Anesthesia for neurosurgery (Part II)

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Abstract
The central nervous system (CNS) deserves special consideration in the perioperative setting for several reasons for an anesthetist. An understanding of neuroanatomy is essential because neuro anesthesia continues to develop and evolution of neurosurgical practice is accompanied by new challenges for the anesthetist. Basic knowledge and expertise of the neuro-anesthetist can directly influence patient outcome. With the recent advancement in functional and minimally invasive procedures, there is an increased emphasis on the provision of optimal operative conditions, preservation of neurocognitive function, minimizing interference with electrophysiological monitoring, and a rapid, high-quality recovery. So, during neuro anesthesia, anesthesiologist needs to know physiology of CNS including cerebral metabolism and cerebral blood flow. Neuro anesthesia can be challenging, because sometimes apparently contradictory demands must be managed, for example, achieving optimal conditions for neurophysiological monitoring while maintaining sufficient anesthetic depth, or maintaining oxygen delivery to neuronal tissue and simultaneously preventing high blood pressures that might induce local bleeding. One of the peculiarities of neuro anesthesia has always been that as much importance is attached to wakening the patient as sending them to sleep. We have included the anesthesia for neurosurgery in two parts. In the first part, we had discussed cerebral anatomy, physiology and intracranial pressure, in the second part we have included anesthesia considerations for surgery for brain tumor, aneurysmal surgery, temporal lobe surgery, trans sphenoidal surgery and traumatic brain injury.

Keywords: Anesthesia, Neurosurgery.

Introduction
The central nervous system (CNS) deserves special consideration in the perioperative setting for several reasons for an anesthetist. An understanding of neuroanatomy is essential because neuro anesthesia continues to develop and evolution of neurosurgical practice is accompanied by new challenges for the anesthetist.[1] Basic knowledge and expertise of the neuro-anesthetist can directly influence patient outcome. With the recent advancement in functional and minimally invasive procedures, there is an increased emphasis on the provision of optimal operative conditions, preservation of neurocognitive function, minimizing interference with electrophysiological monitoring, and a rapid, high-quality recovery.[2,3] So, during neuro anesthesia, anesthesiologist needs to know physiology of CNS including cerebral metabolism and cerebral blood flow. Neuro anesthesia can be challenging, because sometimes apparently contradictory demands has to be managed, for example, achieving optimal conditions for neurophysiological monitoring while maintaining sufficient anesthetic depth, or maintaining oxygen delivery to neuronal tissue and simultaneously preventing high blood pressures that might induce local bleeding.[2] One of the peculiarities of neuro anesthesia has always been that as much importance is attached to wakening the patient as sending them to sleep. We have reviewed the anesthesia for neurosurgery in two parts. In the first part (Anesthesia for Neurosurgery (Part I) (DOI: 10.18231/2394-4994.2018.0001), we had discussed cerebral anatomy, physiology and intracranial pressure, in the current second part we have included anesthesia considerations for surgery for brain tumor, aneurysmal surgery, temporal lobe surgery, trans sphenoidal surgery and traumatic brain injury.

Common Surgical Procedures
Neurosurgery is a complex surgery because every part of the brain undergoing surgery needs some special consideration apart from general principles of management.

Surgery for Brain Tumors[1-3] (Table 1)

1. The fundamental anesthetic considerations in tumor surgery are proper positioning of the patient to facilitate the surgical approach; providing adequate relaxation of the brain to optimize surgical conditions; and preventing well-known devastating complications, such as venous air embolism.
2. Preoperative assessment of the level of consciousness (Glasgow Coma Scale, GCS) (Table-2) and a review of relevant radiologic studies should be performed, and the results should be taken into consideration in the anesthetic plan.
3. Adequate brain relaxation is typically achieved with a standard anesthetic, including sub-MAC volatile anesthesia, an opioid infusion, mild to moderate hyperventilation, and Mannitol.

