Control Of Intracranial Pressure and Basic Considerations in Neuroanaesthesia

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INTRODUCTION

Intracranial pressure (ICP) is the pressure exerted by the cranium on the brain tissue, cerebrospinal fluid (CSF), and the brain's circulating blood volume. Is the pressure inside the lateral ventricles and lumbar subarachnoid space in supine position. it is constantly fluctuating in response to activities and measured in millimeters of mercury (mmHg).

ANATOMY

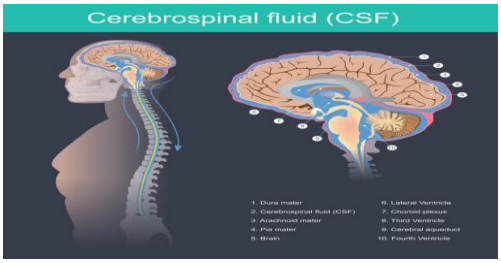


FIGURE 1: ANATOMY OF THE BRAIN AND SPINAL CORD

PHYSIOLOGY

The brain receives about 20% of total body oxygen consumption and 25% of total body glucose consumption, the cerebral metabolic rate of oxygen (CMRO2) is normally 3 to 3.8 mL/100 g/min. Brain glucose consumption is approximately 5 mg/100 g/min. The brain receives about 15% of the cardiac output to meet these high metabolic requirements. Normal cerebral blood flow (CBF) is 50 mL/100 g/min or 750 mL/min. The regional CBF is 80ml/100g/min for the gray mater and 20ml/100g/min for the white mater**.** The critical CBF which is the CBF below which cerebral ischemia becomes evident on electroencephalography (EEG) as slowing of EEG waves ~~It~~ is 20-25ml/100g/min. When CBF is 15-20ml/100g/min causes a flat isoelectric EEG and when the CBF<10ml/100g/min causes irreversible brain injury.[1]

Control of CBF

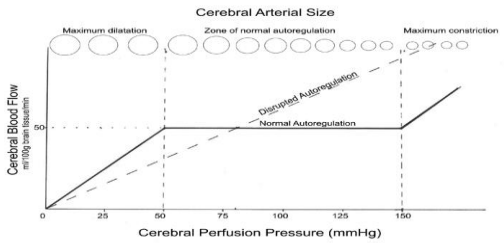
Cerebral perfusion pressure (CPP): is defined as

CPP = MAP – (CVP + ICP)

CPP is usually 70 – 90mmHg, when it is < 50mmHg, slowing of EEG occurs, when it is 25-40mmHg, , flat isoelectric EEG tracing is seen; and when it is < 25mmHg, irreversible brain injury occurs.

Cerebral autoregulation:

Is a physiological process which refers to the capacity of cerebral circulation to adjust its resistance to maintain a constant CBF regardless of changing systemic blood pressure/CPP, it occurs between MAP of 50–150 mm Hg



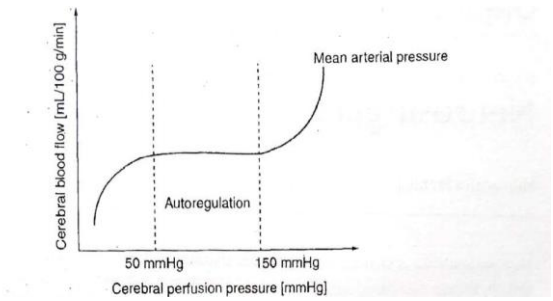


FIGURE 2: CEREBRAL AUTOREGULATION CURVE

A. Respiratory gas tension

1. Arterial carbon dioxide tension ($PaCO_2$):

CBF is directly proportional to $PaCO_2$ tension between 20-80mmHg, CBF changes 1-2ml/100g/min per 1mmHg change in $PaCO_2$.Hyperventilation reduces the $PaCO_2$ and causes vasoconstriction of the cerebral vessels and therefore reduces cerebral blood volume and ICP. Hypercapnia and the resulting vasodilatation and increase in ICP must also be avoided, $PaCO_2$ is therefore best maintained at low-normal levels to prevent raising ICP (35-40mmHg, 4.7-5.3kPa).

2. Arterial oxygen tension (PaO_2):

Oxygen has little effect on the radius of blood vessels at partial pressures used clinically. CBF increases once PaO_2 drops below about 6.7kPa so that cerebral oxygen delivery remains constant. Increasing oxygen will have the reverse effect and causes vasoconstriction which is not clinically significant.

