Recent advances of Neuro-anaesthesia

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Highlights

I. History of Neuroanaesthesia.

II. Neurophysiology review.

III. Effects of Anaesthetic agents on brain.

IV. Clinical aspects of Neuroanaesthesia.

V. Emergence & Post operative care.

VI. Prognosis factors.
I. History of Neuroanaesthesia

- Surgical necessity has yielded the development of anesthesia and advances in anesthesia have provided further development in surgery. This is also true for neuro anesthesia and neurosurgery.

- The earliest neurological surgery record is in the Edwin Smith Papyrus. Trephination (burr holes), done in BC 3000-2500.

- In the middle age anaesthesia was provided with “Narcosis“, by cannabis indica, henbane, opium, wine, and mandrake.
History

- On October 16, 1846: First neuroanesthesia was under the influence of ether anesthesia in the “Ether Dome” amphitheater at the Massachusetts General Hospital by William T. G. Morton for removal of a head and neck tumor by Dr. John C. Warren from a patient, Edward G. Abbott.

- In the late 19th and early 20th centuries, neuroanaesthesia develop as a subspecialty due to interest of surgeons:
  
  William Macewen & Victor Horsley
  in Great Britain.
  Fedor Krause in Germany.
  Harvey Cushing in USA.
History

- (1949) **Albert Faulconer** (father of neuroanesthesiology). With neurologist, **Reginald Bickford**, began work on the electroencephalogram (EEG) responses to anesthetics.

- (1961) **John D. “Jack” Michenfelder, MD** (father of modern neuroanesthesiology) discover profound hypothermia to facilitate the clipping of cerebral aneurysms.
Albert Faulconer, MD,
John D. “Jack” Michenfelder, MD
II. Neurophysiology review

Six interrelated components of neurophysiology that are important to the practice of neuroanesthesia:

1. Cerebral perfusion pressure (CPP).
2. Cerebral blood flow (CBF).
3. Cerebral blood volume (CBV).
4. Intracranial pressure (ICP).
5. CO2 responsiveness (CO2R).
6. Cerebral metabolic rate of Oxygen (CMRO2).
1. Cerebral perfusion pressure (CPP).

- CPP is the net pressure gradient causing cerebral blood flow to the brain (brain perfusion).
- CPP = MAP – ICP.
- Normally CPP is 80-100 mm Hg.
- When CVP is significantly greater than ICP, CPP = MAP - CVP
- CPP determines brain blood perfusion when ICP is elevated, otherwise ABP is main determinant.
- Too low CPP - ischaemia, too high - hyperaemia
2. Cerebral blood flow (CBF).

- CBF Cerebral blood flow (CBF) is the blood supply to the brain in a given period of time to meet brain's metabolic demands.
- CBF = CPP / CVR
- 15-20% of cardiac output.
- Total average CBF is 750ml/min.
- Gray matter has a higher flow than white matter.
- CBF is autoregulated – remains constant over a wide range of MAPs (50 – 150 mmHg).
Cerebral Auto regulation
3. Cerebral blood volume (CBV).

- **Cerebral blood volume** (or **CBV**) refers to the volume of blood that is present at a given moment within the **neocranium** in order to **perfuse** the **brain** and **meninges**.

- 15% of CBV is in the **arterial tree**.

- 40% in **veins** and

- 45% in **nervous tissue** and **capillaries**.
4. Intracranial pressure (ICP)

The pressure that is exerted on to the brain tissue by cerebrospinal fluid (CSF) and blood.

Normal ICP:

- Adults: 10-15 mm Hg/135-200 mm of water.
- Children: 3-7 mm Hg.
- Infants: 1.5-6 mm Hg.
- Neonates: < 2 mm Hg.
MONRO-KELLIE-DOCTRINE

The cranial vault is a fixed space consisting of 3 compartments:

- Parenchyma (neurons and neuroglial tissue) - 80%.
- CSF - 10%.
- Blood - 10%

Therefore, expansion of one compartment results in a compensatory decrease in another in order to maintain ICP.
5. CO2 responsiveness (CO2R).

- CBF is directly proportionate to Paco2 between 20-80 mm hg.
- CBF changes approximately 1-2 ml/100g/min/mm hg changes in Paco2.
- CO2R of the cerebral arterial tree is important in that hypercarbia results in vasodilation and increased CBV.
- Hyperventilation (hypocarbia) causes cerebral arterial vasoconstriction, decreased CBF & CBV and a decreased ICP.
6. Cerebral metabolic rate of Oxygen (CMRO2).

- Normal CMRO2 is in averages \(3-3.8\text{ml/100g/min} \).
- Average \(50\text{ml/min} \) in adults.
- Greater in gray matter than in white matter.
- Because of the relatively high oxygen consumption and absence of significant oxygen reserves, interruption of cerebral perfusion usually results in unconsciousness within 10 s as oxygen tension drops below 30 mm hg.
III. Effects of anaesthetic agents on the brain

- **Volatile anaesthetic agents**
  - Dilate cerebral vessels.
  - Increase CBF/ICP.
  - Impair autoregulation in dose dependent manner.
  - Dose dependent decrease CMRO$_2$. 
Volatile anaesthetic agents

- Luxury perfusion.
- Increase CBF.
- Decrease CMRO₂.