Table 1: Management strategies

Protocol	Management Strategy
Preoperative	1. Note and record the GCS of patient 2. Avoid sedatives / anxiolytics if patient is drowsy or ICP is raised
Positioning and Monitoring	Sitting: 1. Standard ASA monitoring, CVP line and Precordial Doppler if possible(for VAE) 2. Prone / semi recombinant: 3. Standard ASA monitoring, Foley catheter and arterial line if surgery demands.
Intraoperative	Induction: 1. Etomidate / Thiopentone or Propofol with intubation under deep anaesthesia to avoid increase in ICP. 2. Maintainace: 3. Minimize ICP and maintain adequate CPP. 4. Opioid and/or volatile anesthetic (isoflurane is preferred) with or without nitrous oxide. 5. Avoid intraoperative long acting muscle relaxants if motor function is tested. 6. Mannitol (0.25-1 g/kg IV) also can be given before opening of dura. 7. Maintain euvolemia and normocapnia if normal ICP; temporary hyperventilation for relaxing brain if required. tight
Postoperative	1. Avoid coughing, straining during extubation by prior use of lignocaine or small dose of opioid. 2. Always note and Record postoperative GCS

Glasgow Coma Scale (GCS)[4,5]: GCS is a neurological scale which aims to give a reliable and objective way of recording the conscious state of a person for initial as well as subsequent assessment. The scale was published in 1974 by Graham Teasdale and Bryan J. Jennett, professors of neurosurgery at the University of Glasgow's Institute of Neurological Sciences at the city's Southern General Hospital. It has following point scale system:

Table 2: Glasgow coma scale

	1	2	3	4		
Eye (E)	Does not open eyes	Opens eyes to painful stimulus	Opens eyes to verbal commands	Opens eyes spontaneously		
Verbal(V)	Makes no sound	Makes incomprehensible sounds	Speaks incoherent words	Confused and disoriented	Oriented and talks normally	
Motor(M)	No movement	Extension to painful stimuli (decereberate posture)	Abnormal flexion to painful stimuli (decorticate posture)	Flexion / Withdrawal to painful stimuli	Localizes to painful stimuli	Obeys commands

The GCS for intubated patients is scored out of 10 as the verbal component falls away

Generally, brain injury is classified on the basis of GCS score (E+V+M) as:

1. Severe, GCS < 8–9 (In trauma, a GCS of 8 or less indicates a need for endotracheal intubation)
2. Moderate, GCS 9–12
3. Minor, GCS ≥ 13

Cerebral Aneurysm Surgery [2,6,7] (Table 3)

1. The intracranial aneurysm is the most common cause of intracranial hemorrhage. The most common manifestation is sudden onset of severe headache, with nausea and vomiting and focal neurological signs and altered level of sensorium or neck rigidity.
2. Surgical or endovascular intervention is required to prevent hemorrhage in patients who survive hemorrhage to prevent further re-bleeding.
3. Patients with Sub arachnoid hemorrhage due to aneurysm are at risk of many complications which include cardiac dysfunction, neurogenic pulmonary edema, hydrocephalus as well as hemorrhage from the aneurysm.
4. Triple H therapy is the modality of treatment of vasospasm. Triple H therapy includes hypervolumia, hypertension and hemodilution. Nimodipine, a type of calcium channel blockade

decreases the overall morbidity and mortality risk from vasospasm.

5. The major causes of morbidity and mortality from SAH include rebleeding, ischemic cerebal injury, infarction and development of hydrocephalus and seizures.

Temporary clipping time should be less than 10 minutes to avoid risk of ischemia. Effect of hypothermia (32-25○ C) is confounded by various factors which include grade of SAH, time of clip application, neuroprotective agents, anesthetic agents, surgeon experience and intraoperative repair of aneurysm.

Table 3

Protocol	Management Strategy
Preoperative	1. Note and record the GCS of patient 2. History of hypertension, CAD, DM 3. Patient is often on calcium channel blockers. 4. ECG changes may mimic with cardiac event (T-wave inversions, U waves, ST-segment depressions, prolonged QT interval, and rarely Q waves) 5. No sedation (may raise PCO2)
Positioning and Monitoring	1. Supine: the best surgical exposure is related to specific head positions. The proper angle of microscopic view may minimize neurovascular injury and brain retraction. 2. Standard ASA monitoring, Foley catheter and arterial line if surgery demands (HT/CAD).
Intraoperative	Induction: 1. Opioid plus propofol and/or volatile anesthetic. 2. Avoid increases in systemic blood pressure (to prevent rebleeding). 3. Maintain cerebral perfusion pressure to avoid further ischemia (to prevent vasospasm). Maintenance 1. Maintain normal to increased systemic blood pressure to avoid ischemia during surgical retraction and temporary clipping. 2. Maintain normocapnia and avoid unnecessary hyperventilation. 3. Mannitol (0.25-1 g/kg IV) also can be given.
Postoperative	1. Maintain slight head-up position or as required by surgeons. 2. Maintain normal to increased systemic blood pressure to prevent further vasospasm.(HHH therapy is given as needed) 3. Early awakening is recommended to facilitate neurologic assessment.

