430 Surgery Team

Presentation and Management of Raised Intracranial Pressure

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Green: Team's Notes
Blue: Further Explanations
Red: Important notes

Contents

Introduction

The cranium is a rigid container with openings known as Foramens. The major contents of the cranium:

- Brain
- Blood supply (arteries and veins)
- Cerebrospinal fluid (CSF)

The intracranial volume is constant and the normal intracranial contents are shown with intracranial pressure within a physiological range of 10-15 mmHg.

Expansion of the cranium in adults does not occur due to the rigidity of the skull, therefore, other mechanisms occur to compensate any increase in the intracranial pressue.

*In children whose sutures have not yet fused, the cranium itself can expand to accommodate extra volume in cases of increased intracranial pressure.

Figure 1.1 ***Intracranial Contents and their respective volumes***

Content	Volume
Brain (70%) and interstitial fluid (10%)	1400 mL
Blood	75 mL
CSF	75-100 mL

Figure 1.2 ***Normal Intracranial Pressure Values***

Age group	Normal Range (mmHg)
Adults	<10-15
Children	3-7
Term infants	1.5-6

Children have lower ranges of ICP because they have softer bones. Meanwhile, infants have an even lower value than children because their bones have not yet been unified

Basic Principles of Intracranial Pressure

❖ Monro-Kellie-Burrows Doctrine

The Monro-Kellie hypothesis states that: "the cranial compartment is incompressible, and the volume inside the cranium is a fixed volume. The cranium and its constituents (blood, CSF, and brain tissue) create a state of volume equilibrium, such that any increase in volume of one of the cranial constituents must be compensated by a decrease in volume of another"

Note

Brain parenchyma and arterial blood do not participate to any significant extent in the intracranial pressure-buffering mechanism

The compensatory mechanisms of Raised intracranial pressure according to this hypothesis are as following:

1- The venous system: it collapses easily, squeezing venous blood out through the jugular veins or through the emissary and scalp veins

2- CSF: it can be displaced through the foramen magnum into the spinal subarachnoid space

When these compensatory mechanisms have been exhausted, minute changes in volume produce precipitous increase in pressure.

❖ **Volume – Pressure Curve**

- Increase in the volume of one compartment will lead to change in the volume of the other ones.
- If any increase of volume occurs in the brain, an increase in ICP will occur, but with compensation mechanism, the cranium's structures can accommodate the increase in the pressure and maintain balance. When these compensatory mechanisms have been exhausted, even the slightest change in volume will lead to precipitous increase in pressure and at this point, the compensatory mechanisms will fail and the cranium components will no longer be able to accommodate for the raised ICP. At this point, the symptoms of increased ICP will start to appear on the patient.

These symptoms include:

- Headache
- Nausea
- Vomiting
- Numbness and weakness

- Compensation for the rise in the ICP depends on how fast is the increase occurring in;
  - Gradual increase in ICP will be tolerated and compensated for
  - Acute or rapid increase in ICP (e.g. sudden hemorrhage), there will be no time for compensation and the patient might go into coma

*Example: If a tumor develops in the brain, the compensation for the rise of the ICP related to the tumor's development will depend on how fast is the tumor growing [CSF will decrease then the blood volume will decrease]

- Clinical explanation of the curve:
  Example: A patient came to the ER with the symptoms listed above, the doctor examined him and he was conscious, but minutes later the patient collapsed and went into coma. This was later explained that patient was at **the point of the curve where the sudden rise of ICP occurs**.
  -At this point, the raise in 1 cm of the volume on the curve will lead to 3 or 4 times increase in the pressure. (due to fail in compensation)
  -Meanwhile at the early stage, the increase in 1 cm of the volume will lead to reasonable increase in pressure, which is still tolerated (due to compensatory mechanism)

