

Raised intracranial pressure

What is raised intracranial pressure?

Raised intracranial pressure (ICP) can arise as a consequence of intracranial mass lesions, disorders of cerebrospinal fluid (CSF) circulation and more diffuse intracranial pathological processes. Its development may be acute or chronic.

What causes raised intracranial pressure? (Aetiology) ^[1]

- Localised mass lesions: traumatic haematomas ([extradural](#), [subdural](#), intracerebral).
- Neoplasms: [glioma](#), [meningioma](#), metastasis.
- Abscess.
- Focal oedema secondary to trauma, infarction, tumour.
- Disturbance of CSF circulation: obstructive [hydrocephalus](#), communicating hydrocephalus.
- Obstruction to major venous sinuses: depressed fractures overlying major venous sinuses, [cerebral venous thrombosis](#).
- Diffuse brain oedema or swelling: encephalitis, meningitis, diffuse head injury, [subarachnoid haemorrhage](#), [Reye's syndrome](#), lead encephalopathy, water intoxication, near drowning.
- Idiopathic intracranial hypertension ^[2] .

Raised intracranial pressure symptoms

The combination of headache, [papilloedema](#) and vomiting is generally considered indicative of raised intracranial pressure, although there is no consistent relation between the severity of symptoms and the degree of hypertension. A 2021 study exploring the diagnostic accuracy of clinical signs as indicators of a raised ICP found that relying on them alone may lead to undertreatment ^[3] .

- Headache: more worrying when nocturnal, starting when waking, worse on coughing or moving head and associated with altered mental state ^[4] .
- Early changes in mental state include lethargy, irritability, slow decision making and abnormal social behaviour. Untreated, this can deteriorate to stupor, coma and death.
- Vomiting (in early stages without nausea), which can progress to projectile with rising intracranial pressure.
- Pupillary changes, including irregularity or dilatation in one eye.
- Fundoscopy shows blurring of the disc margins, loss of venous pulsations, disc hyperaemia and flame-shaped haemorrhages. In later stages, obscured disc margins and retinal haemorrhages may be seen.
- Unilateral ptosis or third and sixth nerve palsies. In later stages, ophthalmoplegia and loss of vestibulo-ocular reflexes.
- Late signs include motor changes (hemiparesis), raised blood pressure, widened pulse pressure and slow irregular pulse.

- Acute situations:
 - Head injury and obtundation: bleeding can form a rapidly expanding haematoma leading to rapidly rising ICP if not treated promptly.
 - **Syncope**, headache and meningismus: abrupt onset of headache with these symptoms suggests ruptured cerebral aneurysm or vascular lesion.
 - Focal deficit followed by **seizures**: focal deficit can be associated with a mass lesion and when there is oedema or haemorrhage. Intracranial compartment shift can cause increased ICP within minutes or hours; status epilepticus can cause decompensation of cerebral volume regulation.
 - 'Talk and deteriorate': patients typically talk recognisably following head injury, then go into coma in the first two days. The usual cause is an intracranial haematoma.

Investigations

- CT/MRI scanning to determine any underlying lesion.
- Check and monitor blood glucose, renal function, electrolytes and osmolality.

Monitoring intracranial pressure

- Intracranial pressure monitoring is:
 - Used either as a guide to treatment or as a diagnostic test. The most common use of continuous ICP monitoring is in the management of severe closed head injury.
 - Appropriate in patients with severe head injury ([Glasgow Coma Score](#) between 3 and 8 after cardiopulmonary resuscitation) and an abnormal CT scan (haematomas, contusions, oedema or compressed basal cisterns).
 - Appropriate in patients with severe head injury and a normal CT scan if two or more of the following features are noted on admission: age over 40 years, unilateral or bilateral motor posturing, systolic blood pressure <90 mm Hg.
 - Not routinely indicated in patients with mild or moderate head injury; however, a clinician may choose to monitor ICP in certain conscious patients with traumatic mass lesions.

- Other conditions in which intracranial pressure monitoring is used include:
 - **Subarachnoid haemorrhage** with associated hydrocephalus: ventriculostomy allows therapeutic drainage and ICP monitoring.
 - **Reye's syndrome**: active treatment of raised ICP decreases mortality.
 - Brain tumours: may be of value in selected patients deemed at high risk of swelling or obstructive hydrocephalus – eg, following posterior fossa surgery.
 - Normal pressure hydrocephalus.
 - Decompensated hydrocephalus: can be a valuable diagnostic tool in complex cases.
 - Idiopathic intracranial hypertension: as both a diagnostic test and to monitor response to treatment.
 - Other potential indications include hypoxic brain swelling after drowning, meningitis, encephalitis, venous sinus thrombosis and hepatic encephalopathy.