Trans-sphenoidal Surgery [1,8,9] (Table 4)

1. Transphenoidal surgery is done for pituitary adenoma. Anesthesia is challenging as it takes into considerations of endocrine abnormalities associated with disease process. Trans nasal Trans sphenoidal (TTNS) excision has benefits in terms of recovery and reduced mortality.
2. Pituitary tumors are more commonly associated with acromegaly, gigantism and Cushing disease. These patients have also associated diabetes mellitus, hypertension and obstructive sleep apnea. They also have airway abnormalities owing to presence of acromegaly.
3. Intra-operative anesthesia management is based on size of tumour and associated co-morbidities, and physiological disturbances. Dexmeditomidine and remifentanil are newer and excellent drugs used for maintenance of hemodynamic stability. Post-operative management includes strict monitoring, and looking for complications such as diabetes insipidus and CSF leak.
4. Anesthesia management includes perioperative hemodynamic stability, maintenance of normal intracranial pressure, smooth surgical conditions such as lax brain, maintenance of adequate cerebral blood flow, rapid recovery, and emergence at completion of surgery.
5. Gluco-corticois are supplemented in patients with hypo-pituitarism on evening before surgery with 100 mg hydrocortisone, and is repeated just before or at the beginning of surgery and third dose is given on evening after the operation.

Table 4

Protocol	Management Strategy
Preoperative	1. Cardiac abnormalities: LVH, CAD, arrhythmias, conduction disturbances, and CHF are commonly associated with hyper-secretion from pituitary, acromegaly and Cushing's disease. (The role of ECG and ECHO is of prime importance). 2. Possibilities of raised ICP and hydrocephalous. 3. Obesity and associated sleep apnea syndrome. 4. Visual field defects are common. 5. Significant incidence of DM and thyroid disorders. 6. Antihypertensive, antianginals, antiarrhythmics, antacids, etc. should be administered as per scheduled protocol on day of surgery. 7. No sedative should be prescribed.
Positioning and Monitoring	1. Head up position: more risk of VAE. 2. Standard ASA monitoring, plus arterial line, Large IV cannula.
Intraoperative	Induction: 1. Large mask required, mask ventilation may be difficult. 2. Oral intubation, consider awake FOB if likely to be very difficult. Flexometallic tube plus throat pack. 3. Standard induction with Thiopentone / propofol with inhalation agents (MAC<1) is preferred. 4. Proper positioning (obese patient) to prevent pressure induced nerve injury (esp. Ulnar nerve) Maintainance: 1. Balanced technique as used in neuro anaesthesia 2. Induced hypotension: MAP above 65 mmHg 3. Vigilance for complications 4. Disconnection 5. VAE 6. Dissection into cavernous sinus with haemorrhage 7. Pressure on face or eyes
Postoperative	Antiemetic 1. Emergence 2. Look for Clear blood or CSF from airway 3. Aim to minimize coughing Postoperative 1. Attention to complications 2. Diabetes insipidus (usually transient) 3. Pan hypopituitarism Analgesia: Oral ± IM/ IV narcotics Nasal CPAP is contraindicated after TNTS surgery (risk of Tension Pneumothorax).

Awake Craniotomy (Temporal lobe surgery) [1,10,11,12] (Table 5)

1. Awake craniotomy refers to some intracranial neurosurgical procedures in which patients are sedated and pain free yet are able to respond to verbal or visual commands. This is preferred to facilitate monitoring of regions of brain on which surgeon is operating.
2. Some of the relative contraindications of this procedure include difficult airway, obstructed sleep apnea, or orthopnea. Patients who suffer from severe anxiety, claustrophobia or other psychiatric disorders may be inappropriate to undergo awake craniotomy
3. Anesthesia approach depends on length of surgery, patient factors, surgeon and there is no consensus on the best approach for awake craniotomy.

Table 5

Protocol	Management Strategy
Preoperative	1. Detailed history is taken, especially taking in detail history of epilepsy, nature of aura etc, and detailed medication history is also taken in account. 2. Investigation Wada test 1. Unilateral carotid injection of sodium amobarbital (a barbiturate). 2. Determines lateralization of speech, short term memory (establishes cerebral language and Memory representation of either hemisphere).