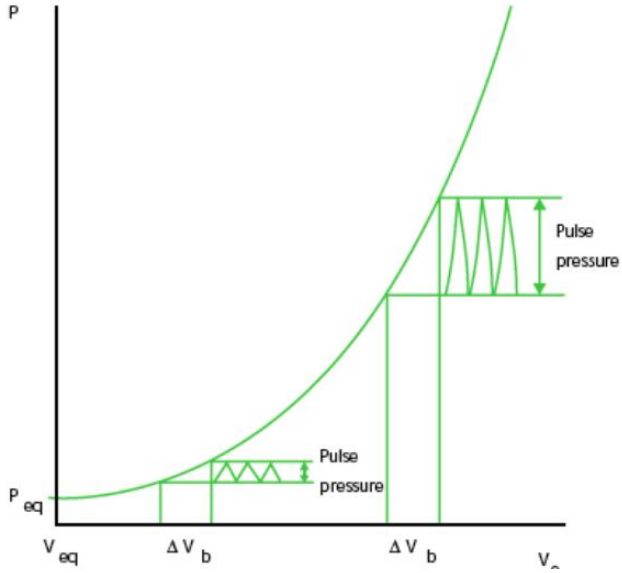

Fig. 1.3 *The volume-pressure curve of the intracranial environment*

Cerebral Blood Flow

Normal cerebral blood flow averages 55 to 60 mL/100g brain tissues/min. This flow is sufficient to meet the metabolic needs of the brain. The most significant factor that determines cerebral blood flow is the Cerebral Perfusion Pressure.

❖ **Cerebral Perfusion Pressure**

- It is the pressure resulting from the blood going into the brain
- **CPP= mean arterial pressure (MAP) – ICP** (with the increased ICP there is tendency for CPP to decrease)
- CPP has a wide range (50-140 mmHg) [in trauma cases (head injury) it is preferred to keep CPP around **70**]

❖ **Cerebral Autoregulation**

Measurement of CPP

- CPP = MAP-ICP
- MAP (Mean arterial pressure) = Systolic heart beats over the diastolic heart beats divided by 3
  - Measured by BP cuff that is connected to a monitor in the ICU
- ICP: measured by the insertion of a catheter into the head
- In cases of head injury, it is best to keep the CPP around 70 or above, so if CPP was around 40, BP must be increased or ICP decreased.
- In cases of increased ICP, the brain gets its perfusion by the mechanism of autoregulation

*Increase in MAP → increase CPP
*Increase in ICP → decrease CPP

Autoregulation is the ability to maintain a constant perfusion pressure under conditions where the blood flow is limited.

The brain dysfunctions when autregulation fails and an extreme decrease in pressure occurs or when there is an extreme rise in pressure.

Mechanism of Autoregulation:

1- Unlike the rest of the body's organs, the brain is unique in tolerating changes in blood pressure.
   - Rise in systolic BP → constriction of the cerebral arteries
   - Decreased Systolic BP → cerebral vessel dilation (compensation mechanism in order to keep the person conscious so they can fix the situation causing the drop e.g. dehydration, this will make the person able to go find water and drink it)
2- With consistent increase in blood pressure (HTN), small aneurysms will start to develop on small arterioles of the brain. When these aneurysms rupture, they will lead to hypertensive hemorrhage to the brain
3- Loss of auto regulation → changes in cerebral blood flow with changes of BP; increase in systolic BP will increase the blood flow to the brain. When it hits the point of extreme abnormality, the autoregulation will fail to maintain the pressure
4- Failure of autoregulation will lead to increase flow with increased BP → bleeding.
5- Failure of autoregulation may be due to: Contusion or Hematoma

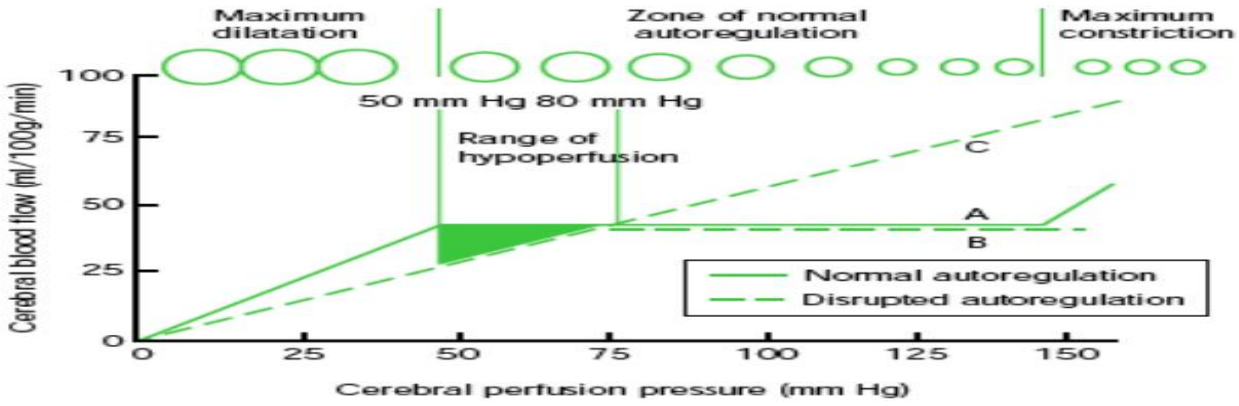

Fig 1.4 *Autoregulation of ICP*

Raised Intracranial Pressure

It is the increase in the intracranial pressure due to any abnormal content in the brain, e.g. Hematoma, tumor.

