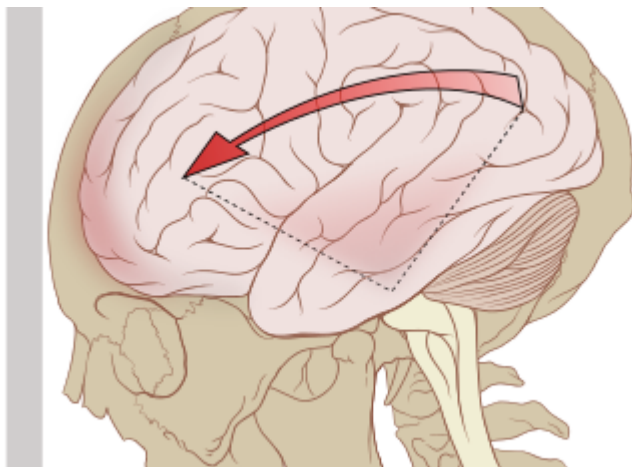


Traumatic Brain Injury: Management of Increased Intracranial Pressure (ICP)

[See online here](#)

Traumatic injuries due to traffic accidents, sports or violence induced incidents, represent one of the major causes of morbidity and mortality nowadays. Significant portion of all trauma cases include head trauma, which can be divided into fractures, brain traumatic injuries, hematomas (both extra axial and parenchymal) and diffuse axonal injuries (DAI). Recognition of the signs and symptoms is of uttermost importance, for head trauma can be as mild as every day bruises and bumps to the head, or severe and life threatening. Rapid recognition, dedicated diagnosis and adequate imaging which lead to proper treatment - both conservative and surgical - are essential for the best possible, complication- and sequel-free, outcome.



Definition of Head Trauma

Head trauma includes a wide spectrum of injuries to the head, including bones, brain, meninges and intracranial vascular structures. But mostly, the term head trauma is a synonym for **traumatic brain injury** (TBI).

Increased intracranial pressure is every rise above the threshold of **20 mm Hg** independently of the reason.

Classification of Head Trauma

Head trauma can be divided:

By the clinical presentation:

- Mild: Loss of conscience shorter than 30 minutes, normal CT and/or MRI scan.

Patient may have cognitive problems like headache, memory problems, attention and mood changes.

- **Severe:** Loss of consciousness longer than 30 minutes, memory loss longer than 24 hours. Neurological deficit, that may be nonreversible, is presented as impairment of movement, higher level cognitive functions, with potential progression to a comatose state.

By type of injury:

- **Open:** Injury that involves fracture of head bones, brain tissue is directly injured by fragments of bone, bullets, knife, etc.
- **Closed:** Intact head bones and dura mater, brain is damaged due to acceleration-deceleration, can be both focal and diffuse.

By the time of presentation:

Primary: Occurs at the moment of injury, and could be of pure mechanical nature (object passing through brain tissue and meninges and causing direct trauma), and brain hitting the bone on/opposite to the impact site such as coup and counter-coup lesion; or acceleration-deceleration injury when unrestricted movement of head leads to strains and causes hematomas, vascular or stalk injury.

Secondary: Injuries that occur hours/days/weeks after trauma, and are not a consequence of direct trauma, but are considered to be consequence of impaired cerebral blood flow (CBF), resulting in local or diffuse brain edema, hemorrhage or increased intracranial pressure(ICP) complicated by electrolyte disbalance potentially resulting in neuronal and/or patient's death.

Recent Updates about Minor Head Injuries

The term mild traumatic brain injury was considered as too vague by the most recent **NICE** guidelines. Accordingly, a new term “**minor head injury**” was presented. Patients with a minor head injury can be classified into those who have lost consciousness for a period that is less than 10 minutes and those who have never lost consciousness after sustaining a blunt head trauma.

Patients who did not lose consciousness after head trauma and have a **Glasgow Coma Score** of 15 out of 15 can be safely discharged home with instructions about the **signs of neurological deterioration**. The signs of neurological deterioration that the patient should be instructed about are:

- Severe headaches that are refractory to acetaminophen
- Repeated vomiting
- Development of seizures
- Any alteration in the level of consciousness

Patients with a minor head injury who have lost consciousness for a period that is less than 10 minutes should undergo a thorough physical examination focusing on excluding the possibility of **significant cerebral injury**. The signs of significant cerebral injury in a patient with a minor head trauma include:

- Severe headaches, confusion, or seizures
- Clinical signs of a basal skull fracture
- A Glasgow Coma Score below 15 at 2 hours post injury
- Focal neurological deficits

- Vomiting for more than 3 times at the emergency department
- History of coagulopathy, bleeding disorders, or use of anti-coagulation therapy
- Age above 65 years

Moreover, if the patient returned to the emergency department after being discharged home with the diagnosis of a minor head injury for any reason, the treating physician should start the process of excluding a significant cerebral injury. A **computed tomography scan** is the gold-standard in excluding a significant cerebral injury in these scenarios.