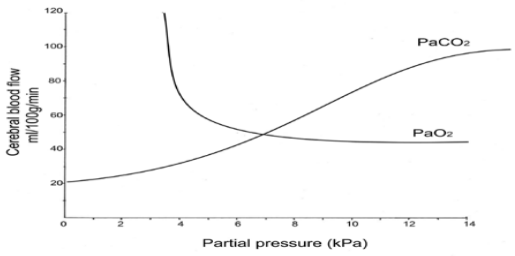


FIGURE 3: THE EFFECT OF $PaCO_2$ and PaO_2 ON CBF

B. Temperature

Decreasing temperature decreases cerebral metabolism and the reverse occurs when temperature is increased. For every 18°C decrease in brain temperature, CMR and hence the CBF decrease by 7%. Decreased temperature causes vasoconstriction which decreases CBF and CMR. Cooling to 32–34°C is recommended in treatment of raised ICP refractory to other treatment modalities, at 20°C EEG becomes isoelectric.

C. Blood viscosity

Is mainly determined by haematocrit (Hct). Hct between 30-50% has a little effect on CBF, CBF varies inversely with Hct. Hct <30% increases CBF as that which occurs with cardiopulmonary bypass, but this also decreases O_2 carrying capacity of blood while Hct >50% decreases CBF as that which occurs with marked polycythaemia.[1,2,3]

CEREBROSPINAL FLUID

The CSF is produced mainly by the choroid plexus of the ventricles and ependymal cells at rate of 0.3-0.4ml/min (500ml/day), drained by the arachnoid villi and arachnoid granulations into the dural venous sinuses. The production of CSF is constant, but if re-absorption is hampered or there is a mechanical obstruction to the CSF outflow, its volume increases causing an increase in ICP.[4]

Function of CSF

- Mechanical protection by buoyancy
- CSF provides a constant chemical environment for neuronal activity
- CSF is important for acid-base regulation for control of respiration
- CSF provides a medium for nutrients after they are transported actively across the blood-brain-barrier

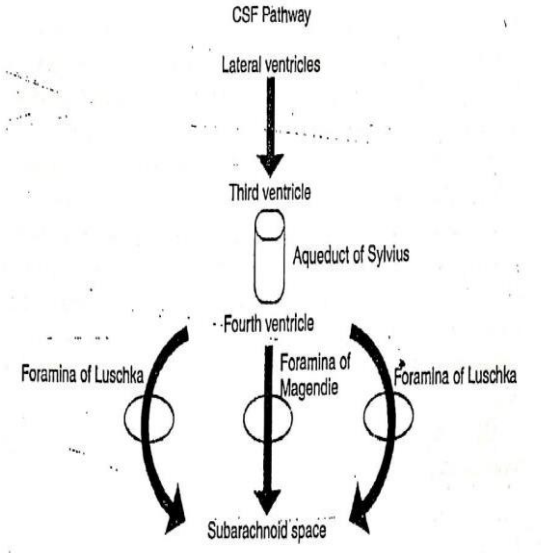


FIGURE 4: CSF PATHWAY

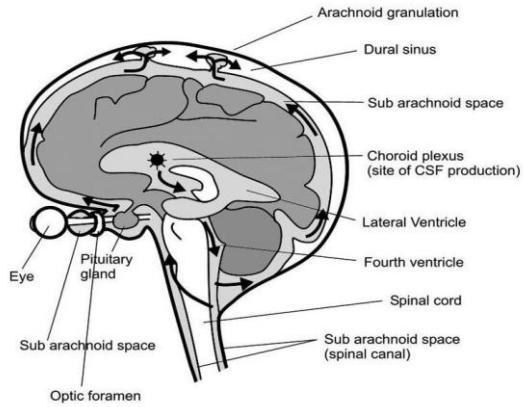


FIGURE 5: PRODUCTION, CIRCULATION, AND ABSORPTION OF CSF (UPDATE IN ANAESTHESIA TUTORIAL 71)

PATHOPHYSIOLOGY

ICP is the pressure within the intracranial cavity, the brain is enclosed within the rigid skull with fixed volume consisting of brain (80%), blood (12%) and the CSF (8%) (Monro-Kelly doctrine).

