Neurosurgical Anaesthesia
For the Training Anaesthetist

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Let's start..

Historical vignette: Do you know that the first recorded anaesthetic death happened in 1848?

The patient name was Hanna Greener, a 15 years old girl who died after chloroform administration for a toenail removal.....
Part 1- ANESTHETIC AGENTS AND BRAIN:

Major goals in neurosurgical anaesthesia are to provide adequate tissue perfusion to the brain and spinal cord so that regional metabolic demand is met and to provide adequate surgical conditions ("relaxed brain").

Anaesthetic drugs and techniques if used incorrectly can worsen the existing intracranial pathologic condition producing a new damage, otherwise if used properly may help to protect the brain.

For the neuro anaesthesia provider, the knowledge of the effects of anaesthetics and anaesthetic technique on cerebral circulation, metabolism, and intracranial pressure both in normal and pathologic conditions is mandatory.

PHYSIOLOGIC AND PHARMACOLOGIC CONSIDERATIONS IN RELATION TO NEUROSURGICAL ANESTHESIA.

The brain vessels diameter changes within seconds in response to the changes in neuronal activity that immediately influence metabolic demand. It seems like there are two types of local cerebral blood flow (ICBF) mod-
phasic response mediated by transient neuronal activity and the tonic activation by local astrocytes using both pathways different mediators.

Cerebral activation can result in a much greater increase in glucose consumption than O2 consumption. Phenomenon that can be explained by "the astrocyte - neuron lactate shuttle hypothesis", in which astrocytic activation by glutamate released from neurons stimulate glucose uptake into astrocytes; glucose is processed glycolytically, resulting in a release of lactate as an energy substrate for neurons.

Anaesthetics cause functional alterations in the SNC as well as metabolic changes.

In general, IV anaesthetics decrease CMR and CBF in parallel fashion, whereas most inhalational agents decrease CMR with an increase in CBF. At first sight, the coupling of CMR and CBF is maintained with IV agents and lost with inhalational anaesthetics.

Because the net effect of anaesthetics on CBF is a balance between their direct effects on cerebral vessels and indirect effects caused by CMR changes, it is probable that the coupling of CMR and CBF is maintained with anesthetics but is modified by direct effects of different agents on vascular tone.
BLOOD FLOW CHANGES IN RELATION TO CEREBRAL PREEFUSION PRESSURE (CPP) AND CO2

CPP and PaCO2 are the most important variables that influence CBF. Autoregulation is the physiologic maintenance of constant CBF over a wide range of CPP values.

\[
\text{CPP} = \text{MAP} - \text{ICP or CVP (whichever is the higher)}
\]

CO2 can produce marked changes in Cerebrovascular Resistance (CVR) and CBF. Over a range of PaCO2 values of 20 - 80 mmHg, for each 1 mmHg increase or decrease in PaCO2 there is a 2 - 4% increase or decrease in CBF. Changes in the extracellular H+ concentration, NO, prostanoids, cyclic nucleotides, intracellular Ca and K channel activity have been regarded as regulatory factors for cerebrovascular reactivity to CO2.

CHANGES IN CEREBRAL BLOOD FLOW AND INTRACRANIAL PRESSURE REGULATION IN PATHOLOGIC CONDITIONS

Brain tissue hypoxia, acidosis, and oedema are the main pathologic consequences of most brain disorders. Cerebral vasoparalysis occurs, and coupling be-
between blood flow and metabolism is impaired. Therefore auto-regulation and CO2 reactivity are also impaired. Strict blood pressure control and respiratory management are required.

In the event of focal cerebral ischemia, hypercapnia can dilate the vessels in the normal area but not in the damaged area, and consequently, blood flow may be shunted from the ischemic to the normal area (intracerebral steal). Conversely, hypocapnia can divert blood flow the normal area to the ischemic area (inverse intracerebral steal or the Robin Hood effect). Because of a lack of evidence indicating the possible deleterious effects of hyperventilation or its beneficial effect on outcome, hyperventilation can not be recommended in patients who have intracranial pathology.

Anaesthesia alters ICP through changes in cerebral blood volume (CBV). Although correlation between CBV and CBF does not always exist, the changes in CBV, in general, appear to be proportional to the changes in CBF. Therefore an increase in CBF causes an increase in CBV and, thus, in ICP.

ICP is also affected by: blood pressure, mechanical effects such as patient's position and respiratory pattern, muscle activity, and rate of production /absorption of cerebrospinal fluid.

EFFECTS OF SPECIFIC ANESTHETIC DRUGS AND OTHER DRUGS
In general, all inhalational anaesthetics are cerebral vasodilators and possesses the capability of increasing ICP.

**Isoflurane**: Most animals studies show that isoflurane produces and increase in CBF that is accompanied by a decrease in CVR and CMRO2, except at low concentration.

The net effect of inhalational anaesthetics on CBF is a balance between a reduction in CBF due to CMR suppression and augmentation of CBF due to direct cerebral vasodilation. It is important to remember that cerebral vasoconstriction in response to hypocapnoea is not abolished by any inhalational agent.

ICP have been reported to be lower in the patients anaesthetised with propofol-fentanyl than those anaesthetised with isoflurane-fentanyl, propofol may be preferable in the setting on unstable ICP.

Isoflurane exhibits a potent cerebral metabolic depressive effect, and many animal studies have demonstrated the neuroprotective properties of isoflurane within clinical relevant concentrations. However, the protection with isoflurane is applicable only to mild insults, being inferior to and less durable than mild hypothermia.