Pathophysiology of Head Trauma

Fractured bones (linear, displaced, impacted) and lesion of underlying dura or arachnoid membrane resulting in epidural or subdural hematomas; brain injuries which consist of intraparenchymal hematomas, contusion of underlying brain tissue and diffuse axonal injuries.

Epidural hematoma is, potentially rapidly expanding blood collection between the bone and dura mater originating from meningeal arteries. It is a life-threatening condition that usually requires a surgical procedure.

Subdural hematoma is blood collection between the dura and the arachnoid membrane that originates from injured bridging veins. It can be both acute and chronic, and requires prompt, usually surgical care.

Intraparenchymal contusion (areas of swollen and damaged brain tissue) and hematomas can occur on the contact site (coup lesions), as well as on contralateral site (known as countercoup lesion).

Diffuse axonal injury (DAI) is one of the most important pathologic features of brain trauma. Microscopic damage of the brain tissue, resulting from the rotational acceleration of the brain and subsequent unrestricted head movement, is complicated to recognize both on clinical exam and on imaging studies (MRI is gold standard for recognition of DAI).

Pathophysiological exam shows **multiple swollen and disconnected axons**, leading to changes in normal neuron function. Mainly decline in cerebral blood flow, both local and global, is the reason for this. It results in **edema, hemorrhage** and **increased intracranial pressure**. **Impaired cerebral blood flow** and raised intracranial pressure lead the cascade of **electrolyte disorders** eventually resulting in **neuronal death**.

Following **Monro-Kellie doctrine** it's known that the adult skull, because the sutures and fontanels are closed, is a rigid nonexpendable vault. Its volume remains constant regardless of the proportion of individual constituents, presented as cerebral parenchyma (80 %), cerebrospinal fluid (CSF) (10 %) and blood (10 %). Also, the rise of volume in one compartment raises intracranial pressure.

ICP is the pressure inside the cranial vault, with a physiological range between 10-20 mmHg in adults and 3-7 mmHg in children.

ICP depends on the brain, CSF and cerebral blood interaction. It depends gravely on CSF production volume, system resistance to reabsorption of CSF and venous pressure in superior sagittal sinus.

Raised ICP can lead to intracranial herniation

- Transtentorial (either lateral or central)
- Tonsillar
- Subfalcine

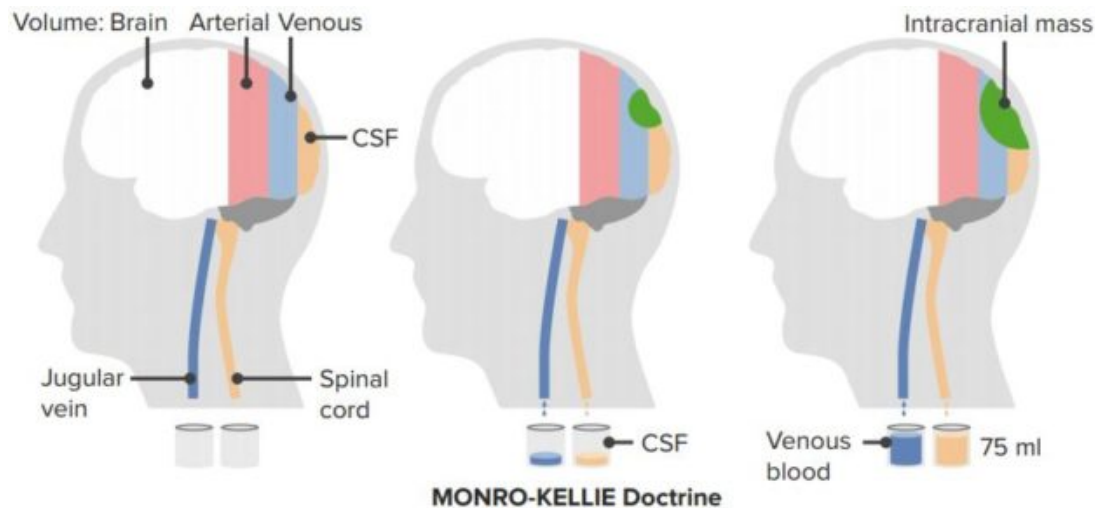
Physiologically, **pressure variations** are rapidly compensated by **CSF displacement** into the subarachnoid space, spinal canal and lumbar cistern that leads to cerebral blood flow (CBF) decrease.