	Videotelemetry 1. Continuous EEG with subdural, parenchymal or foramen ovale electrodes to localize focus of Seizures. Premedication: 1. Anticonvulsant agents avoided
Positioning and Monitoring	Head up position: 1. The patient position is dictated by the location of the lesion. This is usually a lateral or supine position, but with occipital lesions and testing the visual cortex, a sitting position may be used. 2. In any position, it is important that when the patient is fully awake during mapping that they are able to see and communicate with the anaesthetist or neuropsychologist. 3. Sterile drapes must not cover patients face (during communication.) Monitoring: 1. Routine ASA monitoring 2. Gas analysis to confirm airway patency (optional) 3. Continuous neurological assessment 4. Bispectral index(BIS) monitoring 5. Careful attention to patient comfort and warming
Intraoperative	Conscious Sedation Technique("awake throughout"): 1. Propofol is one of the most frequently used drugs either alone or in combination with remifentanil. 2. Droperidol 2.5-7.5 mg plus a narcotic like Alfentanil 5-10µg/kg plus 0.25-0.5µg/kg/min, or Fentanyl 0.7 µg/kg plus 0.7µg/kg/h. 3. Dexmedetomidine may be the ideal sedative (minimal respiratory depression) for awake procedures (A loading dose of 0.5–1.0 µg/kg1 over 20 min is then followed by an infusion rate of 0.2–0.7 µg /kg/ h). "Asleep–Awake–Asleep" Technique. 1. This is often the preferred method for epilepsy surgery (general anesthesia for the initial craniotomy he anaesthetic drugs are either reduced or stopped and the airway device is removed, when the patient has regained upper airway reflexes and it is safe to do so. 2. Once resection of the lesion is complete, general anaesthesia can be re-introduced and with re-insertion of the airway device). 3. The anesthetic drugs used for this technique are varied, but often are the same as those used in the 'awake throughout' technique. General anesthesia Reserved for children, patients with continuous movement disorders, and patients with an increased ICP. If provoking agent is required for seizures, methohexitone 0.3 mg/kg. For seizure termination if necessary, thiopentone 1 mg/kg.
Postoperative	Antiemetic Postoperative attention to complications 1. Bleeding 2. Seizures 3. psychological disturbances Analgesia: Oral ± IM/ IV narcotics Scalp block at the site of insertion of Mayfield pins

Anesthesia for Traumatic Brain Injury [3,15-18] (TBI)

1. The term head injury is interchangeable with traumatic brain injury and brain injury and is defined as trauma to the head other than superficial injuries
2. Maintenance of airway and breathing is of paramount significance in critical care setting and more importantly for patients with head injury as brain is very sensitive to the effects of hypoxemia and hypercapnia
3. Maintenance of cerebral blood flow has to be adequate to avoid cerebral ischemia, thereby minimizing secondary injury to brain. Hypotension, hypoxia, raised ICP and anemia should be avoided.
4. There is 10% incidence of unstable cervical spine injury with TBI. Risk factors include GCS score less than 8 and motor vehicle accident. Thus manual in line stabilization should be maintained to avoid neurological damage
5. Patients with TBI should be orally intubated because of potential presence of basilar skull fracture, risk of which may be exacerbated with nasal intubation

6. All intravenous induction agents except ketamine cause a fall in CBF, CMRO2 and ICP. Etomidate causes a little change in blood pressure despite reducing CMRO2 and may be advantageous but may lead to adrenal insufficiency leading to delayed hypotension
7. Risk of aspiration during airway procedure should be minimized. Administration of Muscle relaxant prevents coughing thereby preventing sudden increase in ICP. Argument against succinyl choline is the potential to increase ICP, however the suspected rise of ICP is only transient and may be blunted with adequate dose of induction agent such as thiopentone
8. Hypotension is extremely detrimental to brain. Goal is to maintain SBP above 90 mmHg, CPP in range of 50-70 mmHg. In absence of ICP monitoring an ICP of 20 mmHg should be assumed and MAP should be kept above 60 mmHg.
9. The optimal Hemoglobin level in TBI patients is still unclear but there is no benefit of a liberal transfusion strategy (transfusion when Hb<10 g/dl) in moderate to severe TBI patients and it is not recommended.
10. Reduction of ICP in patients with head injuries can be accomplished effectively using osmotic diuretics.