**IN SUMMARY**, isoflurane appears to produce a mild increase in CBF and a pro-
nounced decrease in cerebral metabolism, the cerebral protective effect of this agent it is not proven clinically. However, isoflurane may be a desirable anaesthetic for many neurosurgical procedures, including carotid endarterectomy.

Sevoflurane: The increase, if any, of ICP associated with sevoflurane can be blocked with hyperventilation. When Sevoflurane is compared with Isoflurane and Desflurane, the extent of the increase in ICP is in following order: desflurane > isoflurane > sevoflurane.

In patients undergoing trans-sphenoidal pituitary surgery (no mass effect), lumbar CSFP was increased with sevoflurane, but the mean increase was small and comparable to those reported with iso and des.

In patients with cerebral tumors (midline shift less than 10 mm) anaesthetised by either propofol-fentanyl, isoflurane-fentanyl or sevoflurane-fentanyl, the values of ICP was the order of propofol-fentanyl < isoflurane-fentanyl = sevoflurane-fentanyl. Therefore the ICP raising properties of a clinical dose of sevoflurane appear to be mild, but propofol would be preferable in a patient for whom ICP must be rigidly controlled.

We should have in mind that sevoflurane is a biodegradable agent and that its metabolites may be toxic in high concentrations. The compound A is neprotoxic and in prolonged in-
Interventions may increase the possibility of toxicity especially in patients with previous renal derangements.

*Desflurane*: Because of a low blood/gas partition coefficient (0.42) relative to other clinically used volatile inhalational agent, desflurane can provide rapid onset and offset of anaesthesia, which facilitates early neurologic evaluation. In general, desflurane decreases CMR, but CBF may either be increased or decreased depending on the dose used and if the patient is hypo or normo ventilated.

However, because desflurane may have slightly greater ICP-elevating effects than isoflurane or sevoflurane, desflurane should be used cautiously in patients with unstable ICP.

**IV AGENTS:**

*Ketamine*: Traditional teaching is based on the persisting dogma that Ketamine should not be used in patients at risk for increases ICP. Thirty eight years ago, increases in cerebral oxygen consumption, CBF and ICP were indeed reported during anaesthesia with this drug.
Advances in our knowledge of ketamine's cerebral effects and progress in therapeutic interventions for brain injury warrant a reevaluation of this verdict.

Let's then, after few considerations, attempt to answer the question whether use of ketamine in the neurosurgical patient is acceptable.

From the standpoint of cerebral hemodynamics, the available information demonstrates that Ketamine does not increase ICP in neurologically impaired patients during controlled ventilation and coadministration of a GABA receptor agonist. Ketamine has been considered unsuitable for neurosurgical patients because of its cataleptic actions and psychomimetic adverse effects. However, clinical studies did not report an increase in adverse reactions or emergence delirium when Ketamine and GABA-mimetics were used as compared with regimens without Ketamine.

Haemodynamics: Ketamine's stimulation of the CV system may prevent hypotension and thus maintain the CPP, which together with its other advantages over opiate-based sedation could make the drug a first choice in sedative regimens for patients with brain insults.

Neuroprotection: The renewed interest in Ketamine as a clinically available neuroprotectant has been fostered by a shift in thinking about neuroprotection by anaesthetics: from suppression of brain metabolism to inhibition of excitotoxic damage in the injury
cascade seems to be played by the unbalanced activation of NDMA receptors by toxic glutamate concentrations with subsequent cell death, this prompted therapeutic approaches with NMDA receptor antagonists, been Ketamine a good representative of a noncompetitive antagonism of those receptors.

It is also important to highlight that Ketamine has other important pharmacological effects like:

- Inhibition of tumour necrosis factor alpha (TNF-α) and Interleukin-6 (IL-6) gene expressions in lipopolysaccharide (LPS) activated macrophages. (Anti-inflammatory effect)

- Interaction with opioid, monoamine, cholinergic, purinergic and adenosine receptor systems as well as having local anaesthetic effect. (Antinociceptive and Antihyperalgesic effect)

This drug it is not recommended in children younger than 2 years old based on studies done in new born animals where neuroapoptosis have been shown to be induced.

**Propofol:** A non barbiturate hypnotic agent, consists of an oil in water emulsion of:

- Propofol 1%
- Soybean oil 100 mg/ml (10%). Maintains Propofol in a stabilised dispersed medium

- Egg phosphatide 12 mg/ml (1,2%). Emulsifier which stabilises small droplets and consists of phosphatidyl choline and ethan- nolamine from egg yolk lecithin. Both these compounds have lipophilic side chains that interact with the soybean oil. The polar head interacts with the aqueous phase.

- Glycerol 22,5 mg/ml. (Maintain isotonicity with blood)

Neuroprotection:

- Propofol decreases CMRO2, CBF, and ICP

- When a large dose is given, marked vasodilatation can lead to a significant drop in MAP and thus CPP.

- Normal cerebral reactivity to CO2 and autoregulation are main- tained during Propofol infusion

- Provides similar degrees of neuroprotection against focal isch- emia as thiopentone

- Its neuroprotective effects may be related to its action as a free radical scavenger and as an antioxidant via its effect on decreasing free radical induced lipid peroxidation.
- Can cause dose dependent EEG burst suppression. It has profound anticonvulsivant properties.