Normal ICP <15 mmHg in adult

- 3-7mmHg in children

-1.5-6mmHg in term infants

Elevated ICP may be caused by a variety of pathophysiologic mechanisms. The most common contributing factor to increased ICP is cerebral oedema. Mechanisms that result incerebral oedema aregenerally classified into three major forms:

-Cytotoxic oedema; this results from intracellular fluid accumulation within the brain cells cause by cellular injury, typically seen in condition like stoke, trauma, or hypoxia. Cytotoxic oedema is a critical condition that often requires rapid intervention to prevent further injury.

-Vasogenic oedema; is a type of cerebral oedema that results from the breakdown of the blood brain barrier (BBB), leading to an accumulation of fluid in the extracellular space. It commonly occurs in conditions like tumours, infections, trauma, and inflammation. Unlike cytotoxic oedema, vasogenic oedema affects primarily the white matter and can be often be reversible with appropriate treatment to restore BBB intergrity.

-Interstitial oedema; is a type of cerebral oedema associated with obstructive hydrocephalus or elevated cerebrospinal fluid pressure. Interstitial oedema is often associated with hydrocephalus and can worsen if the underlying CSF flow obstruction is not treated.

Increased CBV, because of either increased arterial inflow or decreased venous efflux, can lead to increases in ICP. Inadequate CSF absorption, due to diminished absorption at the arachnoid villi may cause hydrocephalus and elevated ICP. Mass effect from tumours, hematomas, or abscesses can elevate ICP by a direct space-occupying effect. The consequent rise in ICP opposes cerebral perfusion pressure and limits cerebral oxygen delivery. Neuronal cells utilize glucose as a primary and only energy source even during starvation where ketone bodies are available for the rest of body cells. Both hypoglycemia and hyperglycemia are harmful to the brain, the brain depends on a continuous supply of oxygen and glucose, with irreversible injury potentially occurring after only 4 to 5 minutes of global ischemia.

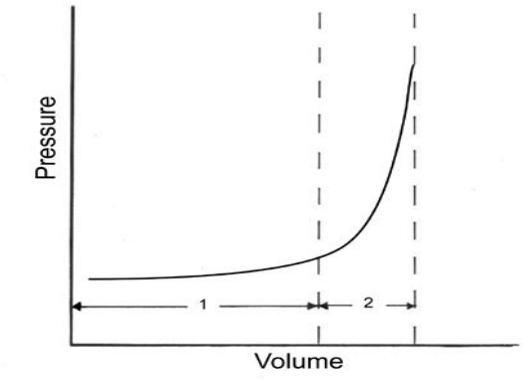
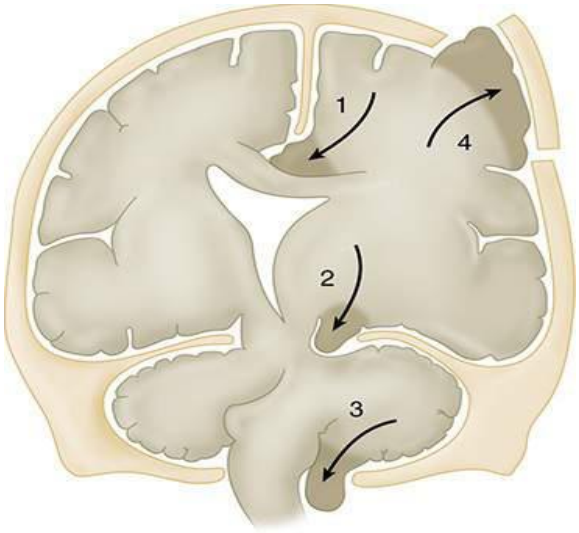


FIGURE 6: INTRACRANIAL COMPLIANCE

ICP that is raised to a sufficient level, for a sufficient duration, causes brainstem ischaemia and death. Sustained elevations in ICP can lead to catastrophic herniation of the brain, herniation may occur at one of four sites:

1. The cingulate gyrus under the falx cerebri (subfalcine herniation)
2. The uncinate gyrus through the tentorium cerebelli (uncal herniation)
3. Any area beneath a defect in the skull (trans calvarial herniation)
4. The cerebellar tonsils through the foramen magnum (tonsillar herniation).[1]