❖ Causes of Raised ICP

- **VITAMIN D** (Mnemonic)**: V**ascular, **I**nfection, **T**rauma, **A**utoimmune, **M**etabolic, **E**ndocrine, **N**eoplasm or **D**rugs
- Causes can be structural (e.g. CSF obstruction) or pathological (e.g. tumor)
- Pathological causes of raised ICP:

Table 2 Examples of causes of raised intracranial pressure

Pathological process	Examples
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	Benign intracranial hypertension

❖ Clinical Presentation

- Headache (most common sign)
  - ✓ **Early morning headache** (pt wakes up with severe headache)← Very Characteristic
  - ✓ Throbbing/Bursting
  - ✓ Increases with sneezing and coughing
- Nausea and vomiting
- Papilledema:
  - ✓ Is considered a reliable symptom but may take time to develop
  - ✓ It is important to examine the fundus in patients having raised ICP
  - ✓ If associated with fundal hemorrhage → acute and severe rise in ICP
  - ✓ Blurred optic disk, large tortures veins along with elevated and floored optic disc margins are seen on examination (See Fig. 1.5)
- Decreased level of consciousness (See GCS)
- Neurological manifestations:
  - ✓ **Pupillary Dilation**
  - ✓ Hemiplegia (Weakness)
  - ✓ Cranial nerve deficit

Pathophysiology

1- Headache:

*Early morning; because during night (sleeping), the patient is lying flat, there is no gravity resistance, so there will be increase flow to the brain → increased ICP

*Increases with sneezing and coughing; because sneezing and coughing will increase the intrathoracic pressure. This increase will prevent the blood from going down from the brain → increased ICP

2- Papillaedema:

Increased ICP will lead to congestion of the veins within the optic nerve → leading to the congestion of the optic nerve itself

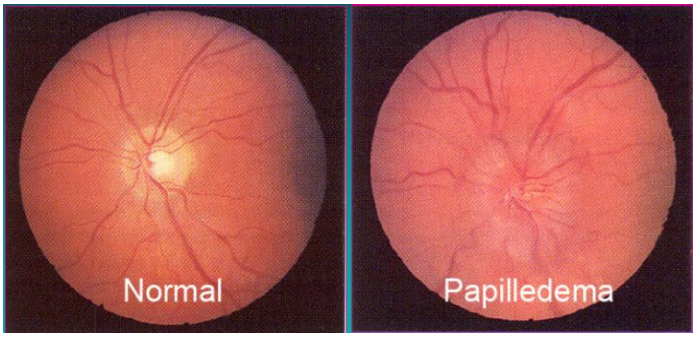

Fig 1.5 *Papilledema due to raised ICP*

Glasgow Coma Scale (GCS)

This is a scale used by ER physicians, neurologists and Neurosurgeons mostly to assess the patient's level of consciousness. In patients with increased ICP, decreased level of consciousness may be present

Figure 1.6 Glasgow Coma Scale

Eye opening (E)	Verbal Response (V)	Motor response (M)
4= Spontaneous **3= To voice** **2= To pain** **1=None**	5= Normal conversation 4= Disoriented conversation 3= Words, but not coherent 2= No words, only sounds 1= None	6= Normal 5= Localizes to pain 4= Withdraws to pain 3= Decorticate posture 2= Decerebrate 1= None
		Total= E+V+M

Fig. 1.6 *Glasgow Coma Scale*

Highest score = 15, Lowest score = 3

Pupillary Dilation

- One of the earliest signs (characteristic)
- Pathophysiology:

* Occulomotor nerve (from midbrain in the brainstem, near the temporal lobe → pupil dilation and constriction (if a lesion pushes the temporal lobe → nerve compression)
*In the beginning of herniation, this nerve will be affected
*The presence of a mass that is compressing the 3rd nerve → ipsilateral pupil dilation and contralateral hemiplegia

- Systemic reaction to increased ICP:
  - Increased BP for compensation (if you decrease the MAP this will kill the patient)
  - If the ICP is increased, how do we maintain good CPP ? → increasing the MAP
  - Respiratory changes : Cheyne – Stokes breathing
- Kernohan's Notch

  -Clinically this sign is seen through: Ipsilateral weakness and contralateral dilated pupils