Dose:

Induction: 1 - 2.5 mg/Kg

Maintenance: 100 - 200 mcg / Kg / min or 4 - 10 mg / Kg / hr

Opioids: The mechanism of action of opioids involves the stimulation of μ, δ and κ receptors located centrally (brainstem, hypothalamus, limbic system, substantia gelatinosa of the spinal cord) and peripherically (GI tract, peripheral histamine receptors). Activation of opioid receptors leads to inhibition of voltage-gated Ca2+ channels and an increase of K+ efflux, causing a reduction in neuronal excitability due to membrane hyperpolarization. More broadly, opioids inhibit the transmission of painful stimuli from the afferent first neuron to the second neuron at the dorsal horn of the spinal cord, both by presynaptic and postsynaptic mechanisms.

Examples:

Fentanyl: Medium acting synthetic opioid with onset of action of 5 mins and duration of 45 mins (bolus). If used as a continuous infusion the duration of action will be 20 min after 1 hour infusion and 180 min after 4 hours infusion.
An elevation of ICP is described with the use of this agent and even if may be only transient, attention should be paid to this effect in patients with unstable ICP.

**Alfentanil:** Short acting opioid with an onset of action of 5 minutes and duration of action of 15 - 20 minutes. After 3 hours infusion, the duration will be 50 - 55 minutes. Comparatively, alfentanil has 1/10 of the potency of fentanyl, 1/3 its duration, and 4 times faster onset. It has little change or slight decrease in CBF and essentially has a minimal effect on ICP. (Dose: 10 mcg/ Kg boluses ) To prevent intubation response, a higher dose is needed (20 - 30 mcg / Kg )

**Remifentanil:** Principally a µ agonist, with relatively little binding to the other receptors. It retains all the pharmacodynamic characteristics of its class (analgesia, respiratory depression, muscle rigidity, nausea and vomiting and pruritus), but with a unique pharmacokinetic profile that combines a short onset and the fastest offset, independent of the infusion duration. It is an excellent choice for a cardiac stable anaesthesia with no risk of respiratory depression and excessive sedation after infusion is stopped. (Bolus dose: 0,5 - 1 mcg/Kg over 1 min and Infusion: 0,1 - 0,5 mcg/ Kg /min)

*After you read the Part 1, I think we should have a cup of coffee and then go to the following Part..*
Part 2: ANESTHESIA FOR SUPRATENTORIAL TUMORS:

For patients the problems associated with supratentorial tumours result from local and generalised pressure, whereas for surgeons the difficulties arise during surgical exposure because the brain is particularly susceptible to damage from retraction and mobilization.

In the mean time the anaesthesiologist is responsible for: maintenance intracerebral perfusion, to avoid secondary insults, control the effects of anesthesia on ICP, brain bulk, and tension perioperatively.

The above can be summarised as:

The anaesthetic goal: Preserve brain from secondary insult

Anaesthetic risk factors: Hypoxemia, Hypercapnia, anaemia, Hypotension

Anaesthetic actions: Conserve cerebral autoregulation and CO2
responsiveness. Maximise brain elastance to decrease retractor pressure

PATHOPHYSIOLOGY OF RISING INTRACRANIAL PRESSURE

The abnormally high intracranial pressure can be explained by the three components stated on the Monro - Kelly doctrine. The ability of these homeostatic mechanisms to compensate depends not only on the volume of the mass but also on the speed at which it arises. (Fig 1).

The intracranial volume effects of tumours are due not only to the mass of the tumour itself but also to the surrounding vasogenic brain oedema. Mostly seen around fast growing tumours with a good response to corticosteroid therapy but can persist or even rebound after surgery.

The Blood Brain Barrier is also affected by intracranial pathologic conditions. Normally the BBB is impermeable to a large or polar molecules and variably permeable to ions and small hydrophilic
non-electrolytes, so any disruption of the BBB will lead to vasogenic oedema proportional to the CPP.

Vasogenic oedema should be differentiated from osmotic oedema and cytotoxic oedema.

Blood osmolality is a critical determinant of cerebral oedema because a 19 mmHg pressure gradient across the BBB is generated for every mOsm.

Fig 1. Hyperbolic intracranial pressure - volume curve. (Light gray) High IC compliance: a change in volume produces minimal
change in pressure; (Dark gray) low IC compliance: a same change in volume as in high compliance area, produces a large change in ICP.

**INTRACEREBRAL PERFUSION AND CEREBRAL BLOOD FLOW**

CBF is regulated at the level of the cerebral arteriole. It depends on the CPP and the PaCO2 (Fig 2). CBF autoregulation, dominant to ICP homeostasis, keeps CBF constant in the face of changes in CPP or MAP.

Autoregulation is normally functional for CPP values of 50 - 150 mmHg and might be impaired by many intracranial or extracranial conditions.

Ischemia results at levels of CBF below 20 ml/100g/min unless CPP is restored (by increasing the MAP or decreasing the ICP) or cerebral metabolic demands are reduced (by deepened anaesthesia or hypothermia).
Fig 2. Cerebral Blood Flow (CBF) autoregulation: For a CPP value between 50 and 150 mmHg, CBF is maintained at 50 ml/100g/min. There is a linear relationship between PaCO2 (20-80 mmHg) and CBF. Hypoxemia increases CBF and hyperoxia decreases CBF.

GENERAL ANESTHETIC MANAGEMENT

Preoperative Assessment:

- Neurologic State of the Patient
- General state of the Patient
- Planned Intervention

- Neurologic State of the patient:
  a) How much is ICP raised?
b) The extent of impairment of intracranial compliance and autoregulation  

c) How much homeostatic reserve for ICP and CBF remains before brain ischemia and neurologic impairment occur?  

d) How much permanent and reversible neurologic damage is already present?  