Beneficial for

- Hypotensive Technique
- Global Ischaemia.

Circulatory Steal in Focal Ischaemia

- CBF increase in healthy areas.
- No increase in ischaemic areas.

Most studies shows all volatile agents are neuroprotective when used in MAC > 1.
# Effects of Volatile anaesthetic agents on CVS & CNS

<table>
<thead>
<tr>
<th>Effects</th>
<th>Halothane</th>
<th>Isoflurane</th>
<th>Sevoflurane</th>
<th>Desflurane</th>
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<tr>
<td><strong>Cardiovascular</strong></td>
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<tr>
<td>Blood pressure</td>
<td>↓↓↓</td>
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<tr>
<td>Heart rate</td>
<td>↓↓</td>
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<td>No change</td>
<td>No change or↑</td>
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<tr>
<td><strong>Cerebral</strong></td>
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<tr>
<td>BF</td>
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<td>ICP</td>
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<td>CMRO2</td>
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<td>BV</td>
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Nitrous oxide

- No change or modest increase in heart rate, blood pressure. Cardiac output.
- Increase CBF & ICP.
- May increase motor activity in CNS.
Iv induction agents:

Iv induction agents preserve brain’s ability to respond to CO₂ and autoregulation.

All Iv induction agents

- Decrease CMRO₂.
- Decrease CBF.

Ketamine exception

- Increase CBF/ICP

Reports of seizure with Etomidate.

So causation with epileptic patients.
Effects of IV anaesthetic agents on cerebral physiology

<table>
<thead>
<tr>
<th>Agents</th>
<th>CMRO₂</th>
<th>CBF</th>
<th>CBV</th>
<th>ICP</th>
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<tr>
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<td>Etomidate</td>
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Opioid

- Analgesia

- *HYPNOTIC SPARING EFFECT*”
  
  Potentiates the cardiovascular effects of hypnotics (so, with moderate dose of opioids, *reduce the dose of hypnotics*).

- Stable haemodynamics during induction, suction & skull pin application.

- Increased parasympathetic tone (large dose: *Bradycardia*).
**Opioid**

- Cerebral hemodynamics
  - Modest reduction in CBF & CMRO₂
  - No effect on ICP (if MAP is maintained)
  - Autoregulation: maintained
  - Cerebral vasoreactivity to CO₂ maintained
  - EEG changes depend on dose (small dose: minimal change)

- Nausea & vomiting: rare after propofol-remifentanil anesthesia.

- Impaired gastric emptying causing constipation & Ileus.

- Large intra-op dose: *delayed emergence*
- **NDMRs**
- No direct effects on CBF, CMRO₂ OR ICP
- Avoid:
  - Pancuronium
    - Increases HR → HTN → increases ICP
- Patients with anticonvulsant therapy may require increased dose due to enzyme induction.
**Succinylcholine**

- Succinylcholine increases ICP transiently. But little effects if given with propofol as drugs oppose one another.
- If difficult airways is anticipated use defasciculating dose of NDMR before the dose of succinylecholine.
Dexmedetomidine

- Adrenergic receptor agonist
  - Sedative & anxiolytic.
  - Analgesic.
  - Maintain respiration.
Several studies demonstrate benefit with craniotomy
  - It Blunts, increase SBP when used preoperatively.
  - Decrease narcotic requirements.
  - Decrease CBF thus ICP.
  - Causes cerebral vasoconstriction.
  - Does not affect brain CMRO2.
**Adenosine**

- A Purine nucleosides composed of an adenine, a ribose sugar molecule & a glycosidic bond.

**Official indication**

- Treatment of paroxysmal Supraventricular Tachycardia
- Aid in the diagnosis of broad or Narrow complex supraventricular tachycardia.
- Quick onset and short duration of action.

**Contraindications**

- II or III---degree AV block or sick sinus syndrome or symptomatic bradycardia (except in patients with a functioning artificial pacemaker).
- Asthma, other obstructive pulmonary disease.
Adenosine

Use in Surgical field

- Coronary artery bypass grafting and deployment of stent-grafts of thoracic and abdominal aorta.

- AVM and basilar artery aneurysm surgery.

- SAH and non-ruptered aneurysms surgery.

- In Sudden intraoperative rupture of an aneurysm.
IV. Clinical aspect of Neuroanaesthesia

A service that provide anaesthesia to patients undergoing neurosurgery, a subspecialty that combines the rapidly advancing basic and clinical neuroscience knowledge with the knowledge of anaesthesia to improve the outcomes of neurological patients.
Aims of neuroanesthesia

- **The primary goal:**
  - to ensure adequate perfusion and oxygenation of the brain

- **The secondary goal:**
  - To provide optimal operative conditions for neurosurgeon to operate (brain relaxation=slack brain).
  - to minimize interference with Electrophysiological monitoring
  - Rapid & high quality recovery.
Check list.

