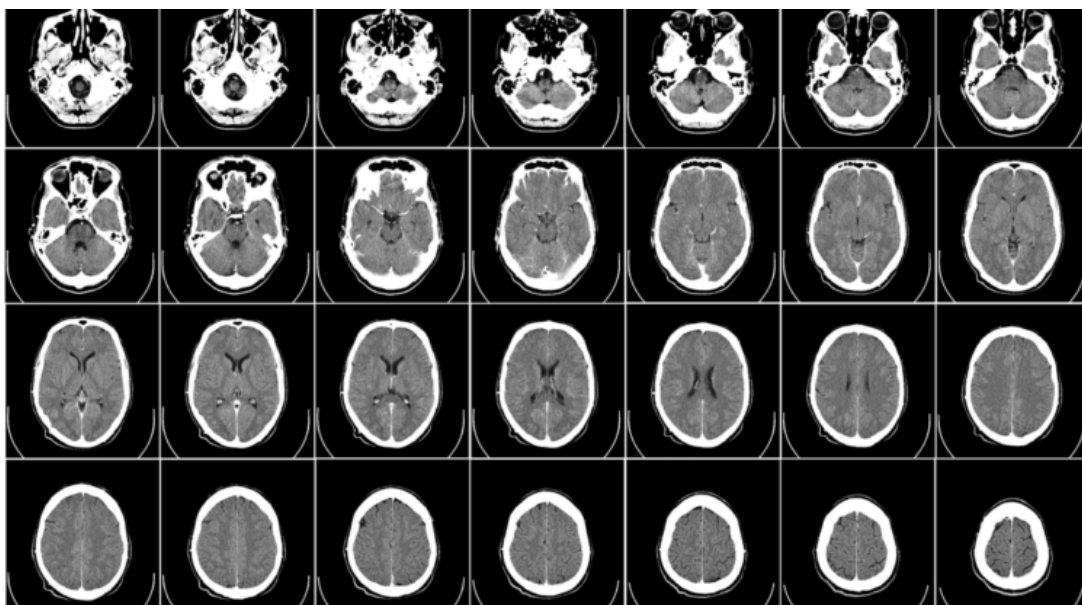


Increased Intracranial Pressure (ICP) in Children — Symptoms and Treatment

[See online here](#)

This article provides a clinical run-through of increased intracranial pressure for the USMLE Step 2 examination. It defines raised ICP, runs through its pathophysiology, progression and special forms, diagnosis, differential diagnosis and treatment (management including patient monitoring).



Definition of Increased Intracranial Pressure

Intracranial pressure is **usually less than or equal to 15 mmHg** in the adult patient, and **slightly lower in children**. A raised intracranial pressure is defined as one above 20 mmHg.

Constant monitoring is needed for a true representation of ICP and **intraventricular monitoring** is considered the gold standard. However, raised ICP can be diagnosed via a thorough history and examination and often therapy will have to be started acutely before monitoring can be initiated and, as such, knowing how to identify raised ICP in clinical practice is incredibly important.

Pathophysiology of Increased Intracranial Pressure

Monroe doctrine says that because the brain is a defined “box” or incompressible space, the volume inside the cranium is fixed.

There are a number of constituents to the inside of the cranium (blood, [CSE](#), brain tissue) and an increase in any one volume will decrease the volume of another.

The brain tissue (or **parenchyma**) usually makes up around **80% of the fixed internal volume** (which itself is usually between 1,400 and 1,700 mL). The **CSF** makes up around **10%** of total volume (around 150 mL) and **blood** the final **10%**. As discussed previously, when any of these is increased, either the others must decrease or the intracranial pressure must increase (or both processes occur simultaneously).

Intracranial pressure is raised by a number of pathological processes, including **masses** (like tumors) **abscesses** and **blood** (hematomas). **Impaired CSF flow** can also cause a rise in intracranial pressure. This is because CSF is produced in the **choroid plexus** (amongst other areas) and is reabsorbed by **arachnoid granulations** back into the venous system. **Blockages** can stop the reabsorption of CSF increasing its volume and raising intracranial pressure.