Patients CT scan or MRI should be examined for the size and localization of the tumour and for signs of increased ICP as:

- Effacement of the lateral ventricle by a tumour mass.  
- Signs of obstructive hydrocephalus  
- Midline shift > 5mm  

Presence of such signs warns that the ICP - volume curve is close to decompensation (knee of the hyperbolic ICP - volume curve in Fig 1)  

- General state of the patient:  
  a) Cardiovascular function  
  b) Respiratory function
c) Coagulation disorders (malignant tumours)

d) Renal system: (Diuretics and electrolytes disturbances, diabetes insipidus)

e) Endocrine system: (Effects of the steroids on hyperglycemia and cerebral ischemia)

f) GI system: (Mucosal effects of the steroids and motility effects of increase ICP)

g) Hypercalcaemia: Bone metastasis

- Planned Intervention:

a) Position

b) Meningiomas!!!! generally in difficult locations, large size, desire of radical excision, significant blood loss!!!!!!

Anaesthetic Strategy:

a) Vascular access: a) Two large bore peripheral IV lines
b) CVL if indicated (substantial bleeding anticipated, major CV compromise is evident, vasoactive drugs to be used. I recommend the IJV under SONAR guidance and minimisation of head down position and neck rotation.

c) Arterial cannulation

b) Fluid Therapy: a) Aim for normovolaemia

b) Avoid hypo osmolar fluids

c) Avoid glucose containing solutions

Few words about Mannitol: Mannitol (C₆H₈(OH)₆) is a sugar alcohol. For clinical use we have a 20% sterile solution in 500 ml bag of water containing 100 g of Mannitol.

Mannitol solutions are acidic pH of 6.3 but preparations have sodium bicarbonate added for pH adjustment. Mannitol may crystallise if stored at room temp but can be made soluble again by warming the solution.

Because of its low molecular weight (182), is freely filtered through the renal tubules. However as it is not reabsorbed, it continues to be osmotically active in the tubules and this
accounts for its action as an osmotic diuretic. Mannitol also causes release of renal prostaglandins that lead to renal vasodilation and an increase in tubular urine flow. Also acts as a free radical scavenger and reduces the harmful effects of free radicals during ischemic reperfusion injury.

Side - effects of Mannitol:

- Fluid and electrolytes imbalance
- Metabolic Acidosis
- Heart Failure
- Pulmonary Congestion
- Hypovolaemia
- Hypotension
- Thrombophlebitis
- Skin necrosis (extravasation)
- Anaphylaxis
- Rebound increases of ICP
Raised ICP: Mannitol exerts its ICP lowering effects via two mechanisms: immediate effect due to plasma expansion and slightly delayed effect related to its osmotic action.

The early plasma expansion reduces blood viscosity and this in turn improves regional cerebral microvascular flow and oxygenation. It also increases IV volume and therefore CO.

Together, these effects result in an increase of regional CBF and compensatory vasoconstriction in brain regions where autoregulation is intact, resulting in a reduction in ICP. Mannitol also establishes an osmotic gradient between plasma and brain cells, drawing water from the cerebral extracellular space into the vasculature, therefore reducing cerebral oedema. An intact BBB is a prerequisite for Mannitol's osmotic action and cerebral oedema may be worsened by mannitol administration if the BBB is disrupted.

Management protocols vary from unit to unit...

The ICP effect of Mannitol is dose dependent. Current guidance recommends that 0.25 - 1.0 g / Kg mannitol should be given by IV infusion over 20 - 30 min ( recent publications highlight better outcomes with boluses than with infusions…. in traumatic Brain Injury (TBI) )

The recommended strategy by the textbook of Neuroanaesthesia (Cotrell - 2010) is to split the total dose (0.5 g/Kg) in more rapid pre craniotomy dose and slower infusion, until brain
dissection is complete. The ICP effect is prompt, removes 90 ml of brain water at peak effect, and lasts for 2-3 hours.

SUGGESTED ANAESTHESIA INDUCTION AND MAINTENANCE SEQUENCE FOR INTRACRANIAL SURGERY

1. Adequate fluid loading (5 to 7 ml/Kg of NaCl 0.9%). EKG in place, pulse oximeter, NIBP or arterial cannula under local anaesthesia

2. Induction of GA: Fentanyl (1 to 2 mcg/Kg or Remifentanil 1mcg/Kg not less than 30 secs). Preoxygenation (using a TV based technique for 3 minutes, FiO2 0.8 and a fresh gas flow of 5L/min), Ketamine 0.3 mg/Kg CI, Propofol 1 mg/Kg bolus. NDMR: Rocuronium or Cisatracurium.

3. Local anesthesia, Remifentanil 0.5 - 1 mcg/Kg or labatalol 5 mgs to prevent CV response to skull pin head holder placement and skin incision.
4. Maintenance:

Propofol: 100 mcg/Kg/min + Remifentanil: 0,05-0.1 mcg/Kg/min

Ketamine: 0,3 mg/Kg/h + Remifentanil: 0,05 - 0.1 mcg/Kg/min + Sevofurane:

0,5 - 1,5%

Does the use of volatiles really matter??

NO!!!! it does not!

Elevated ICP caused by brain disease is associated with worsened outcomes, although this probably reflects the underlying severity of the disease. However, assuming that the drug-induced (usually small) increase in CBF and ICP is equally detrimental is illogical.

Why do volatiles agents increase in ICP?