1. SUBFALCINE
2. UNCAL
3. TONSILLAR
4. TRANSCALVARIAL

FIGURE 7: SITES OF BRAIN HERNIATIONS

EFFECTS OF DRUGS ON CBF

1. Inhalational agents

- N_2O
  - This is controversial.
  - Causes cerebral vasodilatation which is attenuated by hyperventilation
  - Avoided when intracranial airspaces exist
- All volatile anaesthetic agents increase CBF
  - Isoflurane most potent in decreasing $CMR0_2$

2. Intravenous anaesthetic agents

- Most cause decrease in CBF & $CMRO_2$ except for ketamine.
- Lidocaine decreases CBF & $CMRO_2$.
- Autoregulation and CO_2 responsiveness is preserved.

3. Muscle relaxants

- No direct effect on CBF & $CMRO_2$ because they don't cross the blood brain barrier.

4. Vasoactive drugs

- **Vasopressors**
  - Phenylephrine, epinephrine & norepinephrine may increase CPP indirectly.
  - Have little effect on ICP because they do not cross the BBB.
- **Vasodilators**
  - Na nitroprusside, nitroglycerin and hydralazine can ↑CBF & ICP by direct cerebral vasodilatation.
  - ß blockers have minimal effect.

TABLE 1: Comparative effects of anaesthetic agents on cerebral physiology (Morgan & Makhail's Clinical Anaesthesiology - 6th edition)

Agent	CMR	CBF	CSF Production	CSF Absorption	CBV	ICP
Halothane	↓↓	↑↑↑	↓	↓	↑↑	↑↑
Isoflurane	↓↓↓	↑	±	↑	↑↑	↑
Desflurane	↓↓↓	↑	↑	↓	↑	↑
Sevoflurane	↓↓↓	↑	?	?	↑	↑
Nitrous oxide	↓	↑	±	±	±	↑
Barbiturates	↓↓↓↓	↓↓↓	±	↑	↓↓	↓↓↓
Etomidate	↓↓↓	↓↓	±	↑	↓↓	↓↓
Propofol	↓↓↓	↓↓↓↓	?	?	↓↓	↓↓
Benzodiazepines	↓↓	↓	±	↑	↓	↓
Ketamine	±	↑↑	±	↓	↑↑	↑↑
Opioids	±	±	±	↑	±	±
Lidocaine	↓↓	↓↓	?	?	↓↓	↓↓

↑↑, increase; ↓, decrease; ±, little or no change; ?, unknown; CMR, cerebral metabolic rate; CBF, cerebral blood flow; CSF, cerebrospinal fluid; CBV, cerebral blood volume; ICP, intracranial pressure.

CLINICAL FEATURES OF RAISED ICP

Early features:

- Headache,
- Nausea and vomiting,
- Papilloedema,
- Seizures.

Late features:

- Focal neurological lesions, increasing blood pressure and bradycardia.
- Agitation, drowsiness, coma.
- Irregular ventilatory pattern, Apnea.
- Ipsilateral then bilateral papillary dilatation.
- Decorticate then decerebrate posture.

INVESTIGATIONS

- **Computerized tomography of Brain (Brain CT scan)**
  - This measures the size and shows the location of the lesion, size of the ventricles, presence of midline shift and evidence of generalizedor peri-tumour cerebral oedema

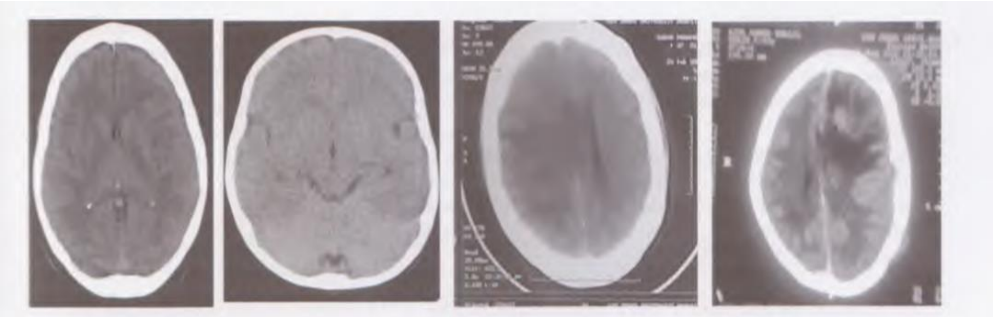


FIGURE 8: CT SCAN OF THE BRAIN (SPOTLIGHTS IN ANAESTHESIA VOLUME 2)