  -This sign is mainly used to clinically if there is bleeding in the brain to estimate the side of bleeding

  -CT scan is done before taking the patient to the OR to identify the exact location of bleeding

- Raised ICP in infants

  Because infants' skull's sutures are not yet fused, this will give them the ability to accommodate the increased ICP by widening the skull, which results in:
  - Widened sutures
  - Increased head circumference
  - Dilated head veins

Cheyne – Stokes Breathing

Pressure on the brainstem will lead to pattern of breathing in which there is a sudden arrest in breathing (apnea) followed by suddenly breathing fast (tachypnea)
The pattern repeats, with each cycle usually taking 30 secs – 2 mins
This is not seen in all cases of increased ICP

Kernohan's Notch

When there is a growing hematoma for example in the right side, with the increase in size of the hematoma, it will compress the brain and pushes it to the opposite side and the whole brainstem will pushed against the contralateral tentorium resulting in
- Ipsilateral weakness
- Contralateral dilated pupil

- "Sun-set eyes" [the eyes are pushed down making the patient appear as always looking down]
- Head is enlarged with tense and bulging fontales (esp. when the baby is crying)

❖ Complications

- Brain herniations: The brain is supported by dural folds the prevent movement of the brain. There are 2 major dural folds; the falx cerebri and the tentorium cerebella.

  There are 5 main types of brain herniations:

  1- Cingulate Herniation (Sub-falcine herniation)
  Mass develops in supratentorial compartment →pushes the cingulate gyrus causing it to herniated under the falx to the opposite side
  There are no clinical signs and symptoms specific to a cingulated herniation

  2- Uncal Herniation (most dramatic and most common herniation observed clinically)
  The uncus is the inferomedial-most structure of the temporal lobe. An expansile mass (increasing the ICP) will cause the uncus to herniate above the tentorium, compressing the brain stem.
  The uncal herniation will manifest clinically as:
  - Decreased level of consciousness (patient might go into coma)
  - Dilated ipsilateral pupil (3rd cranial nerve affected)
  - Contralateral hemiplegia

  3- Central transtentorial herniation
  If there is a mass or lesion compressing the upper part of the brain, there will be a downward displacement of the entire brain through the tentorial incisura
  Clinical manifestation of central herniation:
  - Bilaterally small reactive pupils
  - Cheyne – Stokes Breathing

  4- Tonsillar Herniation
  This type of herniation is the most fatal type, results mainly from acute expansion of the posterior fossa lesions. So, a enlarged lesion near to the cerebellum causing increased ICP will cause the tonsil of the cerebellum to herniated through the foramen magnum into the upper spinal canal, compressing the lower medulla (where the center of respiration lays → Patient will stop breathing)

  5- Outside herniation
  If a skull fracture is present and there was massive increase of ICP → brain will herniate through the fracture (easiest way to go out)

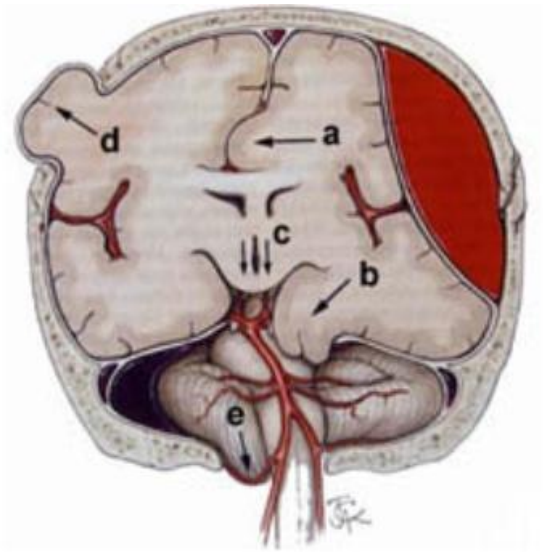

Fig. 1.7 *Brain Herniations*
a- Cingulate herniation
b- Uncal herniation
c- Central herniation
d- Outside herniation
e- Tonsillar herniation

❖ Investigations

The most significant factor determining morbidity and mortality in patients with neurosurgical disorders is increased intracranial pressure.