ICP is regulated in few stages:

- Firstly, the increased intracranial volume doesn't affect ICP because CSF is displaced in the ventricular system and spinal canal, thus compensating volume change.
- Next level is volume overload when the system becomes decompensated and finally ceases to function which leads to prompt and rapid changes in ICP, even after minor volume increase.
- In long-standing, high-pressure situations brain parenchyma undergoes reductive changes and neurons and glial cells are lost.

So when **buffer mechanisms** fail, increased intracranial pressure (ICP) leads to a **decrease in blood supply** and **decreased cerebral perfusion pressure (CPP)**.

CPP is calculated as a result of subtraction of mean arterial pressure (MAP) and intracranial pressure (ICP) values.



"Why does ICP occur. MONRO-KELLIE Doctrine" Image created by Lecturio

Cerebral blood flow (CBF) is represented by 15 % to 25 % of the cardiac output, and approximately 40 to 50 mL/100g of brain tissue/min. But blood flow volume isn't the only thing that regulates oxygen supply to the brain. Oxygen supply is inadequate in cases of underlying anemia and hypoxia.

Cerebral autoregulation

Vasodilatation or **vasoconstriction** are mechanisms that ease the cerebral autoregulation function in order to maintain CBF at an appropriate level and fulfill the brain's metabolic oxygen requirements.

Cerebral autoregulation is a multifactorial issue in first line sensitive to MAP and partial pressure of arterial carbon dioxide (PaCO₂), and to a lesser extent to partial pressure of arterial oxygen (PaO₂) and other parameters. Nevertheless, once the upper or lower limits of autoregulatory mechanisms are exceeded, CBF becomes solely dependent on MAP, which makes **arterial pressure monitoring and therapy** essential in head trauma patients.

Clinical Features of Head Trauma

Knowing that head trauma may present itself in a very wide spectrum of clinical scenarios, and always having in mind that minor injury appearing patients may be the ones with rapidly fatal outcome, one must very seriously and carefully evaluate every trauma patient.

Signs and symptoms are nonspecific; primary estimation is roughly based on **Glasgow Coma Score** that constitutes of measured motor response, verbal response and eye-opening response with the following values:

Motor response

- 6 - Obeys commands fully
- 5 - Localizes to noxious stimuli
- 4 - Withdraws from noxious stimuli
- 3 - Abnormal flexion, i.e., decorticate posturing
- 2 - Extensor response, i.e., decerebrate posturing
- 1 - No response

Verbal response

- 5 - Alert and Oriented
- 4 - Confused, yet coherent, speech
- 3 - Inappropriate words and jumbled phrases consisting of words
- 2 - Incomprehensible sounds
- 1 - No sounds

Eye opening

- 4 - Spontaneous eye opening
- 3 - Eyes open to speech
- 2 - Eyes open to pain
- 1 - No eye opening

The final score is made by adding the values of 1, 2 and 3, and the sum has the following meaning:

Mild (13-15):

- Reversible symptoms, loss of consciousness shorter than 30 minutes.

Moderate disability (9-12):

- Loss of consciousness greater than 30 minutes;
- Physical or cognitive impairments which may or may not resolve;
- Benefit from rehabilitation.

Severe disability (3-8):

- Coma: unconscious state, without meaningful response and voluntary activities.

Persistent vegetative state:

- Vegetative state lasting longer than one month.

Brain death:

- No brain function;
- Specific criteria needed for making this diagnosis (neurological, biochemical, CT or MRI scan).

Mild traumatic brain injury (GCS 13-15)

Also known as **concussion**, this is the most frequent form of TBI and often missed at initial examination. It constitutes of brief change in mental status namely confusion, disorientation, short loss of memory and/or loss of consciousness for less than 30 minutes.

There are some common, but unspecific symptoms such as headaches, fatigue, poor attention/concentration, irritability-emotional disturbances, and also nausea, loss of smell, mood changes, getting confused.

The potential problem makes the fact that symptoms may not, and often are not, present right after injury, taking days and weeks to develop. In older individuals, slow-growing subdural hematoma with confusion, memory loss and subtle balance disorders is the most overlooked trauma sequel that can lead to a range of serious outcomes, fatal end included.

Severe traumatic brain injury (GCS below 13)

This is a serious medical condition presenting as a spectrum of **cognitive function disorders** such as longstanding or irreversible loss of memory, loss of consciousness for more than 30 minutes; **motor function disorders** such as extremity weakness, impaired coordination and balance; **sensation disorders** namely hearing, vision, impaired perception and touch; and very often **emotion alterations** like depression, anxiety, aggression, loss of impulse control, personality changes.

Clinical staging and diagnosis consist of detailed neurological examination and use of **various trauma score charts** (GSC is a common option, but there are many other systems); **brain imaging is indispensable**, routinely in first line CT scan.