- **Magnetic Resonance Imaging of the Brain (Brain MRI):**

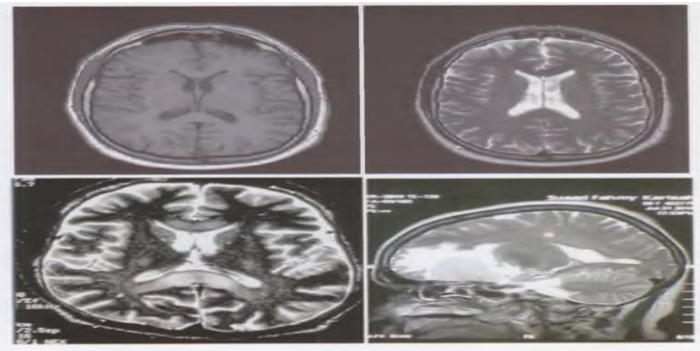


FIGURE 9: BRAIN MRI (SPOTLIGHTS IN ANASTHESIA VOLUME 2)

TABLE 2. Characteristics of different methods of ICP monitoring.

Methods	Advantages	Disadvantages
Intraventricular catheter	• Gold standard • Allows drainage of CSF to lower ICP • Re-zeroing possible	• most invasive method • high infection rate • difficult to insert
Intraparenchymal probe	• low infection rate	• measure local pressure
Subarachnoid probe	• low infection rate • no brain penetration	• limited accuracy • high failure rate • periodic flushing necessary
Lumbar CSF pressure	• extracranial procedure	• inaccurate reflection of CSF may be dangerous when brain oedema is present
Tympanic membrane displacement	• non invasive	• insuffucient precision
Transacranial doppler	• non invasive	• limited precision

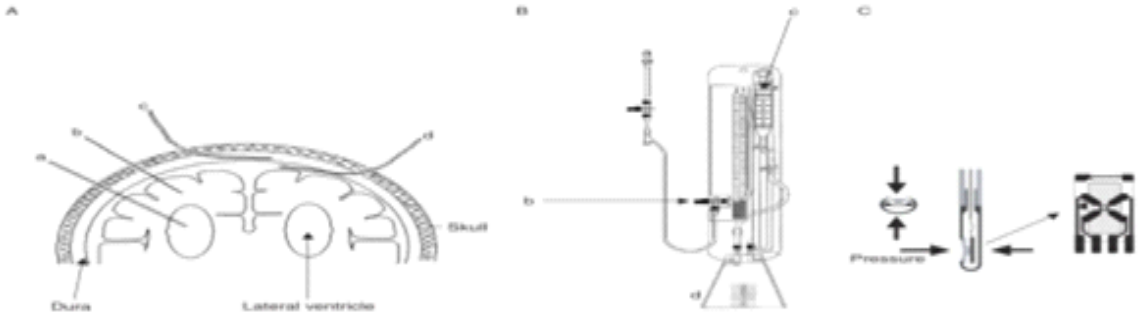


FIGURE 10: ICP MONITORING

TABLE 3: ICP monitoring waveforms

Wave phase	Wave name	Represents
P1	Percussion	Arterial pulsation
P2	Tidal	Intracranial compliance
P3	Dicrotic	Aortic valve closure

The height order of the wave phases on monitoring should be P1>P2>P3. If P2 is highest, this indicates potential raised ICP.

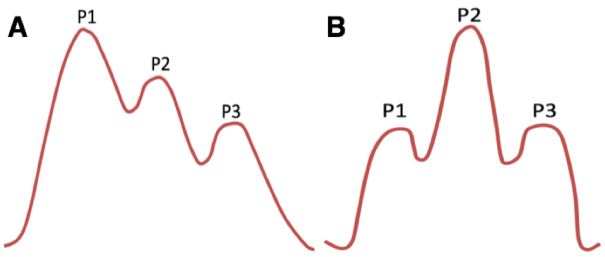
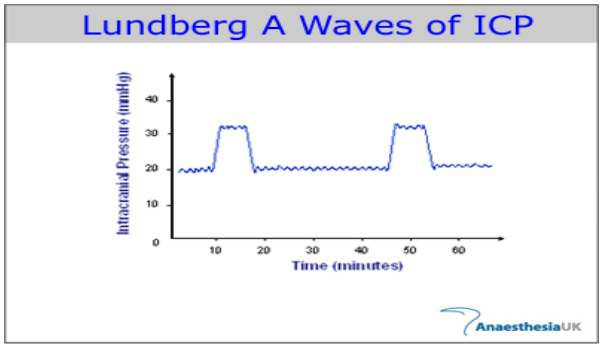


