such as meningitis
- Hypertension
- Status Epilepticus
- Brain Tumours
- Reduced cerebral blood flow caused by underlying conditions such as cardiovascular conditions
- Toxins
- Strokes

The incidence of ABI varies depending on the causes. According to Headway (2022), between 2019-20 there were 356,699 admissions in the UK related to acquired head injuries in all ages. However, rates can differ depending on interpretation of what classifies as a head injury, meaning that an accurate rate of head injuries in children and young people proves difficult. According to Trefan et al (2016), in 2012-2013 there were 34,932 hospital episodes involving children and young people that related to an ABI across the UK. No further up to date statistics specifically looking at paediatric incidences can be found.

Understanding your patient's underlying health condition or complications resulting from preventative or therapeutic treatments is critical, especially as any patients could be vulnerable to neurological deterioration. Examples include:

- Patients who acquire conditions relating to impaired detoxification, metabolic and/or excretion ability, such as a patient in liver or/and renal failure or have an underlying metabolic disorder. These conditions increase the risk of blood-brain barrier interruption due to increasing toxicity or altered blood pH.
- Patients who acquire conditions relating to respiratory or cardiovascular impairment that could reduce cerebral perfusion.

Time Out 2

Types of head injuries

Go to Headway: The Brain Injury Association website and look through Types of Brain Injury: https://www.headway.org.uk/about-brain-injury/individuals/types-of-brain-injury/ . Make notes on the different types of head injuries you are likely to see in infants, children, and young people.

Acquired Brain Injuries in Children and Young People

Majority of ABI in children are mild and are often related to falls. The ever-changing development of the skull, face, brain, and neck muscles can increase the susceptibility of children to distinctive types of head injuries, often not associated with adults (Araki, Yokota and Morita, 2017). For example, in relation to the size of a child's body, the head is heavy, large and rests on a poorly supported neck, due to weak muscles and ligaments, therefore both head and cervical spine injuries may easily occur. Children therefore have a greater head-to-body weight ratio and weaker neck musculature and so, this may lead to greater chance of acceleration/deceleration injury of the brain upon impact and a higher vulnerability to shearing forces within the brain (Hung, 2020). In addition to this, due to the heavy

weight of the head in comparison with the body, young children are much more likely to fall head-first and thus, obtain head injuries via this mechanism (Joyce, Gossman and Heucker., 2022).

Additionally in children, the scalp has higher vasculature, which has the potential for lethal blood loss as even a small reduction of blood volume may result in haemorrhagic shock, especially in neonates, infants, and toddlers (Clarke and Sokoloff, 1999). This may occur without the presence of external bleeding and therefore, not be an obvious initial thought when the child or infant presents in the clinical setting, hence highlighting the significance of neurological assessment, especially in trauma scenarios.

Like the brain, the spine develops progressively and only resembles an adult spine at approximately eight to nine years of age (Copley et al., 2018). Due to this, many spinal injuries in paediatrics occur in the cervical region of the spine; in younger patients, subluxations or dislocations of the cervical spine are more commonly seen in the upper cervical region and associated with neurological injury (Meoded et al., 2011).

During the last thirty years, an expansion of neuroimaging research demonstrates that in adolescence the brain remains in a growth and development period, contrary to the longstanding belief that the brain was fully matured by puberty (Sowell et al., 2001). The frontal lobes, responsible for planning, working memory and impulse control, are one of the last aspects of the brain to mature, thus, may not be fully developed until halfway through the third decade of life (Sowell et al., 2001). This suggests that there is a link between early age and a lack of maturity in relation to judgement, impulse control and risk-taking and therefore, the younger the individual, the more likely they are to act on impulse and not think about the risks associated with actions, however, this peaks during adolescence (Albert et al., 2013). Due to the undeveloped frontal lobe in relation to risk-taking and decision-making children and young adults (under the age of 25) are more vulnerable to acquiring head injuries – especially traumatic brain injuries (TBI), which are often caused by a blow or other traumatic injury to the head or body.