They do so by increasing CBF and CBV (desflurane and sevoflurane might also increase CSF production) but that does not explain that an increase in tissue perfusion induced by volatile agent can induce ischemia even when CPP decreases? How can an increase in CBF produce a critical reduction in CBF?
Patients with critical ICP should be a concern, the preop assessment showing somnolence, stupor, or a recent alteration in level of consciousness will indeed indicate prudence with the use of these agents. But to conclude that we should no use volatiles in neurosurgical patients is neither rational nor logical.

CHEMICAL BRAIN RETRACTOR CONCEPT

- Mild hyperosmolarity:
  - NaCl 0,9% (304 mOsm/Kg) as a baseline infusion; give Mannitol 20% (1245 mOsm/Kg) 0,5 - 0,75 g/Kg as discussed above or Hypertonic Saline - 7,5% (2498 mOsm/Kg) 2 - 4 mls/Kg before bone flap removal.
- IV anesthetic (Propofol), adequate depth of anaesthesia
- Mild hyperventilation (PaCO2 not less than 30 mmHg, mild hyperoxygenation
- Mild control of Hypertension: MAP around 100 mmHg in order to decrease CBV and ICP
- Normovolaemia, no vasodilators
- Head up position, check no compression of the IJV
- Minimal PEEP

**EMERGENCE FROM ANAESTHESIA:**

Emergence from anaesthesia has respiratory, CV, metabolic-endocrine and neurologic consequences.

In the early Post operative period after elective craniotomy, autoregulation is often impaired, with 20\% of patients demonstrating raised ICP.

Extubation criteria must be strictly observed: respiratory drive and airway protection are likely to be impaired after brain surgery, and both hypercapnia and hypoxia carry the risk of causing additional systemic secondary brain damage.

Analgesia, prevention of hypothermia and shivering and timely tracheal extubation to avoid fighting of the patient against the tube are required to limit catecholamine release and haemodynamic changes.

**Neurosurgical Awakening:**
NS awakening should maintain:

a) Stable BP and thus CBF
b) Stable oxygenation and CO2 tension
c) Stable CMRO2
d) Normothermia

NS awakening should avoid:

a) Coughing
b) Airway overpressure during extubation
c) Patient-ventilator dyssynchrony

Early Emergence. Preconditions:

a) Normovolemia, normothermia
b) Normotension (MAP - 80 mmHg)
c) Normocapnia (PaCO2 - 35 mmHg)
Part 3: ANAESTHETIC MANAGEMENT OF CEREBRAL ANEURYSM SURGERY

PREOPERATIVE CONSIDERATIONS:

The main steps in preoperative evaluation are as follows:

a) Assessment of the patient's neurologic condition and clinical grading of the Sub Arachnoid Haemorrhage (SAH)
b) A review of the CT and angiogram
c) Monitoring ICP
d) Evaluation of other systemic functions potentially affected by SAH
e) Communication with the neurosurgeon regarding positioning and special monitoring requirements
f) Optimise the patient

SNC: To allow better assessment of surgical risk and prognosis a modified Hunt and Hess (HH) grading scale need to be applied.
These clinical grading schemes allow evaluation of operative risk, communication among physicians about patient's condition, and conduct of comparative studies of therapy on outcome.

Despite successful surgical treatment, delayed ischemic neurologic deficits resulting in permanent neurologic injury or death can occur in patients whom the complication vasospasm develops. CT findings are often graded according to Fisher's grading system (Table 2).

Table 1. Modified Hunt and Hess Clinical Grades for Patients with Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria</th>
<th>Mortality %</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>Unruptured aneurysm</td>
<td>0</td>
</tr>
<tr>
<td>0</td>
<td>Asymptomatic or minimal headache and slight nuchal rigidity</td>
<td>2-5</td>
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Table 2. Fisher Grades for CT findings in SAH

<table>
<thead>
<tr>
<th>Grade</th>
<th>CT Finding(s)</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>No Blood detected</td>
</tr>
<tr>
<td>2 (vertical layers &lt; 1 mm thick)</td>
<td>Diffuse thin layer of subarachnoid blood</td>
</tr>
</tbody>
</table>
Localized clot or thick layer of subarachnoid blood (vertical layers > 1 mm thick)

Intracerebral or intraventricular blood with diffuse or subarachnoid blood

Although the surgical mortality and morbidity vary with different institutions, patients in good preoperative condition (HH grades I - II) can be expected to do well; patients with grade V status have a high mortality and morbidity.

Regarding cerebral pathophysiology, the higher the clinical grade, the more likely the occurrence of vasospasm, elevated ICP, impairment cerebral autoregulation, a disordered cerebrovascular response to hypocapnia, cardiac arrhythmias and myocardial dysfunction. Patients with worse clinical grades have also a tendency to become hypovolemic and hyponatremic.

ICP: Correlates well with clinical grade. Normal in patients with grade I and II status but elevated in those with grade IV and V. However normal ICP does not necessarily imply normal intracranial compliance therefore it is important not to normalise the ICP too rapidly, because doing so may increase the transmural pressure...
(TMP) gradient across the aneurysm wall and cause further haemorrhage. A CPP value of 60 to 80 mmHg is a reasonable goal.

**SYSTEMIC EFFECTS**

**Intravascular volume status and Hyponatraemia:**

The intravascular volume status have found to be abnormally low in 36 to 100% of patients with SAH, and the level of hypovolemia correlates with the clinical grade.

Hypovolaemia has been observed to be associated with hyponatraemia in approximately 30 to 57% of cases of SAH. The aetiology of hyponatraemia is still a matter of debate, SIADH and the Cerebral Salt Wasting Syndrome are the most common causes.

Other significant electrolyte abnormalities are hypokalaemia and hypocalcaemia.