FIGURE 11: ICP WAVEFORMS

TABLE 4: THE WAVEFORMS SEEN ON ICP TRACE

The waveforms seen on the ICP trace

	Associated with	Amplitude	Duration
A (plateau) waves	Cerebral vasodilatation Reduced cerebral compliance	50–200 mm Hg	5–20 min
B waves	Changes in respiratory pattern	<50 mm Hg	1 minute
C waves	Blood pressure and systemic vasomotor tone	<20 mm Hg	7–15 s



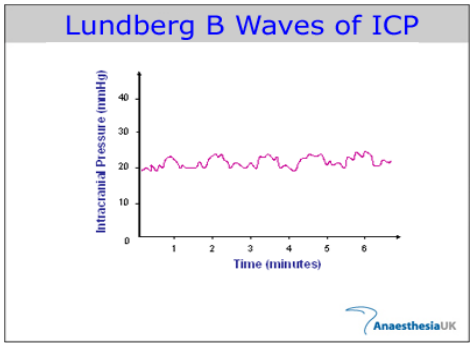


FIGURE 12: LUNDBERG WAVES OF ICP

ANAESTHETIC CONSIDERATIONS

- Emergency or elective.
- Prolonged and delicate surgery.
- Maintenance of ICP.
- Remote access to the patient.
- Unusual positioning of the patient.
- Unusual equipment and technique e.g. stereotactic devices and intra-operative wake-up in epilepsy surgery.[5]

A. Pre-operative assessment

A routine pre-anaesthetic evaluation for clinical assessment, risk stratification, and optimization before surgery. This would include:

- **Adequate History**
    - General and Neurological

      History of headache, nausea, vomiting, visual disturbance, gait disturbance, seizures, polydypsia, menstrual irregularities etc, Past medical and surgical history, history of other comorbidities, medications, and alcohol

➢ **Physical examination and neurological examination**

The patient should be examined from head to toe; checking for fever, dehydration, pallor, jaundice, cyanosis.

On neurological examination, the level of consciousness will be assessed for any alteration in mental status that could indicate raised ICP, pupillary diltation will be assess to rule out raises ICP or brainstem compression. Motor functions, sensory functions, cranial nerves and reflexes should all be assessed.

➢ **Investigations:**

Basic:

FBC: adequate haemoglobin (Hb) for oxygen delivery is essential in neurosurgery, as the brain is very sensitive to hypoxia. Low Hb levels may compromise oxygen delivery and increase the risk of ischemic injury. If Hb is low it may be necessary to optimize with transfusion or other measures preoperatively. Low platelet count could increase bleeding risks during surgery, particularly in procedures involving vascular brain tissues. Elevated white blood cell may indicate infection, which is a concern because neurosurgery requires a sterile environment to prevent complications like meningitis or abscess.

Electrolytes, Urea and Creatinine, are essential to ensure optimal brain function, fluid balance, and renal status before surgery.

Liver Function Tests are important for assessing the liver's ability to metabolize drugs, manage coagulation, and handle perioperative stress. Chest X-Ray is often reviewed to assess the patient's respiratory and cardiac status, which can impact anaesthesia management and overall surgical risk.

ECG, ECHO are vital tools for assessing cardiac condition as haemodynamic stability is crucial for brain perfusion and safe anaesthesia.

Special radiological (CT, MRI): CT scan detect acute intracranial bleeding, identifies tumours, detect ventricular enlargement, as well as cerebral oedema, while MRI detect and differentiate tumours, provides detailed imaging of the ventricles and CSF flow.

➢ Group and crossmatch blood for intra-operative use

➢ Informed consent

➢ Fasting guidelines

➢ ASA status classification.

➢ Premedication: Sedative or opioid, corticosteroids and anticonvulsants.

➢ Prophylactic antibiotics: broad spectrum preferably.

➢ Counsel patient and relatives for possible ICU or HDU admission.