In addition, infants and younger children are also at an increased risk of infection, due to their immunological vulnerability. This is because their immune systems are not yet fully developed and hence, are more susceptible to infection (Maródi, 2006). Although certain protective antibodies are passed from the mother to the foetus via the placenta, the levels of antibodies in the foetus' blood are not high enough to fight some infections (Palmeira et al., 2012). Example of such infections include bacterial meningitis, which can lead to septicaemia and thus, brain injury, viral infections (for example: cytomegalovirus, Epstein-Barr, chicken pox or shingles) can also cause great harm to the brain through encephalitis, which is a swelling or irritation of the brain.

The difficulty with assessing a child neurologically, who are suspected to have an ABI, especially early in life is that they may appear to be "well" within that moment, but as their age increases, cognitive and behavioural problems may begin to appear (Varshneya et al., 2019). In adults, immediate effects from an ABI can occur, but for

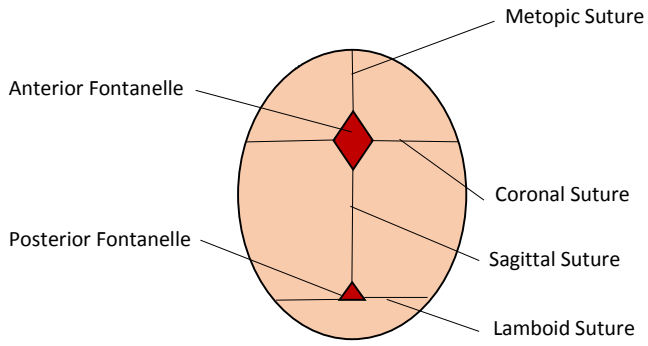
children and infants, this can be very different and may take much longer, until the injury becomes more obvious as it progresses. This is because due to children's brains developing until late adolescence, it is sometimes only when the brain fully develops that the true extent of a brain injury can be known. Additionally, it can be difficult for a caregiver to deduce if their child requires medical attention or for even general practitioners to decide upon referrals, especially in the early stages of an event. For example, in early meningitis, infants may present with vomiting, off their feeds, pyrexia and irritability, which of course, is highly like a variety of common childhood illnesses both bacterial and viral (Kim., 2010). Therefore, early detection may be prevented. This demonstrates the importance of a thorough neurological examination, in addition to neuroimaging where appropriate, to reduce the risk of missing injury. We will explore in-depth neurological examination for infants, children, and young adults in part 2.

Intracranial Pressure

Within the skull, the intracranial space is occupied principally by brain matter, which constitutes 80-90% of the volume. Cerebrospinal fluid (CSF) occupies 6-10%. Blood occupies the least amount of intracranial volume, 4-10%%. We often refer to this as the Monro-Kellie doctrine.

Each of these components are contained within the skull, a fixed cranial vault that provides protection and support for the cranial contents. In infants and toddlers up to the age of two, the skull is separated by fibrous bands called sutures which is important as their brains will be rapidly developing and growing (Figure 1). From the age of two, the skull plates fuse together leaving no gaps (Stanford Medicine Children's Health, 2022).

Figure 1 – Infant skull showing suture sites

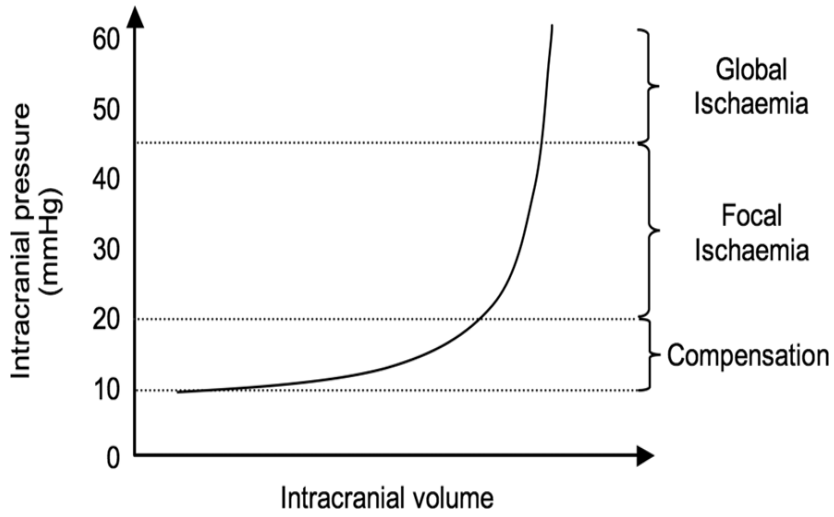


The Monro-Kellie doctrine states that the volume of all three components together within the cranial cavity is constant. Any increase in one of these components should cause a decrease in one or both the remaining components (Wilson, 2016). For a