**Cardiac Effects:**

The effects of SAH on the myocardium can range from EKG alterations, leakage of cardiac troponins or wall motion abnormalities evident on echo.
EKG changes: Occurs in 40 to 100% of patients with SAH. Should be considered benign, associated with wall motion abnormalities or associated with actual myocardial injury.

Benign: - Sinus Brady
    - Sinus Tachy
    - AV dissociation
    - PVC
    - Nonspecific ST segment depression
    - T wave inversion
    - U wave

Wall motion abnormalities: - Symmetrical T wave inversion
    - Prolonged QT interval > 500 msec
    - ST segment elevation

Myocardial Injury: - Q wave
    - ST segment elevation
    - Elevated cardiac enzymes values

Myocardial Function: After SAH, damage to the myocardium can be indicated by an increase in circulating levels of cardiac Tro-
ponin I (cTi) found in up to 68% of patients. This elevation have been found to be associated with regional wall motion abnormalities and left ventricular dysfunction as well as hypotension, delayed cerebral ischemia from vasospasm, and death and disability at 90 days.

Predictors of ventricular dysfunction: - Female
    - Elevated cTi
    - HH grade III - V

Anaesthetic Implications: If prolonged QT interval, T wave abnormalities and Q wave are present, the patient should undergo prompt correction of electrolyte disturbances. The use of b blockers are not warranted.

Always have in mind the possibility of concurrent AMI then cardiac enzymes and echo evaluation should be obtained in suspicious cases.

Respiratory System:

Pulmonary oedema has been observed to accompany SAH in 8 to 28% of cases. Again is correlated closely with clinical grade.

Aspiration and hydrostatic pneumonia are other potential complications.
Concurrent medical treatments:

a) Diuretics: Fluids and Electrolytes disturbances

b) Anticonvulsivants: (Used more than 7 days) Phenytoin and Carbamazepine - Antagonise actions of NDMR leading to higher dose requirements and shortened duration of action. Lamotrigine and Levetiracetam effects have not been rigorously studied.

c) Antifibrinolytics: Higher incidence of vasospasm, hydrocephalus, venous thrombosis and pulmonary embolism

d) Calcium Channel Blockers (Nimodipine (PO) and Nicardipine (IV): Clinical experience suggests that these drugs do not present any difficulty for anaesthetic management but mild hypotension have been described with their use.

REBLEEDING AND VASOSPASM:

VASOSPASM: In patients who initially survive SAH, cerebral vasospasm causing ischemia or infarction remains an important cause of morbidity and mortality.
It has been established that the lower limit of CBF compatible with normal brain function is approximately 15 to 20 ml / 100g / min. Thus considerable reduction in CBF can occur from vasospasm without clinical symptoms.

Clinical Manifestations: Detectable by angiogram at 72 hours, incidence peaks 7 days and seldom seen after two weeks.

Clinical manifestations include: decrease level of consciousness, new onset of focal signs and mutism, the diagnosis is confirmed by angiography.

Treatment:

Pharmacologic:

a) Calcium channel blockers: Nimodpine (controversial)

b) MgSO4: (Concern about hypocalcaemia and arterial hypotension) 30 - 60 mg/Kg given as a bolus immediately after induction.

c) Statins: (Doubtful)

Non Pharmacologic:
a) Surgical: Recommended to be operated within 48 H after haemorrhage

b) Reducing ICP

c) Triple H treatment: (Hypervolaemia, Hypertension, Haemodilution) Therapy that is most successful if instituted early, however prophylactic treatment initiated before aneurysm clipping is associated with a significant risk of rebleeding. Hypervolaemia is described to be achieved with albumin 5% or crystalloids. Starches and Dextran are not recommended because the potential complication of coagulopathy through interference with platelets and factor VIII.

Anaesthetic Considerations: Although it is not possible to predict the occurrence of post operative vasospasm, patients with good SAH grades and low Fisher grade tend to have a lower incidence. These patients should be kept in a normovolaemic state, with volume loading initiated toward the end of the operation, after the aneurysm has been clipped. Intraoperative hypotension can be provided safely, if so requested by the surgeon.

Treatment of postoperative hypertension should not be treated too aggressive.
INTRAOPERATIVE CONSIDERATIONS AND INDUCTION OF ANAESTHESIA

The goal during induction of anaesthesia for aneurysm surgery is to reduce the risk of aneurysm rupture by minimising the TMP while simultaneously maintaining an adequate CPP. Both TMP and CPP are determined are determined by the same equation (MAP - ICP). Therefore these goals represent opposite objectives.

As a general principle, the patient's blood pressure should be reduced to 20 - 25% below the baseline value, and prophylaxis for the normal hypertensive response to intubation should be instituted before tracheal intubation is attempted.

It is important to have in consideration the patient's clinical grade. Patients with SAH grades 0, I and II generally have normal ICP and are not experiencing acute ischemia. Therefore patients will tolerate a bigger transient decrease in BP (30 - 35%, or systolic BP at about 100 mmHg).

In contrast, patients with poor clinical grades frequently have increase ICP, low CPP and ischemia. The elevated ICP decreases the TMP and partially protects the aneurysm from rerupture. These patients may not tolerate transient hypotension as well, and the duration and magnitude of BP decrease should be moderate.
The same consideration applies to the use of hyperventilation. Patients with good clinical grade should not be hyperventilated, because the reduction in CBF will lead to a reduction in ICP and, consequently, an increase in TMP. Conversely, patients with poor clinical grades should be managed with moderate hyperventilation to improve cerebral perfusion. To reduce the risk of aneurysm rupture or ischemia, the change in TMP or CPP should always be gradual, not abrupt.