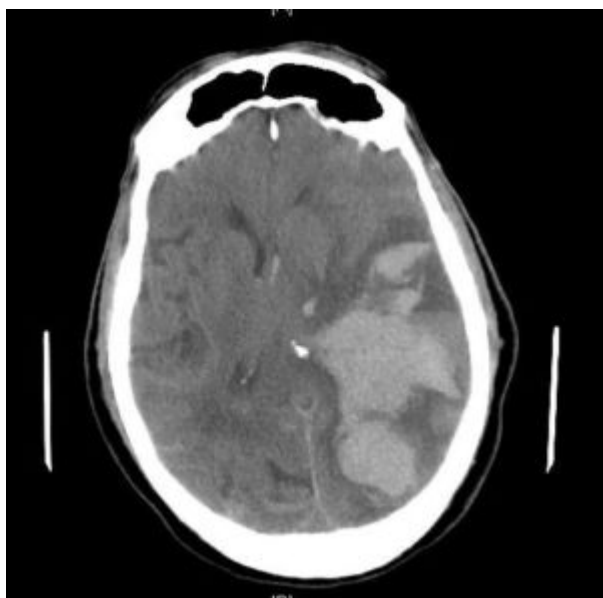


Image: "CT scan of Subfalcine Herniation." by RadsWiki. License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

Herniation can occur when **focal or global pressure** force portions of the brain into the wrong area. For instance:

- Subfalcine herniation
- Uncal herniation
- Tonsillar herniation

Progression and Special Forms

When the intracranial pressure increases beyond a certain point, herniation can occur. This can be:

- **Subfalcine (cingulate gyrus) herniation** where the cingulate gyrus is displaced underneath the falx to the opposite side. This can cause compression of branches of the anterior cerebral artery.
- **Transtentorial (uncal, mesial temporal) herniation** where the medial aspect of the temporal lobe is compressed against the tentorium cerebelli.
- **Tonsillar herniation** where there is displacement of the cerebellar tonsils through the foramen magnum. This can cause compression of medullary respiratory centers.

Diagnosis of Increased Intracranial Pressure

Clinical manifestations of increased ICP include:

- Headache (especially a constant ache which intensifies upon awakening)
- Nausea
- Vomiting
- Drowsiness
- Double vision or blurry vision

A patient with raised intracranial pressure will usually have a **headache**. The most widely accepted theory is that this is the result of **cranial nerve V pain fibers** running along the dura and blood vessel. Patients may also have **reduced consciousness**; this can be from local lesions or from pressure pushing on the reticular formation (which is a structure in the **brainstem** involved in consciousness). Patients may also experience **vomiting**.



Image: "Papilledema is a swollen optic disc caused by increased intracranial pressure." by Jonathan Trobe, M.D. - University of Michigan Kellogg Eye Center - The Eyes Have It. License: [CC BY 3.0](https://creativecommons.org/licenses/by/3.0/)

On examination, **funduscopy** should be performed. Often raised ICP can be visualized as **papilledema**. A **full cranial nerve examination** should be conducted as cranial nerve VI palsies are common with raised ICP. (Cranial nerve VI, the abducens nerve, controls the lateral rectus muscle of the eye.

Patients will present with eye resting in the adducted position. They may complain of double vision or "diplopia"). If patients exhibit **Cushing's triad** (bradycardia, respiratory depression and **hypertension**), then immediate intervention is needed.

Other symptoms and examination findings are localized and can be different dependent on the localization of the lesion. Herniation syndromes can also result in **neurological deficits**.

The only truly reliable diagnosis is **investigation via IVP monitoring**, although most diagnoses will rest on clinical findings and a good history.

Differential diagnosis

Below is a summary of the major reasons for an increase in intracranial pressure. Usually, the differential for those with raised ICP will be much smaller than this list as patients don't usually present with "raised intracranial pressure" and diagnosis is more obvious given the total clinical picture. However, this is an important list to keep in mind if the immediate cause is unknown.



Image: "CT scan of a brain with hydrocephalus." by Lucien Monfils - Own work. License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

- Space occupying lesions, i.e. a tumor or a hematoma
- Edema
- Increased CSF production
- Decreased CSF absorption
- Obstructive [hydrocephalus](#)
- Obstruction of venous outflow
- Idiopathic causes

Space occupying lesions are self-explanatory in that they are tumors or hematomas.

Cerebral edema can be caused by a number of things, including [traumatic brain injury](#), an **infarction** and **ischaemic encephalopathy**.

A **choroid plexus papilloma** can cause an increased CSF production, whilst decreased production can be the result of **adhesions on the arachnoid granulations** (typically the result of [meningitis](#)). An obstruction in the venous outflow can be caused by **venous sinus thrombosis** or **jugular vein compression**.

