

Causes and consequences of increased intracranial pressure



CONSEQUENCES OF INCREASED INTRACRANIAL PRESSURE

What is the Intracranial Pressure?

Intracranial Pressure (ICP) is the hydrostatic pressure of the cerebrospinal fluid (CSF) in the subarachnoid space ^[4].

Cerebrospinal fluid is a watery fluid circulating in the subarachnoid space surrounding the brain and the spinal cord. This fluid is synthesized by the choroid plexus in cerebral ventricles and it is absorbed by the arachnoid granulations into the venous sinus system. So the CSF is the surrounding nature of the brain.

Increased intracranial pressure

Normal values for intracranial pressure are varying with age. Normal values for adults and older children are 10 to 15 mmHg, 3 to 7 mmHg for young children and 1.5 to 6 mmHg for term infants. ICP may be sub atmospheric in newborns ^[5].

Commonly 5 to 15 mmHg (7.5 to 20 cm H₂O) is concerned to be normal adult ICP value ^[5]. 20 to 30 mmHg values are concerned as mild intracranial hypertensions but 20 to 25 mmHg values requires treatments and values more than 40 mmHg are severe life threatening situations ^[5].

Causes for increased intracranial Pressure

Increased intracranial pressure can be developed either by an increase in the pressure in CSF or by a space occupying lesion (such as brain tumors,

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bleeding in the brain, fluid surrounding the brain or swelling of the brain tissue. But in some situations those two types of reasons can be interrelated with each other. (e. g.: When the brain is swelling, its vasculature becomes compressed and this may lead to increase ICP.

There is a concept called Monroe Kellie Doctrine. According to that hypothesis, skull is an enclosed rigid structure containing no compressive structures such as brain, blood and CSF. So an increase in one constituent or an expanding of one of them results in an increase in the intracranial pressure [6] Pg. 76 .

Intracranial = Brain + CSF + Blood + Mass lesion

Volume volume volume volume volume

But in infants, in the case of their skulls are not completely ossified, their skulls are some kind of incompatible with this hypothesis.

According to that hypothesis an expanding mass, an increase in brain water content , an increase in cerebral blood volume (by vasodilation or venous outflow obstruction) or increase in CSF are the factors for an increase in intracranial pressure [6] Pg. 76 .

However there are some compensatory mechanisms for regulating intracranial pressure increases [6] Pg. 76 .

- Immediate actions – 1. Decrease of CSF volume (CSF outflow to the lumbar theca.

2. Decrease of cerebral blood volume.

- Delayed actions – Decrease of extra cellular fluid.

There are number of causes responsible for increased intracranial pressure.

They may be either occurring individually or in combination with others.

1. Primary causes for increased ICP [7, 8, 9]

This is also called as Intracranial Causes. They occur within the cranium.

- Brain tumor – Tumors lead to increase in brain volume. So according to the Monroe Kellie doctrine ICP increased.
- Trauma – There are various types of head injuries. They can be close or open (penetrating) injuries. It can be lead to concussion (shaking of the brain as a result of trauma), scalp injuries, skull fractures. Those traumas may cause to bleeding within the brain tissue or bleeding in the layers that surrounds the brain.

There are three types of bleedings occurring in the layers surround the brain.

1. Subarachnoid hemorrhage – bleeding into the subarachnoid space.
2. Subdural hematoma – bleeding into the subdural space
3. Extradural hematoma – Bleeding into the epidural space.

All those types of bleedings cause in increasing ICP.

- Non traumatic intra cerebral hemorrhage – These hemorrhages can occur by aneurysm of cerebral arteries in the brain. Aneurysms are localized excessive swellings of an arterial wall. So they have more potential to rupture and this leads to subarachnoid hemorrhage.