child under the age of 2, increasing intracranial pressure can increase the head circumference, due to the skull plates not yet being fixed together. Therefore, children under the age 2 can compensate further for increasing intracranial volume in comparison to an older child or adult.

Compensation of intracranial volume can be achieved up to an intracranial pressure of 20mmHg (Czosnyka, 2004). For any pressure above 20mmHg, intracranial hypertension occurs which increases the risk of reduced cerebral perfusion which in turn may lead to focal or/and global ischaemia. **(Figure 2).**

Figure 2: Graph showing the impact on changes in the intracranial space due to a mass/lesion



Time-Out 4

Go to Nationwide Children's and look through Increased Intracranial Pressure. Make note of the variable signs of ICP increase depending on the age of the child: https://www.nationwidechildrens.org/conditions/increased-intracranial-pressure

Mechanisms of Injury

ABI can be classed as either focal or diffuse head injuries, caused by either penetrating (open) injuries or non-penetrating (closed) injuries. Focal ABI occur in specific locations resulting from a direct force, causing compression of brain tissue under the skull, or brain tissue moving in the opposite direction to the impact (acceleration/deceleration injury). Diffuse ABI occur over a widespread area of the brain that can cause axonal injury, ischaemia and oedema caused by factors including:

- Hypoxia – interruption to cerebral blood flow, prolonged seizure activity or reduced oxygenation.
- Infections – examples include meningitis.
- Toxins – examples include paracetamol overdoses.
- Metabolic alterations – examples include diabetic ketoacidosis, kernicterus, or hepatic encephalopathy.

Presentation of a child or young person can depend on the primary mechanism of injury, the location and distribution of the injury, and whether secondary complications occur **(Figure 3**).

Figure 3: Table showing key differences in clinical presentation depending on location within the brain.

Location of Injury	Clinical presentation
Frontal lobe	• Behavioural changes/problems such as agitation, aggression, reduced inhibitions. • Mood changes such as depression. • Loss of strength in muscles. • Aphasia. • Reduced voluntary muscle movements, such as walking or holding a pencil. • Short-term memory loss.
Temporal lobe	• Short-term memory loss. • Hearing loss. • Difficulty with forming words or sentences. • Loss of sense of smell. • Reduced or absent understanding of written or spoken language.
Parietal lobe	• Reduced or absent sensation, sometimes down one half of the body. • Dyscalculia. • Difficulty reading.
Occipital lobe	• Visual impairments. • Difficulty seeing or identifying colours. • Difficulty recognising or interpreting familiar objects.
Cerebellum	• Cerebellar ataxia (jerky or uncoordinated movements). • Imbalance. • Visual problems. • Slurred speech.

Mechanical damage to brain tissue and blood vessels can result from shearing or tearing forces, with children being more vulnerable to shearing and tearing forces

due to limited neck control and larger heads compared to body size. Vulnerabilities then differ across the lifespan as summarised in **Figure 4**

Figure 4: Table showing injury characteristics and vulnerabilities specific to traumatic brain injuries.