- To check whether the patient has increased ICP or not: Any patient coming to the ER with headache and vomiting → check for papilledema and do an urgent head CT
- If patient comes in with signs of infection (fever) along with headache and vomiting, first check for papilledema and do an urgent head CT, if both are negative, then you can do an LP (Lumber Puncture) to exclude Meningitis (LP is contraindicated in increased ICP)
- ICP Waveforms

  Any pressure in the body has a waveform. The waveform of the ICP is corresponded to the Cardiac waveform;

- During Systole phase in the heart →Pulsation of the heart → great vessels → internal carotid artery → pulsation in the brain → rise in the ICP waveform
- The waveform of normal ICP typically shows 3 arterial components superimposed on the respiratory rhythm

- ICP is best measured directly and continuously from the cranial cavity. There are 2 commonly used pressure-monitoring systems in contemporary neurosurgical practice.
  1- Intraventricular catheter : ICP is mainly monitored through the insertion of the catheter in the right ventricle of the brain → measure pressure and reduce fluid
  2- Fiberoptic transducer – tipped catheter system

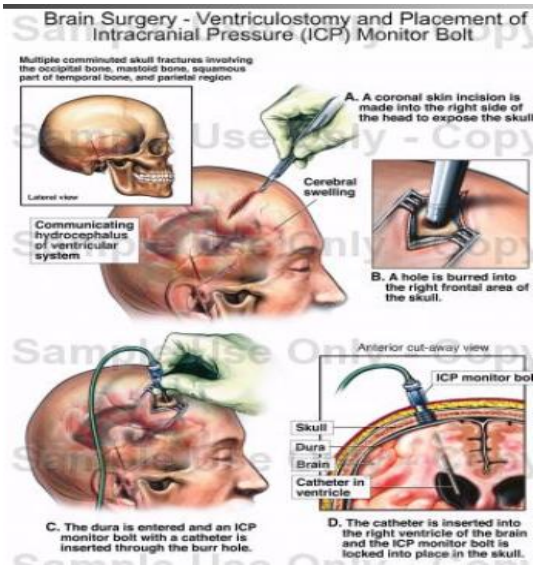

Fig. 1.8 *Monitoring ICP through intraventricular catheter*

❖ Treatment

- General measures:
  - Elevation of the head (30 degree) → increase the venour return
  - No neck compression
  - Mannitol (osmotic diuretic) for patients who have decreased level of consciousness (or furosemide)
  - Steroids (Dexamethazone) for **tumors only** (to control the edema around the tumor)
  - Hyperventilation: controlled to PCO2 = 35-40 mmHg (Hyperventilation will get rid of CO2 (CO2 is a potent vasodilator)→ leading to decrease in blood vessels' size → less blood will reach the brain)
  - Sedation, muscle relaxants and Hypothermia to decrease the metabolic rate
  - Barbiturates (Terminal option): induction of coma with short acting barbiturates is the last resort in managing increased ICP when all other measures fail. Short acting barbiturates are introduced to put the brain in relaxation

- Specific treatment: The most direct way to normalize raised ICP to the physiologic range is to **<u>eliminate the cause</u>** (Causes; remember "VITAMIN D" mnemonic)
    - Vascular – SAH/ICH
    - Infection/ Abscess: sustained infection that appears a round space that is enhanced when given contrast. It appears mainly in IV drug abusers and immunocompromised patients with sinusitis
    - Trauma: it can be either
        1- Localized (e.g. Epidural and subdural hematomas), will lead to the compression of the brain → herniation (subfalcine herniation and temporal herniation)
        2- Diffused: commonly due to rotational forces . The diffused scattered blood inside the brain will give a salt and pepper appearance
        3- Tumor: Dissection of the tumor
        4- Hydrocephalus: Ventricles are enlarged and CSF is diffused inside the brain → treatment: Shunt
- Case Example:
  A 35 yr old gentleman was brought by peramedics, they explained that he was in a motor vehicle crash. In the ER, patient was unconscious, with increased HR and decreased BP and right hemiplegia.
  How do we manage this patient?
    1- ABC:
        - Airway → Endotracheal intubation (for any unconscious patient, always intubate because if the airways are blocked, the patient will die within seconds)
        - Breathing → Chest tube
        - Circulation → IV fluid infusion then check for any external bleeding, stop it
    2- 2 large central IV lines to restore fluid and blood (if needed)
    3- Urgent head CT
    4- If fever is present, LP after CT, if the CT ruled out increased ICP (**LP is contraindicated in increased ICP**)

References

1. Amro AlHabib, M.D. – Presentation and Management of Raised Intracranial Pressure, 2013
2. Setti S. Rengachary, Robert H. Wilkins – Principles of Neurosurgery; Mosby-Year Book Europe; 1994
3. John A. Jane, M.D., PhD – Clinical Neurosurgery; Williams and Wikins; 1996
4. Peter F. Lawrence – Essentials of Surgical Specialties; William and Wikins; 2007