- Ischemic stroke – Stroke (or Brain Attack) is the death of brain cells due to an inadequate blood flow ^[4]. So in strokes, as a response to the brain cell death, brain swelling occurs.
- Hydrocephalus – Hydrocephalus is an increase in CSF volume. Cerebrospinal fluid is secreted by the choroid plexus of the lateral, third and fourth ventricles and flows in a caudal direction and enters the sub arachnoid space through the foramina of Lushka and Magendie. At the end of the circulation absorb into the arachnoid granulations. Rate of formation CSF usually is about 500ml/day.

Hydrocephalus occurs commonly due to impaired absorption and rarely by excessive secretion. There are two types of hydrocephalus,

1. Obstructive hydrocephalus – Obstruction of the CSF flow within the ventricular system.
2. Communicating hydrocephalus – Obstruction of the CSF flow outside the ventricular system.

Increased intracranial pressure can be seen as a direct effect of hydrocephalus.

- Idiopathic (benign) intracranial hypertension ^{[6] Pg. 363} – This term means an increase in intracranial pressure without any mass lesion or hydrocephalus. Some clearly identified causal links (e. g.: venous outflow obstruction to CSF absorption) or obscured causal links (e. g.: diet, endocrine, hematological, drugs) cause for that.
- Other causes – Pseudotumor cerebri, pneumocephalus, abscess, cyst.

2. Secondary causes for increased ICP

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These are also called as extra cranial causes. So it is caused by extra cranial factors. Examples include,

- Airway obstruction
- Hypertension or hypotension
- Hypoxia or hypercarbia
- Posture
- Seizures
- Hyperpyrexia
- Drugs
- Other (High altitude, cerebral edema, hepatic failure)

3. Post-operative causes for increased ICP

This type of intracranial hypertension may occur after a neurosurgical procedure.

- Mass lesion/hematoma/edema
- An increase of cerebral blood volume by vasodilation.
- Disturbances of CSF flow.

Clinical symptoms appear with increased intracranial pressure ^[11, 12]

- Headache ^[11]
- Papilledema – This is the swelling of the optic nerve occur most of times as a result of increased intracranial pressure ^[10] .
- Projectile vomiting – This is a vomiting without nausea.
- Increased blood pressure
- Double vision

- Pupils do not respond to changes in light
- Visual field abnormality – Loss of peripheral and inferior nasal visions.
- Seizure or convulsions
- Neurological problems include balance issues, numbness and tingling, memory loss, paralysis, slurred or garbled speech or inability to talk.
- COMA
- Stiff neck – Neck becomes mostly stiff and painful. Some researchers says the reason for that is the stretching of the spinal nerve sheaths where they exit the spinal cord by the pressure passing down from the brain ^[10] .
- Pain in the arms
- Loss of consciousness
- Back pain
- Shallow breathing

In addition to those symptoms, in infants can be seen following ^[12] ,

- Separated sutures on the skull
- Bulging fontanellae
- Not feeding or responding normally

Consequences of increased intracranial pressure

Intracranial hypertension is involved with the pressure in the skull or in another words, the pressure around the brain and the spinal cord. Therefore increased intracranial pressure becomes a life threatening situation. There are some possibilities or consequences of increased intracranial pressure involved.

These complications can be categorized into different topics such as Displacement effects, Hydrocephalus, complications in cerebral perfusion, seizures.

- Displacement effects [5, 3, 6 pg. 76-80]

The cranial cavity is divided into compartments, separated by dural folds called falx cerebri and tentorium cerebelli. These folds limit brain structures within those compartments, but due to a space occupying lesion, pressure gradients occur in between these compartments. As a result brain shifts and herniation occur. This herniation can be categorized into two subjects,

1. Supratentorial herniation (uncal, central, cingulate, transclaviral)
2. Infratentorial herniation (upward, tonsillar)

Supratentorial herniation

1. Subfalcine herniation – In this case the cingulate gyrus is pushed laterally away from the expanding mass (like hematoma) beneath the falx cerebri. This may interfere with blood vessels in the frontal lobes which are placed at the site of injury. This may cause intracranial bleeding and severe rise in intracranial pressure and more dangerous types of herniation. Symptoms are not clear related to this herniation but usually present with abnormal posturing and coma. This type of herniation can be a precursor to other types of herniation.
2. Uncal (transtentorial, uncinata, mesial temporal) herniation – This is the herniation of the Uncas in medial temporal lobe from the middle cranial pressure into the posterior cranial fossa into the posterior cranial fossa across the tentorial opening. So the Uncas of the temporal

lobe is forced into the gap between the midbrain and the edge of the tentorium. There are main possible complications of this herniation,