Age	**Common type or cause of acquired head Injury for age range**	**Mechanism and/or rationale**
Neonate	• Intracranial Haemorrhage • Head injury through delivery • Subgaleal Haemorrhage • Cephalic Haemorrhage • Abusive Head Trauma (AHT)	• Vaginal deliveries may lead to head compression or trauma from passage through birth canal, or by use of obstetric instruments to assist with the birthing process. • AHT, such as non-accidental injury – shaken baby
Infants	• AHT • Accidental head injury • Skull fractures • Subdural haematoma • Extradural haematoma	• Infants are reliant on care givers (e.g., dropped from carers' arms or fell from furniture). • Inappropriate supervision or childcare practices. • Large head:body ratio • Head is unsupported by weak neck muscles • AHT is the most common cause of traumatic brain injury-related hospitalisation and death in this age group (Burrows et al.,2015)
Toddlers and school age children	• Accidental head injury • Falls • Skull fractures • Cerebral oedema	• Motor ability increasing, but special awareness, or knowledge of consequences can lead to increased risk of injury. • Falls from heights or downstairs for example. Falls from places are the most common cause of head injury in this age range (Pinto et al., 2012). • Pedestrian injury (e.g., car vs pedestrian/cyclist.)
Adolescents	• Sports-related head injuries • Concussion • Falls • Blunt force trauma	• Contact sports involvement often associated with mild TBI (concussion) • Not wearing protective equipment (e.g., helmets).

		• More independence and less adult supervision. • Peer pressure and reduced resistance to peer influence (Albert et al., 2013). • Risk-taking.

The outcome of a primary mechanism of the injury cannot be improved, whether caused by focal or diffuse injuries. Only preventative measures such as treating blood loss are effective to prevent further complications.

ABI can cause cellular, metabolic, and biochemical changes to the brain which can differ depending on the primary or/and secondary insult. This can cause disruption in the integrity of the blood brain barrier (BBB), which is normally a tight junction of cells that prevents anything harmful entering the delicate brain cells. An injury can cause either of the following:

- Trigger an inflammatory response in response to pathogens, tissue injury or allergens. Inflammatory mediators are released by mast cells to cause vasodilation of cerebral capillaries to encourage an innate immune response and clotting cascade to aid tissue repair. If inflammation is prolonged, vascular leakage causes an influx of proteins such as albumin, thrombin and immune cells increases osmotic pressure within the brain, leading to cerebral oedema and thus, increased intracranial pressure. Children are further vulnerable to diffuse cerebral oedema due to having a higher cerebral blood flow in comparison to young people and adults, influenced by having a higher cardiac output (Zimmerman, 2021). But the underlying pathophysiology to why this happens remains unclear.
- The risk of pathogen transmission into the meninges and parenchyma due to inflammation. Infants and young children having an immature immune system are more vulnerable to illnesses such as meningitis and encephalitis due to delayed white blood cell response. Pathogens that cause infections of the brain include Group B Streptococci, E. Coli, Herpes Simplex and mycobacterium tuberculosis.
- Inflammatory mediators that respond to pathogens can increase permeability of capillaries within the meninges which increases the risk of vascular leakage.
- Injury to neurons can cause glutamate, considered the primary excitatory neurotransmitter in the nervous system, to enter extracellular spaces which triggers sodium and calcium ions into neurons (The Royal Children's Hospital Melbourne, n.d). Because of oxidative stress and a large influx of calcium ions resulting from glutamate disruption, mitochondria will fail to produce ATP (energy) which can result in neuron death and necrosis.

Time Out 3 activity

Communication of neurological deterioration

Imagine you are needing to communicate to a family member who is showing anxiety. How would you explain concepts such as cerebral oedema that prevents more intense feeling of anxiety?

Another complication particularly TBI can cause cerebrospinal fluid (CSF) leaks, which is one of the most common complications following a TBI (Oh et al., 2017). CSF forms the blood-CSF barrier (BCSFB) circulation, to remove waste products and metabolites through its continuous renewal. An adult produces approximately 500ml of CSF per day, at a rate of 18-25ml/hr. Infants and young children can produce CSF at a rate of 10-12ml/hr (Thwaites et al, 2009).

A CSF leak occurs when CSF escapes via a hole or tear in the dura mater, the outermost layer of the meninges. The dura may be injured or teared by TBI, or surgical procedures which involve the sinuses, brain or spine. It may also be damaged by clinical procedures, such as lumbar punctures, including spinal taps, spinal anaesthesia and myelograms. In addition, spontaneous CSF leaks may occur due to increased intracranial pressure. For example, patients with hydrocephalus, where an increasing volume of CSF builds up within the skull resulting from either congenital defects such as spina bifida, lesion compression on the CSF pathway or blood clots in the CSF pathway.