a. Mannitol is the most commonly used agent (0.25 to 1 g/kg) and is available for IV administration in either a 20% or 25% solution. With repeated doses one must be cautious that serum osmolarity should not be allowed to exceed 320 mOsm. Intravascular volume depletion should be avoided.
b. In patients with severe TBI and elevated ICP refractory to mannitol treatment, 7.5% hypertonic saline administered as second tier therapy can increase cerebral oxygenation and improve cerebral and systemic hemodynamics.
c. Hyperventilation is an effective way to reduce ICP. It is useful in the setting of an acutely increased ICP that needs to be controlled until more definitive therapy can be initiated.
d. Barbiturates may be used as an adjunct to other therapy for controlling ICP. Barbiturate therapy is appropriate only in patients who are hemodynamically stable and have been adequately resuscitated. Propofol is a reasonable alternative to barbiturates for ICP management.
e. Current recommendations are that patients with TBI should be maintained at normocapnia except when hypocapnia is necessary to control acute increases in ICP.

11. Coagulation disorders are a common problem after TBI. Coagulation disorders can also result from secondary brain injury. Hemostatic drugs such as antifibrinolytics, such as trenaxamic acid and procoagulants such as recombinant factor VII are sometimes used in treatment of coagulopathy after TBI.
12. Hyperglycemia after TBI is associated with increased morbidity and mortality. Tight glucose control with intensive insulin therapy remains controversial due to recurring dangerous episodes of hypoglycemia. Evidence shows a target glucose range of 80–180 mg/dl seems reasonable in postoperative period.
13. Patients may present with head injuries in isolation or in conjunction with other injuries. Up to 50% of patients with severe traumatic brain injury have major extra cranial injuries. As a result the presence of coexisting injuries should be actively sought and excluded. A systematic approach to evaluation and initial management, such as that proposed by Advanced Trauma Life Support, should be adopted for these patients.
14. Prophylactic hypothermia is of no use unless it is maintained for more than 48 hrs (decreases mortality).
15. High dose methyl prednisolone is of no use in moderate to severe TBI, rather it increases mortality.

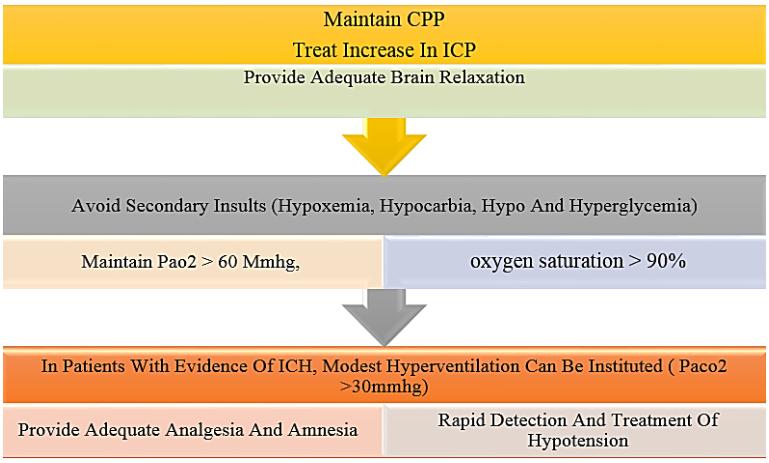

Fig. 1: Anesthetic management: The major goals of anesthetic management of TBI include

Patients with TBI requiring surgery can be subdivided into those who require emergent surgery and those who require non-emergent surgery.

Emergent Surgery: These patients are already intubated in casualty area. Their neurologic condition can be determined by GCS score, examining the pupils, and reviewing the CT scan.

1. The patient's hemodynamic status is also extremely important. Patients may demonstrate a Cushing's response (hypertension and bradycardia), which signifies brain stem compression from increased ICP. These classic findings may be masked by hypovolemia, and their absence does not rule out brain stem compression.
2. These patients usually do not have ICP monitors in place, but one can assume the presence of intracranial hypertension. The presence of midline shift on CT scan and pupillary abnormalities on physical examination reinforce this diagnosis.
3. Moderate hyperventilation should be used in these patients until the dura is opened because the elevation in ICP is likely more detrimental than short-term hyperventilation.
4. Blood pressure management is critical in these patients.

Non-emergent Surgery: Patients with TBI frequently have other injuries, especially fractures requiring operative fixation. The timing of surgery in these patients remains a controversial issue. In the setting of refractory elevations in ICP or very labile ICP, only emergent surgery should be performed.

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