1. Compression of cranial nerve (III) – In the case of herniation, ipsilateral oculomotor nerve may compress as it passes between the posterior and superior cerebellar arteries. In initial states ipsilateral dilation of pupil (do not respond to light) can be seen as the first clinical sign because the parasympathetic fibers are placed outside the nerve, which are getting paralyzed first during the compression. After that as the herniation improving moreover the contralateral pupil may also dilated and further compression of the nerve may lead to interfere with the somatic supply of extra ocular muscles (except lateral rectus which is supplied by abducent nerve and the superior oblique which is supplied by trochlear nerve) causing the deviation of the eye to downwards and outwards.
2. Compression of the midbrain cerebral peduncles – Commonly the ipsilateral cerebral peduncle gets compressed showing contralateral hemiparesis or hemiplegia. Since the herniation displaces the midbrain laterally, the contralateral cerebral peduncle gets compressed against the edge of the tentorium cerebelli resulting ipsilateral hemiparesis or hemiplegia (when it happens alone) or quadriplegia (when both cerebral peduncles are compressed).
3. Compression of the posterior cerebral artery – Posterior cerebral artery or its branches may be compressed against the free edge of the tentorium cerebelli causes hemorrhagic infraction on the medial and inferior sites of the ipsilateral occipital lobe. The lesion may often

confine to the posterior cerebral artery, leading to homonymous heminospia. If the occipital lobe lesions are bilateral, cortical blindness is a clinical sign (patient may not understand visual images, but pupillary reflexes are intact)

4. Compression of the brain stem – Compression of the brainstem may low in the midbrain and may gradually increase caudally. As a result patient will become comatose and develop cardiac and respiratory changes.

There are two types of events mainly occurring during the brainstem compression.

1. Secondary brainstem hemorrhages (Duret hemorrhages) – Due to the compression and stretching of vessels (especially veins) these hemorrhages occur. Death may ensure due to the direct destruction of the pons and midbrain.
2. Changes in respiratory, postural and oculomotor actions – These changes occur due to the compression transmitting downwards from the midbrain. Finally as a result of damage to the medulla leads to slow irregular respiratory movements, irregular pulse and falling of blood pressure, as well as death is due to the respiratory arrest.
3. Central herniation – This is due to a supratentorial space occupying lesion and downward displacement of brainstem and diencephalon. Progressive decline in neurological status so called Rostrocaudal Detoriation (or Rostrocaudal Decompensation) can be seen in this situation. Lesions located medially or within the frontal pole will not compress the midbrain and diencephalon laterally and they straight to

Rostrocaudal dysfunction of the brainstem leading bilateral progression of impairment.

Herniation may stretch the branches of the basilar (pontine) arteries and tear them generating Duret hemorrhage, usually causes to death because of the infraction of the midbrain and the pons. Clinical signs initiating with changes in consciousness start with reducing alertness leads to drowsiness, stupor and finally coma. There are list of incidents occur with central herniation and their related causes.

1. Respiratory changes due to various sites of lesions

Site of lesion Respiratory pattern

- Diencephalon Cheyne-Stokes respiration
- Midbrain Central neurogenic hyperventilation
- Pons Apneustic respiration
- Medulla Ataxic respiration

2. Changes in postural reflexes

- Decorticate Rigidity – Sign of leg extension and arm flexion caused by widespread lesions in the cerebral cortex.
- Decerebrate rigidity – sign of extension of both arms and legs due to the lesions disconnecting cerebral hemispheres from the brainstem (e.g. Upper midbrain lesions)

3. Pupillary changes – Studying those pupillary changes in comatose patients may helpful in revealing the general location of lesions.