ICP is directly affected by changes of CSF within the brain and spinal cord, which may be because of:

- Change in the rate of CSF production
- Obstruction of CSF flow within the ventricular system
- Change in absorption rate of CSF

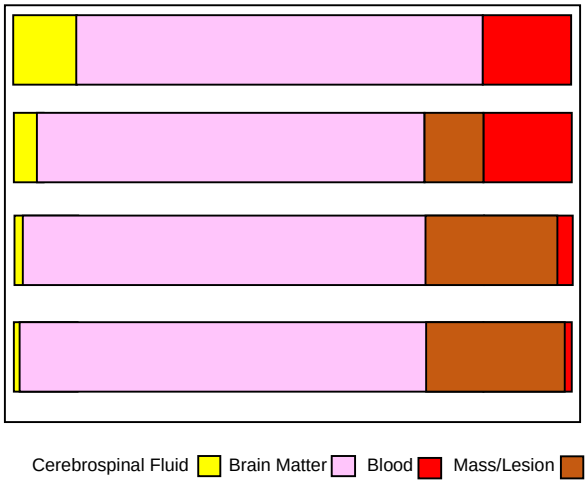
An opening within the subarachnoid space can also lead to life-threatening CNS infections, such as meningitis (Kaufman et al., 1990). When assessing paediatric patients, diagnosis of both CSF leaks and infections can be obscured by other injuries or symptoms, such as for patients with basilar skull fractures or patients of whom, have an extra-ventricular drain (EVD) fitted, which encourages a closed system for CSF drainage for assessment and monitoring purposes. As such, thorough investigation is required which takes in account symptions of CSF leak (Figure 5).

Figure 5 - Table showing typical symptoms of CSF leak

Symptoms of CSF leak		
Loss of sense of smell	**Blurred or double vision**	**Hearing changes or hearing loss**
Seizures	**Neck pain and stiffness**	**Loss of appetite**
Headaches	**Dizziness or vertigo**	**Balance and gait problems**
Light sensitivity	**Nausea and vomiting**	**Hearing pulse in ears**
Leakage from nose	**Leakage from ears**	**Tinnitus**

During expansion of an intracranial mass /lesion, drainage of CSF and cerebral blood are the main methods of compensation to allow space for the new lesion. (Figure 6). CSF can move between the cranium and the spinal subarachnoid space, whilst cerebral autoregulation helps to maintain cerebral blood flow to help maintain cerebral perfusion pressure. When this is exhausted (no more cerebrospinal fluid can be displaced) intracranial pressure (ICP) begins to rise. Increasing intracranial volume can then alter cerebral blood flow, which will consequently reduce cerebral perfusion pressure. This relates to the Monro Kellie Doctrine as one or two components need to reduce to compensate within a limited space.

Figure 6: Diagram showing the relationship between intracranial volumes an intracranial lesion such as a bleed, tumor etc.

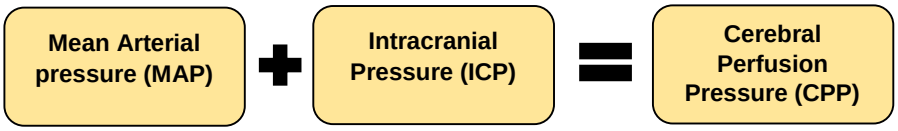


Cerebral Perfusion Pressure

Normally, the brain can regulate blood flow through cerebral blood vessels by changing the cerebral vascular resistance via autoregulation. This requires a stable mean arterial pressure (MAP) and intracranial pressure, which allows cerebral perfusion to be optimised to help with brain function which is influenced by cerebral perfusion pressure.

When intracranial pressure increases because of an injury, or MAP reduces, cerebral blood flow can be altered, reducing cerebral perfusion pressure. This increases the risk of secondary injuries as patients could be vulnerable to cerebral oedema, further neuronal death and brain herniation.

The relationship between mean arterial pressure and intracranial pressure is referred to as cerebral perfusion pressure (CPP). It is calculated using the formula below:



Prevention of secondary insults to the brain is therefore a priority when managing patients with neurological injuries to reduce the risk of interrupted cerebral perfusion caused by increasing intracranial pressure. Thus, proving why an understanding of neurological anatomy and physiology and assessments are vital to improve patient outcomes (National Institute for Health and Care Excellence, 2017).