Management of Increased Intracranial Pressure

Although early interventions will often be performed without direct monitoring of raised ICP, therapies are reversible and therefore the first line of management should be to place an **ICP monitoring device**. The main goal of treatment is to maintain ICP below 20 mmHg. CT scans can show a raised ICP (i.e. with the presence of a tumor or a hemorrhage); however, they are unreliable and other methods to define ICP should be performed.

Intraventricular monitors are considered the gold standard. **Infection risk** is very high with this equipment and occurs in some 20% of patients. There is also a small, but substantial **risk, of hemorrhage** (at around 2%) – this risk is greater in those with **coagulopathies**.

Other methods of measuring ICP include **intraparenchymal devices** (that are inserted directly into brain parenchyma via a small borehole in the skull), **subarachnoid bolts** or **epidural monitoring devices**.

Many simple bedside practices can make a massive difference in decreasing ICP. **Raising the head of the bed by 45 degrees** or **not putting circumferential tape around the neck** for securing ETT (jugular venous pressure).

Patients should be **hyperventilated** with around **25-30 mmHg of PCO₂**, although this is not absolute. Hyperventilation works because CO₂ induces vasoconstriction and can decrease the volume of intracranial blood (reducing one of the three components determining intracranial pressure). It is useful in raised ICP as a result of **cerebral edema**, **intracranial hemorrhage** or **tumor**.

However, its use is not indicated in those with traumatic brain injury or acute stroke. This is because it reduces cerebral perfusion and can worsen neurological outcomes. As such, the **underlying cause of raised ICP should be discussed before hyperventilation**.

Patients can be given **antiepileptic therapies**. This is because patients are at an increased risk of developing seizures with a raised ICP and these can complicate the clinical picture. As such, antiepileptics should be prescribed to any patient where a seizure has occurred or has been suspected of occurring. **Prophylactic treatment** can also be given, but there are currently no clear guidelines as to when and where to give this.

In many cases where the underlying cause is not known or is untreatable, **diuretics** can be given to reduce blood volume and thus ICP. The first line diuretic is usually **Mannitol** at 2 g/kg (although some evidence suggests it should be given at 0.5 g/kg).

Hypertonic saline can be given to acutely lower the ICP. The volume of saline is governed by no guideline and will be different between hospitals. Some studies have suggested that hypertonic saline bolus is more efficacious than Mannitol.

In patients with a **CNS neoplasia**, IV steroids (**glucocorticoids**) are indicated and have a role in management. Glucocorticoid therapy should be avoided in cases of increased ICP due to head trauma, hemorrhagic ICP, and ischemic stroke.

In some instances, **drainage of CSF can be performed** to relieve ICP. This is indicated in patients with **hydrocephalus**. CSF can be drained at a rate of 1-2 mL per minute for 2 minutes each procedure.

Patients may also undergo **decompressive craniotomy**, whereby the finite volume that can be housed in the skull is increased; therefore, reducing pressure on brain parenchyma, etc. However, this surgery is not without complications and herniation can occur; infection or hemorrhage may worsen the clinical picture.

An increase in intrathoracic pressure and obstruction of cerebral venous outflow due to muscle activity may facilitate a rise in ICP. In these cases, sedation may be required. Sedation is most commonly achieved with morphine, propofol or midazolam. If ineffective, a neuromuscular blockade should be considered. This, however, is associated with an increased risk of complications such as pneumonia and sepsis.

References

Fishman R. Cerebrospinal fluid in diseases of the nervous system, WB Saunders, Philadelphia 1980.

Mokri B (June 2001). "The Monroe-Kellie hypothesis: applications in CSF volume depletion". *Neurology*. 56 (12): 1746–8.

Tuite, G., Chong, W., Evanson, J., Narita, A., Taylor, D., Harkness, W., Jones, B., Hayward, R. and Eppley, B. (1997). The Effectiveness of Papilledema as an Indicator of Raised Intracranial Pressure in Children with Craniosynostosis. *Journal of Craniofacial Surgery*, 8(1), p.81.

[Evaluation and management of elevated intracranial pressure in adults](#) via uptodate.com

Legal Note: Unless otherwise stated, all rights reserved by Lecturio GmbH. For further legal regulations see our [legal information page](#).

Notes