Conceptually, it is convenient to think of the induction phase consisting in two parts:

a) Induction to achieve loss of consciousness

b) Prophylaxis to prevent a rise in BP in response to laryngoscopy and intubation.

INDUCTION AND MAINTENANCE SEQUENCE:

Same as described in Part 2

BRAIN RELAXATION:
Same as described in Part 2 under the heading CHEMICAL BRAIN RETRACTOR CONCEPT

Just to add that in case brain relaxation may remain unsatisfactory and refractory to the regimen just described, the practitioner should:

a) Make sure that there is no hypoxaemia or systemic hypertension

b) Check the patient’s neck to rule out venous obstruction

c) After communication with the surgeon, implement a head up tilt to facilitate venous blood and CSF drainage

d) Rule out any possibility of intracerebral haematoma.

INTRAOPERATIVE ANEURYSM RUPTURE:

Management of aneurysm rupture during surgery partially depends on the ability to maintain the blood volume during the rupture. The keys to anaesthetic management are good communication with the surgeon and close monitoring of the patient’s vital signs as well as the surgical conditions.

If temporary occlusion is not planned or not possible and blood loss is not significant, the MAP should be decreased
transiently to 50 mmHg or even lower to facilitate surgical control.

If temporary occlusion is the method of bleeding control, normotension should be maintained to maximise collateral perfusion.

EMERGENCE FROM ANAESTHESIA:

Communication between the surgeon and the anaesthesiologist is again essential for optimal management of the patient’s emergence from anaesthesia.

If the surgical procedure is uneventful, patients with preoperative SAH grade I or II should be allowed to awaken, and their tracheas may be extubated in the operating room.

Because hypertensive therapy is effective in reversing delayed cerebral ischemia from vasospasm, modest levels of post operative hypertension (<180 mmHg systolic) are not aggressively treated.

SAP > 200 mmHg should be treated with Labetalol (5 - 10 mg increments) until BP is controlled.

Regarding decision to extubate patients, it is better to err on the side of
A BIT OF PHYSIOLOGY:

Blood Flow: Spinal Cord Blood Flow (SCBF) has been studied extensively in animal models. Average SCBF is about 60 ml/100g/min, including a threefold to fourfold gray matter - white matter differential in blood flow. Autoregulation in the cord mimics that in the brain, with flow well maintained with MAP of 60 - 120 mmHg. Likewise, the effects of arterial blood gas tensions are similar to those in the brain; hypoxemia and hypercapnia cause vasodilatation and hypocapnia causes vasoconstriction (Fig 3).

Injury to the spinal cord disturbs autoregulation of blood flow. Trauma to the cord results in a decrease in SCBF and loss of autoregulatory function. The nature of the operative procedure itself may also have an effect on SCBF. This effect is well recognised with spinal distraction and instrumentation but may also occur during other operations, such as simple laminectomy.
Among the individuals suffering general traumatic injuries, the cervical spine is involved in 4.3% of cases, the thoracolumbar spine in 6.3% of cases and the spinal cord in 1.3%. Spinal injuries have a predilection for the more mobile areas of the spine, which include the cervical spine (75% at the level of C3-C7) and the thoracolumbar junction (16% at the level of L1 junction).

Traumatic spinal injuries most commonly occur after impact forces that result in the following seven basic types of spinal trauma:
hyperflexion, hyperextension, compression, rotation, shear, avulsion and a combination of these types

**NEUROLOGIC ASSESSMENT:**

**Table 3: American Spinal Injury Association (ASIA) Impairment Scale**

<table>
<thead>
<tr>
<th>ASIA Grade</th>
<th>Type of Injury</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Complete</td>
<td>No motor or sensory function</td>
</tr>
<tr>
<td>B</td>
<td>Incomplete</td>
<td>Sensory but not motor function preserved below the level of injury</td>
</tr>
</tbody>
</table>

C  Incomplete  Motor function is preserved, but majority of key muscles below the neurologic level have a muscle < 3

D  Incomplete  Motor function is preserved, but majority of key muscles below the neurologic level have a muscle > 3

E  Normal  Motor function and sensory function are normal
MEDICAL MANAGEMENT:

Pulmonary System:

Most abnormalities are the result of the adverse effects of SCI on pulmonary lung volumes and pulmonary mechanics.

The normal muscles of respiration are composed of the intercostal muscles (intercostal nerves originating from the thoracic spinal cord) and the diaphragm (supplied by cervical innervation originating from C3 to C5), this muscle being responsible for about 65% of the vital capacity.

Lesions above C3 produce nearly complete respiratory paralysis resulting in the patients inability to produce a tidal breath or to cough.

An interesting point is that tetraplegic patients will increase vital capacity in supine position due to the effect of gravity on the abdominal contents in the presence of paralysed abdominal musculature.
Level of Spinal Cord Injury and Corresponding Respiratory Function:

**Level above C3**: Paralysis of diaphragm and accessory muscles, resulting in apnea and lifelong ventilatory dependence.

**Level C3-C5**: Partial to complete diaphragmatic paralysis, paralysis of accessory muscles, marked reduction in lung volumes with hypoxemia, recurrent atelectasis and pneumonia, probably tracheostomy, mechanical ventilation dependent although they might be weaned.

**Level C5-C7**: Paralysis of accessory muscles, marked reduction in volumes with hypoxaemia, recurrent atelectasis and pneumonia.