Cushing's Triad

Early identification using a thorough assessment can lead to swifter medical intervention which is key to reduce any impacts of secondary insult on the brain resulting from reduced cerebral perfusion. Increasing intracranial pressure can cause a medical phenomenon called Cushing's Triad.

Cushing's reflex can also be observed as a warning sign that brain herniation is present. Cushing's reflex is a nervous system response to when MAP becomes lower than intracranial pressure. It has three key signs:

- Hypertension – triggered by baroreceptors in the carotid sinus and aortic arch stimulating a sympathetic nervous response, releasing catecholamines such as adrenaline from the adrenal glands. Peripheral vasoconstriction caused by catecholamine release from the adrenal glands occurs which increases cardiac output in a bid to increase mean arterial pressure. Wide pulse pressures can also be seen when observing a patient's blood pressure due to the increasing cardiac output.
- Bradycardia - this is triggered by a subsequent parasympathetic response from the baroreceptors to compensate for a higher cardiac output.
- Irregular breathing – caused by medulla compression and ischaemia as the medulla houses the central chemoreceptors, resulting in changes in the patient's breathing such as irregular breathing and apnoea. This is often indicative of the start of brain herniation.

Other signs that can occur during Cushing's Triad to indicate elevated intracranial pressure include:

- Nausea and vomiting – can be projectile vomiting.

- Altered mental state – caused by increasing intracranial pressure resulting in brain ischaemia to particular parts of the brain – presentation can vary.
- Reduced arousal – patients may be harder to stimulate and will have slower voluntary responses.
- Unequal pupils (Mydriasis) – caused by disruption to efferent neural pathways or compression to the 3rd cranial nerve. Can also be an initial sign of brain herniation (Adoni and McNeet, 2007).
- Severe headaches – caused by compression of brain matter against the skull and reduction in CSF.

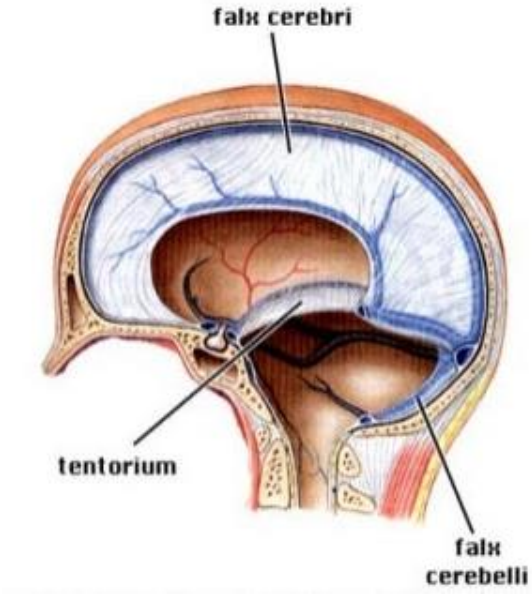
Brain herniation

The brain contains inward dura mater folds that form dural reflections to make up compartments within the cranial cavity. This provides subdivisions of the brain, for example separating the right and left hemisphere of the brain. Knowledge of these will help with explanation on how brain herniation occurs in response to increasing intracranial pressure (Figure 7). These include:

- Falx Cerebri – a large crescent shaped dural fold that separate the right and left cerebral hemispheres.
- Falx Cerebelli – dura mater fold that separates the left and right cerebellar hemispheres.
- Tentorium cerebelli – a dural fold that separates the occipital and temporal lobe away from the cerebellum, as well as providing a passage for the midbrain called the tentorial notch.

Each compartment is designed to provide structural support for the two central hemispheres and the four brain lobes.

Figure 7: Diagram showing some of the dural reflections described earlier.



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Brain herniation is a medical phenomenon that occurs when increasing intracranial pressure causes brain tissue from one intracranial compartment to move into another intracranial compartment (Knight and De Jesus, 2022). It is a sign that compensatory mechanisms stated within the Monro-Kellie Doctrine cannot keep the intracranial pressure stable as the intracranial volume has reached a critical volume.