B. Intra-operative management

Ideal induction agent for neuro-anaesthesia should:

1. Decrease the cerebral metabolic rate of oxygen ($CMRO_2$).
2. Maintain adequate balance between cerebral blood flow (CBF) and tissue metabolic requirements.
3. Maintain cerebral perfusion pressure (CPP) and CBF.
4. Maintain cerebral autoregulation and vascular reactivity to CO_2.
5. Have anticonvulsant action.
6. Have a short and easily reversible action.

Reassess the patient's neurological status before induction and confirm availability of ICU or HDU.

Establish venous access with large bore cannulae and central line for correction of fluid deficit and maintainance and for administration of drugs.
Monitors: ECG, NIBP, pulse oximeter, and capnography for minor cases.
Additional monitors: urine output, temperature, neuromuscular blockade, invasive BP, ABG and CVP (for major cases).

INDUCTION

Goal is to have a smooth induction and limit the hypertensive response to laryngoscopy and intubation, prevent secondary insult to already injured brain.

- Prevent rises in ICP
- Secure the tube
- Position- could besupine, prone, sitting,

Smooth induction:

- To avoid further increase in ICP is a good choice.
- The pressor response of intubation can be avoided.

Induction agent: avoid drugs which increase CBF and ICP

Thiopentone provides greater brain protection as it decreases CBF and ICP, Propofol decreases CBF and ICP and also allow early recovery and etomidate decreases CBF and ICP. However, ketamine should be avoided, because it increases CBF and ICP.

Inhalational induction in children should be avoided because it increases the CBF and ICP.

Muscle relaxants:

Nondepolarizing muscle relaxant such as rocuronium, vecuronium, cis-atracurium can be used. Succinylcholine may increase ICP due to fasciculations. The endotracheal tube should be armoured latex (non-kinkable) tube and should be well secured and rechecked after positioning.

MAINTAINANCE

Anaesthesia should be maintained byTIVA with propofol or inhalation technique with volatile agent. Neuromuscular blocking agents (NMBA) can be administered by continuous infusion or intermittent boluses and analgesia maintained with intermittent boluses of fentanyl orremifentanil.

Permissive hyperventilation:

Is needed to maintain PaCO2 between 35-40mmHg to decrease ICP, avoid severe hypocarbia as it causes cerebral vasoconstriction which increases cerebral ischemia, avoid hypercarbia as it causes cerebral vasodilatation resulting in increase in CBF and ICP. Avoid PEEP and high airway pressures because both increase CVP which in turn increases ICP.

Fluid management:

IV fluids should be used judiciously and be sufficient to maintain IV volume and haemodynamic stability. Dextrose containing solutions should be avoided unless indicated and hyperosmolar fluids cause fluid shift. Normal saline (0.9%) is the preferred crystalloid.

Temperature control:

Permissive hypothermia between 33-35^{o}C decreases $CMRO_2$ and may increase the period of ischemia tolerated intra-operatively. Normothermia should be achieved before patient awakens to avoid shivering which markedly increases oxygen demand.

Thromboembolic prophylaxis:

Neurological patients are at risk for DVT and PE. Heparin should not be used because of risk of bleeding in confined cavity.

Mechanical means – graduated compression stocking and intermittent pneumatic stocking can be used.

C. Post-operative management

Emergence:

The patient should not be allowed to cough through the endotracheal tube.

Systemic hypertension is common and may contribute to the development of post-operative haematoma. Extubation should be smooth and impeccable.

After application of head dressing and full access to the patient is regained, extubation is done.

Post-operative elective ventilatory support

This may be considered depending on:

- Patient’s pre-operative neurological status.
- Intra-operative events (duration and complexity of surgery, haemodynamic instability).
- Evidence of raised ICP (tense dura/tight brain).

Post-operative monitoring:

- Regular neurological observations.

- Any neurological deterioration should raise suspicious of Intracerebral bleeding/oedema
- Haemodynamics should be closely monitored to maintain adequate CPP.
- Urine output should be monitored.

CONCLUSION

Neuroanaesthesia is an extremely interesting and fascinating aspect of the anaesthetic practice. It involves the anaesthetic management of neurosurgical patients and requires the understanding of anatomy, pathophysiology and the disease process. Specific technical skills, and co-operation with a wider number of team members and clear communication with the patient and their family is paramount.

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