Raised intracranial pressure treatment and management^[1] ^[5]

In the acute emergency situation the priority is maintaining adequate arterial oxygen tension and ensuring normal vascular volume and normal osmosis. It is also essential to maintain normoglycaemia.

Otherwise, treatment will depend on the underlying pathology.

First-line therapies

- **Avoid pyrexia**: this increases intracranial pressure and is an independent predictor of poor outcome after severe head injury.
- **Manage seizures**: they contribute to raised intracranial pressure and should be managed aggressively using standard anticonvulsant loading regimens.

- **CSF drainage:** when an intraventricular catheter is used to monitor intracranial pressure, CSF drainage is an effective method for lowering ICP. This can be accomplished by intermittent drainage for short periods in response to elevations in ICP. The principal risks of ventriculostomy are infection and haemorrhage.
- **Head of bed elevation:** elevating the head of the bed to 30° improves jugular venous outflow and lowers ICP. In patients who are hypovolaemic, this may be associated with a fall in blood pressure and an overall fall in cerebral perfusion pressure.
- **Analgesia and sedation:** usually with intravenous propofol, etomidate or midazolam for sedation and morphine or alfentanil for analgesia and antitussive effect.
- **Neuromuscular blockade:** muscle activity may further raise intracranial pressure by increasing intrathoracic pressure and obstructing cerebral venous outflow. If this does not respond to analgesia and sedation then neuromuscular blockade is considered.
- **Mannitol (an intravascular osmotic agent):**
 - The major problems associated with mannitol are hypovolaemia and the induction of a hyperosmotic state. Hypertonic saline may be superior in some situations^[6].
 - Serum osmolality should not be allowed to rise over 320 mOsm/kg.
 - Mannitol therapy for raised intracranial pressure may have a beneficial effect on mortality when compared with pentobarbital treatment but may have a detrimental effect on mortality when compared with hypertonic saline.
 - There are insufficient data on the effectiveness of pre-hospital administration of mannitol.
 - Hypertonic saline 3–30% has been shown to be effective in patients with raised intracranial pressure following a stroke when mannitol has been ineffective.

- **Hyperventilation:** this lowers ICP by inducing hypocapnoeic vasoconstriction and has been shown to be effective in reducing raised ICP. However, hyperventilation also induces or exacerbates cerebral ischaemia in a proportion of patients. Prophylactic ventilation with hyperventilation for patients with head injury has not been shown to produce any benefit one year after injury.

Second-line therapies

- **Barbiturate coma:** barbiturates in high doses are effective in lowering refractory intracranial hypertension but ineffective or potentially harmful as a first-line or prophylactic treatment in patients with head injuries. The use of barbiturates in the treatment of refractory intracranial hypertension requires intensive monitoring and is associated with a significant risk of complications, especially hypotension. Withdrawal of treatment should be gradual to avoid rebound intracranial hypertension.
- **Optimised hyperventilation:** this involves the use of more aggressive hyperventilation, with measurement of jugular venous saturation in an attempt to prevent hyperventilation-induced ischaemia. The main problem with this approach is that focal areas of cerebral ischaemia may be produced even though global measures suggest adequate oxygen supply.
- **Hypothermia:** cooling to 35°C (rather than 33°C) is effective in lowering refractory intracranial hypertension and has fewer systemic complications in, for example, the pulmonary system, infections, coagulation and electrolytes. There appears to be a significant rebound in intracranial pressure when induced hypothermia is reversed.
- **Decompressive craniectomy:** this technique has been reported as being beneficial in a number of disorders, including head injury, cerebral infarction, spontaneous intracerebral and subarachnoid haemorrhage and Reye's syndrome. There is no evidence to support the routine use of secondary decompression craniectomy to reduce unfavourable outcome in adults with severe traumatic brain injury and refractory high ICP. However, decompression craniectomy may be a useful option when maximal medical treatment has failed to control ICP.

Further reading

- [Rufai SR, Jeelani NUO, McLean RJ](#); Early Recognition of Raised Intracranial Pressure in Craniosynostosis Using Optical Coherence Tomography. J Craniofac Surg. 2021 Jan–Feb 01;32(1):201–205. doi: 10.1097/SCS.0000000000006771.
- [Wakerley BR, Mollan SP, Sinclair AJ](#); Idiopathic intracranial hypertension: Update on diagnosis and management. Clin Med (Lond). 2020 Jul;20(4):384–388. doi: 10.7861/clinmed.2020-0232.

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Authored by:	Peer Reviewed by: Dr Colin Tidy, MRCP	
Originally Published: 20/11/2023	Next review date: 01/12/2021	Document ID: doc_1680

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