Brain herniation can occur in different ways. The most common types of herniation are summarised below:

- Uncal herniation – caused by a mass lesion such as an extradural haematoma causing the innermost part of the temporal lobe (the uncus) to be pushed towards the tentorium. This exerts pressure on the midbrain (the top part of the brainstem), which compresses on the 3rd cranial nerve causing pupil dilation in the eye where the compressed 3rd cranial nerve is.
- Central (transtentorial) herniation – this is when central structures such as the diencephalon (thalamus, hypothalamus and limbic system) and parts of the temporal lobe are pushed through the tentorium putting pressure on the cerebellum. It can also cause pontine haemorrphage which can be identified by pinpoint and unreactive pupils.
- Subfalcine herniation – this happens when a curved fold called the cingulate cortex gets pushed under the falx cerebri. The cingulate cortex covers the corpus callosum of the limbic system which plays a vital role in emotion formation and processing, learning and memory formation.
- Tonsillar herniation – this is when the cerebellar tonsils is push down into the foramen magnum, an opening in the occipital bone of the skull where the

medulla oblongata connects to the spinal cord. This causes ischaemia of the brain stem which causes respiratory and cardiovascular dysfunction. We often refer to this as 'coning.'
- External (transcalvarial) herniation – when brain tissue is forced through a fracture line in the skull. This can be done as a preventive measure to relief intracranial pressure in a procedure called a craniectomy.

Critical signs of brain herniation include:

- Decorticate or decerebrate posturing – Transmission through the rubrospinal and corticospinal tract is interrupted due to increased pressure within the skull, particularly the mid-brain as a result of central herniation. Decorticate posturing is indicative of a central herniation, whilst decerebrate herniation can indicate the start of tonsillar herniation (Munakomi and Das, 2021).
- Bilateral dilated pupils (Anoxia mydriasis) – caused by transtentorial herniation or anoxia leading to global iscaemia (Adoni and McNeet, 2007).
- Reduction or loss in brainstem reflexes – this includes blinking and the gag reflex.
- Loss of consciousness/comatose.
- Respiratory and/or cardiac arrest – resulting from ischaemia to the brain stem causing respiratory and cardiovascular autonomic regulation to reduce.

Time Out 5

Assessing a child or young person deteriorates neurologically.

Before Part 2, think about key assessments that are necessary to aid a thorough neurological assessment on a child or young person who presented with neurological deterioration.

Conclusion

The importance of understanding the pathophysiology of ABI is fundamental to improving early recognition of neurological deterioration in children and young people. The need to identify signs of neurological deterioration to prevent secondary insults to the brain is significant to increasing recovery chances for children and young people. Understanding the underlying pathophysiology will also benefit Part 2 of the Neurological Observations in Infants, Children and Young People series, where we will explore how to perform neurological observations to promote evidence-based practice, consistency, and further underlying physiology to why we conduct these neurological observations, relating back to topics covered in this part.

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Revisions made for Part 1

- Changes to learning outcomes.
- Change to Time-out activity 1.
- Change in citation regarding definition of acquired head injuries.
- Change in statement regarding the need to understand a patient's underlying health condition.
- Section about the brain removed to encourage more focus on pathophysiology of acquired head injuries and reducing word count.
- Diagram of dural folds moved to brain herniation section
- Diagram of brain lobes removed and replaced with a table (figure 3).
- Diagram and section about brain hemispheres removed.
- Removal of table discussing primary head injuries to reduce word count and content already being briefly covered in previous table.
- Figure 4 showing injury characteristics and vulnerabilities specific to traumatic brain injuries moved from Acquired brain injury in children and young people section into Mechanisms of Injury section as felt this would be more suitable.
- Section added within mechanisms of injury section that discusses cerebral oedema, infection risk and glutamate – replacing previous section about pathophysiology of head injuries
- Expansion on cerebral oedema included.
- Expansion on risks regarding meningitis and why infants and children are more vulnerable added.
- Section about CSF leak include to accompany mechanism of injury section – particularly in relation to TBI.
- Intracranial pressure section added to before mechanisms of injury section and content moved into the section to provide context of intracranial pressure.
- Figure 5 from previous article split up into different sections.
- Additional information added to the brain herniation section.