**High thoracic**: Partial paralysis of accessory muscles, reduction in lung volumes with atelectasis, increase incidence of pneumonia.
Other pulmonary problems are pulmonary oedema, aspiration pneumonitis, coexisting blunt chest trauma. Ventilatory failure and aspiration might occur as early as 4-5 days after the injury.

Cardiovascular System:

SCI has profound effect on the CV system, the magnitude again depending on the level of the injury. Complete cervical SCI has the most pronounced physiologic effects: CV instability, dysrhythmias, and ventricular dysfunction, whereas SCI below T5 results in varying degrees of hypotension caused by the functional sympathectomy below the level of injury.

It is important to remember that bradycardia associated with cervical spine injury remains most problematic during the first 2 weeks after an acute cervical SCI and preventive measures to avoid severe bradycardia are encouraged: sedation, 100% before suctioning and limiting the time allowed for suctioning.

MAP is recommended to be kept ≥ 85 mmHg for the first 7 days following acute SCI. Because autoregulation is lost after SCI, aggressive use of fluids or vasoactive medications for the
correction of hypotension is crucial for optimal preservation of neurologic function and reduction of secondary injury.

Care should be taken in the administration of potent agonists, because substantial increases in cardiac afterload may impair CO and precipitate LV failure. An inotropic agent may be the agent of choice.

Gastrointestinal System:

Frequently seen are: ileus, gastric distention, peptic ulcer and pancreatitis.

Coagulation:

DVT has been reported to occur in 40 - 100% of patients with acute SCI. Pulmonary embolism is also common to see and is the third leading cause of death in such patients.

Muscular:

Proliferation of acetylcholine receptors and spasticity leading the patient to hyperkalaemia from succinylcholine.
Autonomic Hyperreflexia:

Occurs in 85% of patients with spinal cord transections above T5 in whom the splachnic outflow remains intact, it is secondary to autonomic vascular reflexes, which usually begin to appear about 2 to 3 weeks after injury.

Afferent impulses originating from bladder or bowel distention, childbirth, manipulation of the urinary tract, or surgical stimulation are transmitted along the pelvic, pudendal, or hypogastric nerves to the isolated spinal cord and elicit a massive sympathetic response from the adrenal medulla and sympathetic nervous system, which is not longer modulated by the normal inhibitory impulses arising from the brainstem and hypothalamus.

Infections:

Leading causes of death in patients with SCI, include pneumonia and urosepsis.

ANESTHETIC CONSIDERATIONS:
PRE OPERATIVE EVALUATION:

Should be based on the medical problems previously discussed.

AIRWAY MANAGEMENT FOR CERVICAL SPINE INJURY:

Should be based on the following approaches:

a) Awake fiberoptic intubation

b) Video laryngoscope (CMAC)

c) Direct laryngoscopy with in line stabilisation

d) Fiberoptic Intubation using LMA as conduit
   - LMA Classic
   - LMA Fastrach

e) Fiberoptic lighted stylets

f) Retrograde Intubation

g) Surgical AW.
Few Comments:

- Straight vs. curve blade: No difference in cervical spine movement

- Normal laryngoscopy: Extension at occipito-atlantal and C1-C2 articulations, C2 - C5 displaced only minimally.

- Manual in line stabilisation (MILI): Reduces neck mobility during intubation, recommended method of reducing neck mobility during tracheal intubation, head held in neutral position without axial traction, the anterior aspect of collar, if present, should be removed before laryngoscopy.

ANAESTHESIA INDUCTION AND MAINTENANCE:

Induction of anaesthesia for spinal surgery carries the same considerations as those for any other GAs. Concerns related to patient comorbidities should be addressed as appropriate.

Muscle relaxants should be used with consideration of the potential for a hyperkalaemic response in the instance of succinylcholine use.
It is important that every effort be expended to keep the head and spine in a neutral position during the positioning of a patient if any unstable fragment, spinal stenosis, severe root impingement by disc fragment, and preexisting neurologic deficit.

Maintenance: Technique described in Part 2 is applicable for SCI surgery.

Just to say that selecting the particular anaesthetic technique for operations on the traumatised spine with acute SCI is of less importance than optimising medical management during the procedure.

To date, no evidence has been presented that conclusively favours one anaesthetic agent or technique over another in a patient with acute SCI. However, maintenance of MAP at or above 85 mmHg and an adequate CO have been shown to prevent secondary injury to the spinal cord.

**MONITORING AND POSITIONING**

Same principles as any other surgery regarding indications and complications are applied.
Haemodynamic monitoring is recommended for frequent determination of blood pressure, ABG, Hb levels and blood glucose.

Remember that in Prone position, the tongue of the patient might protrude outside the mouth compromising the venous return, therefore a severe iatrogenic oedema might be formed with subsequent airway compromise. A Guedel OP cannulae (Size 1 -white- or 1,5 -yellow-) should be used to prevent this complication.

POST OPERATIVE CONSIDERATIONS:

Cervical Spine Surgery extubation:

Patients with prolonged procedures (5 hours), exposing more than three vertebral levels that include C2, C3, or C4 and with more than 300 ml blood loss, should remain intubated or should extubated over an airway exchange catheter and watched carefully for respiratory insufficiency.

Common complications to look for:

- Neurologic deficits
- Post operative visual loss.

Neuroanesthesia is as challenging as fascinating, the suppression and control of central integrative mechanisms of pain acting on the same unique and extreme vulnerable structures where the surgeon at the same time works, requires a profound clinical, physiological and pharmacological knowledge.

I hope this succinct review will help you to achieve some of the above mentioned standards