- **CT scan** can visualize bone fractures, epidural or subdural hematomas, intraparenchymal hematomas or contusion of brain parenchyma, vascular structure lesions.
- **MRI** examinations are reserved for specific situations, primarily to assess DAI. In rare occasions, SPECT and PET scan may be useful.

In a non-acute setting, cognitive evaluation done by a neuropsychologist is essential, and depending on the patients' needs, physical, occupational and speech therapy should address specific deficits.

Differential Diagnosis

Due to diverse signs and symptoms, and in scenarios of inadequate anamnesis, present TBI must be differentiated from the following:

- Acute stroke
- Acute and subacute subdural hematoma
- Brain metastasis
- Cerebral aneurysm with/without rupture
- Subarachnoid hemorrhage
- Epileptic tonic-clonic seizures
- Decompensated hydrocephalus
- Epileptiform encephalopathies.

Management of Head Trauma and Increased Intracranial Pressure

Management of head trauma requires a **multidisciplinary team**. Depending on specific conditions and the severity of injury, outdoor clinic to ICU patient treatment may be necessary.

Hematomas, epidural and subdural, require **surgical decompression** depending on the neurological status and elevation of ICP.

Contusion and DAI injuries usually require **aggressive conservative treatment** to prevent raised ICP and its complications.

Firstly, that includes **elevation of the head of the bed** to improve venous drainage; maintenance of PaO₂ >100 mmHg and PaCO₂ 30-35 mmHg by adequate ventilation and oxygenation; use of the **lowest possible AW pressures** to avoid obstruction of venous drainage; keeping MAP at pre-intubation level.

Also **mannitol** (or 3 % saline) in 20-30 min intervals is used to reduce ICP by reducing **cerebral parenchymal cell water**. Mannitol decreases the intracranial pressure dramatically by decreasing the overall water content of the brain, reducing blood volume by vasoconstriction, and reducing cerebrospinal fluid volume. The end-result is increased cerebral blood perfusion. Recent studies are suggesting that mannitol might also have direct cyto-protective effects.

Sedation by decreasing anxiety, fear and response to pain block the increase of ICP. **Steroids** should be **avoided** in the treatment of **cytotoxic cerebral edema** which is the type of cerebral edema you see in a patient with traumatic brain injury. Steroids should be also avoided in patients with cerebral edema due to ischemic stroke and hemorrhagic stroke. In fact, steroids might be associated with a higher rate of complication in patients with traumatic brain injury.

In 2016, the brain trauma foundation gave **recommendations for specific treatment options** and desired **threshold values**:

- **Blood pressure** thresholds should not exceed 100 mmHg for patients 50 to 69 years old or 110 mmHg or above for patients 15 to 49 or above 70 years as it is considered to decrease mortality and improve outcomes.
- **Intracranial pressure** thresholds: treating ICP higher than 22 mmHg is

recommended because values above this level are associated with increased mortality.

- A **combination of ICP values and clinical and brain CT findings** may be used to make/alter management decisions.
- **Cerebral perfusion pressure:** recommended target CPP value for survival and adequate outcomes is 60 – 70 mmHg (although minimum optimal CPP threshold may depend upon the autoregulatory status of the patient).
- Avoiding aggressive attempts to maintain CPP higher than 70 mmHg with fluids and pressors because of the risk of **adult respiratory failure**.
- **Advanced cerebral monitoring**, such as jugular venous saturation lower than 50% may reduce mortality and improve outcomes.

Complications of Traumatic Brain Injury

Both mild and severe traumatic brain injury can lead to complications, the most common being **post-traumatic seizures**. The use of **anti-epileptics** is protective against **early** post-traumatic seizures, however, it appears it has no effect whatsoever on the risk of **late** post-traumatic seizures. Accordingly, prophylactic anti-epileptic treatment against early post-traumatic seizures should be discontinued on the seventh day of the in-hospital care of the traumatic brain injury patient.

Other complications of traumatic brain injury include:

- Communicative [hydrocephalus](#)
- [Deep vein thrombosis](#)
- Spasticity
- Gastrointestinal and genitourinary complications
- Heterotopic ossification
- Post-traumatic longstanding agitation

References

Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons. **Guidelines for the management of severe traumatic brain injury**, J Neurotrauma, 2007

Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition, via www.braintrauma.org

Samir H. Haddad, **Critical care management of severe traumatic brain injury in adults**, Scand J Trauma Resusc Emerg Med. 2012

Jha S. **Cerebral Edema and its Management**. Medical Journal, Armed Forces India. 2003;59(4):326-331. doi:10.1016/S0377-1237(03)80147- 8.

<https://www.nice.org.uk/guidance/cg176>

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