1. Small reactive pupils – Compression of the diencephalon impairs sympathetic nerve fibers originate there and these impairment affects the sympathetic dilation of pupil straight to constricted small pupils.
2. Dilated fixed – Compression of one cranial nerve (iii) by the uncus compressing parasympathetic fibers travelling outside the nerve and this impairment of parasympathetic supply causes to dilate the pupil of the same side and loss of reaction to the light changes in that pupil.
3. Midposition fixed – Bilateral compression of both oculomotor nerves or compression of the midbrain results in impairment of both parasympathetic and sympathetic fibers in both sides travelling to the pupil and as a result pupils come in to a midposition and are non-responsive to light fluctuations.
4. Ocular movements – Pathways for ocular reflexes are localized in the brain stem, so that they are useful in testing pathways in comatose patients. Abducent and contralateral oculomotor nuclei are connected by the Medial Longitudinal Fasciculus (MLF) to produce conjugate deviation of the eyes.

Caloric stimulation or oculovestibular reflex is, when water put into ear, a passive head turning occurs. Usually the oculomotor responses to that stimulation also in a similar way producing oculoccephalic reflex or Doll's eye movements.

This eye movement does not occur in conscious patients because their pupil will stay looking straight ahead in front of the face when the head is turned, so this can be only seen in comatose patients.

When a comatose patient shows the oculoccephalic reflex, his brainstem is intact (Both eyes are deviated into the same sides opposite to the head movement, when the patient's head is turning side to side. The eyelids must open and hold to observe the deviation of eyes). But if the MLF is affected the eyes will not move towards the same side. But to show these results CN III should intact.

4. Extracranial/Transcalvarial herniation – This is the herniation of the brain through an opening in the cranial cavity formed by trauma or at a surgical site.

Infratentorial herniation

1. Tonsillar herniation ^[6] pg.: 79 – This is the downward herniation of the cerebellar tonsils through the foramen magnum. Usually caused by posterior cranial fossa mass lesion. But also can be due to a midline Supratentorial mass or as a result of edema. In that case the compression of the medulla leads to a depression of the vital centers for respiration and cardiac rhythm control. Sudden cardiorespiratory arrest or a slow progression over a day or two may be manifested as clinical symptoms.
2. Upward/Cerebellar herniation ^[5] – Increased pressure in the posterior cranial fossa leads to upward movement of the cerebellum through tentorial opening.

Midline shift of the brain

Midline shift is the shifting of the brain from its center line ^[1]. This is a direct result of increased intracranial pressure and can be occurred by traumatic <https://assignbuster.com/causes-and-consequences-of-increased-intracranial-pressure/>

brain injury, stroke, hematoma, or birth deformities. So midline shift can be used as an indicator of ICP and a midline shift of over 5mm indicates an immediate surgery ^[1]. There are 3 structures mainly investigating in a midline shift. They are septum pellucidum (between right and left ventricles), third ventricle and the pineal gland ^[2]. The degrees of displacement of these structures are aided in determining the severity of the shift.

Interaction with cerebral blood flow

There is a connection between cerebral perfusion pressure (CPP), mean systemic arterial pressure (MAP) and Intracranial pressure (ICP) as follows ^[5].

$$CPP = MAP - ICP$$

As the CPP is the driver of the cerebral perfusion, cerebral blood flow is determined by both MAP and ICP. Therefore CPP can be reduced by an increase of ICP or a decrease of MAP. However the brain can auto regulate the cerebral blood flow through an auto regulatory process in 50 to 150 mmHg CPP range. But below 50 mmHg CPP values the brain can't compensate and cerebral blood flow and cerebral perfusion pressure decreased.

Seizures

This is a sudden electrical activity of brain ^[7]. Most of the time acute increased intracranial pressure may cause for trigger a seizure ^[3].

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