1. Type of surgery, anatomy effected.

2. Tightness of intracranial space.
   - Method of anesthesia, Osmotherapy.

3. Positioning.


5. Risk of ischemia.

6. Neurophysiological monitoring

7. Postoperative care.
Craniotomy, no signs of high ICP, nonruptured aneurysm, low grade SAH

- **Induction:**
  - Fentanyl 1-2 mcg/kg.
  - Thiopental or Propofol in sedative dose.
  - NDMR, moderate hyperventilation and intubation.
  - 20-40ug fentanyl / .1-.3mg remifentanil before head holder pins.
  - Maintenance of stable hemodynamic.
  - Invasive blood pressure measurement.
  - Urinary catheterization as needed.
  - CVP catheterization if need.
Maintenance:

- O₂ in nitrous oxide with Sevo-/Isoflurane in up to < 1 MAC along with Propofol infusion.
- Fentanyl boluses or Remifentanil infusion.
- NDMR as incremental doses/infusion.
- Hyperventilate to PaCO₂ of 25-30mm of Hg.
- No PEEP unless needed.
- IVF with 0.9% NaCl; avoid dextrose containing and hypoosmolar solutions.
- Can use hetastarch and albumin if needed.
- Osmotic diuretics and/or loop diuretics before opening of duramatter.
- Anti-convulsant & steroid therapy according to need of the surgery.
**Craniotomy with signs of high ICP or tight brain during surgery**

- **Induction** As before
- **Maintenance:**
  - High ICP
    - Propofol infusion,
    - Oxygen in air,
  - Volatile agents MAC >1
    - Remifentanil infusion or fentanyl boluses.
    - NDMR infusion or in increment.
Craniotomy with signs of high ICP or tight brain during surgery

Tight brain:

- Discontinue all inhaled anesthetics,
- Propofol infusion,
- FiO2 100%.
- Fentanyl bolus or Remifentanil infusion.
- Hyperventilation,
- Osmotherapy,
- Good neuromuscular block,
- Bolus of thiopental.
Good cooperation between neurosurgeon and anesthesiologist is essential.

- large iv-canula in cubital vein.
- Adenosine 5mg/ml readily Available on anesthesia table (saline for flushing).
- Temporary external cardiac pacing available.
- Fluid bolus (no hypovolemia) & SBP should be at the preoperative level.
- FiO2 up.
- Administration of bolus Adenosine when told by neurosurgeon.
- Anesthesiologist informs when asystole occurs and blood pressure development after that.
- Dose can be repeated only when well recovered from the initial dose.
Adenosine 2 doses:
35 mg -> asystole 20 seconds
40 mg -> asystole 30 seconds
Setting of monitoring

**Routine:**

- Continuous electrocardiography.
- Pulse oximetry.
- End-tidal capnography.
- Invasive/noninvasive blood pressure.
- Temperature.
- Urine output.
**Specialized.**

- Central venous pressure:
  1. For medical reasons.
  2. If large blood loss is expected e.g. meningioma.
- Transoesophageal echocardiography.
- Somatosensory/motor evoked potential:
  Brainstem/Spinal surgery.
- Facial nerve monitoring:
  Acoustic neuroma surgery.
- Electroencephalography monitoring:
  Epilepsy surgery.
- Bispectral analysis.
- Intracranial pressure monitoring.
BIS MACHIN
V. Emergence & Postoperative Care

**Emergence:**

- The goal of emergence is to maintain stable respiratory and cardiovascular parameters & preventing adverse CNS effects.
- A delayed emergence with deferred extubation in the ICU may achieve better thermal and cardiovascular stability after major neurosurgical procedures (thereby limiting secondary insults).
- But the diagnosis of complications relies on rapid neurological examination after early awakening and an awake patient is the best and the cheapest neuromonitoring available.
Emergence:

- During emergence for successful extubation, the patient should be:
  1) awake,
  2) fully reversed from neuromuscular relaxation and spontaneously breathing,
  3) hemodynamically stable, and
  4) normothermic.
Postoperative care

The basic goals of postoperative neurosurgical care are:

- Provide smooth emergence from anesthesia.
- Optimize post-operative hemodynamic, volume, and electrolyte status.
- Optimize airway and respiratory status.
- Treat coagulopathic states and hemostatic disorders.
- Optimize management of post-operative complications.
- Have reliable and appropriate systemic and neuromonitoring tools.
Postoperative care

These goals depend on:

- What was status (medical and neurological) of the patient before surgery?
- What neurological disease is being treated?
- What other neurological disorders does the patient have?
- What position was the patient in during surgery?
- What procedure was performed (procedure specific and expected complications)?
- What happened during surgery, e.g. blood loss, vascular injury?
- What anesthetic technique was used?
v. Prognosis factors

- Tumor size and location.
- Treatment (perioperative care).
- Age at presentation,
- Karnofsky performance score (KPS) at presentation, (preoperative clinical condition).
- Histologic findings.
- Molecular genetic factors.
- Experience of the surgical team.
Neuro surgical OT
Thank you