ANAESTHETIC MANAGEMENT OF POSTERIOR FOSSA SURGERY

G.S. Umamaheswara Rao

Professor, Department of Neuroanaesthesia,
National Institute of Mental Health and Neurosciences, Bangalore 560 029

Anaesthetic management of patients during posterior cranial fossa surgery poses unique challenges to the anaesthesiologists because of the proximity of the surgical lesions to vital cardiorespiratory centres and cranial nerves. In addition, complications associated with surgical positioning demand specialised monitoring and meticulous intraoperative care.

SURGICAL LESIONS

A variety of congenital, inflammatory, neoplastic, traumatic and vascular lesions in the posterior fossa, may require surgical intervention. A list of some of the common conditions is given in table 1.

Table 1. Common Surgical Lesions in Posterior Fossa

1) **Congenital:**	Arnold Chiari malformation
2) **Neoplastic:**	Midline masses:
	a) Medulloblastoma
	b) Ependymoma
	c) Brainstem glioma
	d) Intraventricular papilloma
	Cerebello-pontine angle tumours
	a) Schwannoma of VIII, V or lower cranial nerves
	b) Meningioma
	Cerebellar tumours: Astrocytoma, Meningioma
3) **Inflammatory:**	Cerebellar Abscess, Cerebellar Tuberculoma
4) **Traumatic:**	Extradural haematoma, Cerebellar contusion/haematoma
5) **Vascular:**	Aneurysms of vertebrobasilar system, Arteriovenous malformations
6) **Miscellaneous:**	Trigeminal neuralgia, Hemifacial spasms

PREOPERATIVE ASSESSMENT

Patients requiring posterior fossa surgery range from small children to the elderly. Apart from the general preanaesthetic assessment that is relevant to the individual age group, the following evaluation must be performed:

Obstructive hydrocephalus with raised intracranial pressure (ICP) is a common complication of mass lesions in the posterior fossa. Headache, vomiting, papilloma, enlarged lateral ventricles on CT scan and presence of periventricular lucency suggest raised ICP. History must be obtained about earlier cerebrospinal fluid (CSF) diversion procedures (ventriculo-peritoneal or ventriculo-arterial shunt or endoscopic third ventriculostomy). A functioning preoperative shunt reduces the risk of intraoperative raised ICP, while the concerns relating to raised ICP (e.g., intraoperative swollen brain) are high in patients with untreated hydrocephalus.

Lower cranial nerve dysfunction may be present in patients with cerebello-pontine angle and brainstem tumors. Dysphagia, and impaired gag reflex may result in preoperative pulmonary aspiration; the patient may have active pulmonary infection when he presents for surgery.

Repeated vomiting and dysphagia may cause dehydration in these patients. Preoperative hydration status must be evaluated to ensure intraoperative haemodynamic stability.

Cardiac reserve must be evaluated in the elderly, hypertensive patients and those with significant cardiac disease. Limited cardiorespiratory reserve may limit the positioning options. The risk of orthostatic hypotension in sitting position is increased in patients with uncontrolled hypertension, advanced age and hypovolemia. Pre-existing cardiac arrhythmia may interfere with interpretation of intraoperative cardiac changes due to brainstem dysfunction.

In patients planned for surgery in sitting position, information should be obtained regarding the presence of patient foramen ovale (PFO) or any other right-to-left intracardiac shunt. Autopsy studies have demonstrated a 27% incidence of PFO, but preoperative testing has shown functional PFO only in 6% (1).

SURGICAL POSITION

Sitting position, which was most favoured until about 15-20 years ago is slowly giving way to horizontal positions (prone, lateral and supine) because of concerns relating to venous air embolism. Some centres however continue to practice it frequently (2,3). Limited use of sitting position should remain in the neurosurgeon's armamentarium so that, where necessary, the patient is not denied the benefit of sitting position. Assessment of the risk benefits of individual cases is of great importance. The patient must be adequately informed of the specific risks of venous air embolism, hypotension and neurological complications. Preoperative echocardiography is recommended for identification of patent foramen ovale (4).

Supine Position

This position has a limited role in posterior fossa surgery; its use is limited to microvascular decompressive procedures (for trigeminal neuralgia) and surgery on acoustic neurinomas through translabrynthine approach. The head is turned laterally and the ipsilateral shoulder is pulled away from the operative site. Injury to brachial plexus may occur if the stretch on the shoulder is excessive. Supine position leads to a progressive reduction of functional residual capacity and dependent atelectasis leading to increased pulmonary shunt over a period of time.

Prone Position

Prone position is commonly used for midline lesions and medial cerebellar lesions. This position significantly reduces the risk of air embolism though it is possible if the head is considerably elevated above the heart level. Reduction of functional residual capacity in this position is less than that in supine position if pressure on the abdomen is avoided. Postoperative blindness is a serious complication that may be caused by central retinal arterial thrombosis. Dependent positioning may lead to massive face and tongue swelling. Special care must be taken to fix the endotracheal tube securely so that it does not slip out during the course of surgery. Extreme flexion of the neck may cause a kink of endotracheal tube. An armoured tube decreases this risk.

Lateral Position

Cerebello-potine lesions and lateral cerebellar lesions are operated upon in this position. The lower arm is supported in a sling to allow proper fixation of the head in the three-pin frame. Using an axillary roll prevents brachial plexus injury. Respiratory compromise is less than in supine position, but may occur rarely due to redistribution of ventilation perfusion ratios. Park bench position is a modification of lateral position used for cerebello-pontine angle surgery; the head is elevated above the level of right atrium, the neck is flexed and the head and neck are rotated 30° to the opposite side.

Sitting Position

Sitting position offers the advantages of excellent surgical access and decreased blood loss. Despite a large volume of knowledge pertaining to the life-threatening complications associated with sitting position, a 1994 British survey revealed that 34% of the neurosurgical centres in

Britain still use this position for infratentorial surgical procedures (2). In another German survey, sitting position was preferred for posterior fossa surgery by 45% of the neurosurgeons (5). To decrease the risk of haemodynamic instability, most often, a 'lounging position" rather than a vertical sitting position is employed. A recent study, reported very low incidence of complications in paediatric posterior fossa surgery carried out in sitting position (3).

Contraindications for Sitting Position: Certain preoperative conditions, which subject the patients to increased risk of air embolism, preclude surgery in sitting position. Examples of such conditions are patent ventriculoatrial shunt, cardiac conditions resulting in right atrial pressure greater than left atrial pressure, patent foramen ovale (PFO), symptoms of cerebral ischemia in sitting position. Extremes of age, uncontrolled hypertension and chronic obstructive pulmonary disease (COPD) are some relative contraindications for sitting position (4).

Some frequently encountered complications during posterior fossa surgery are listed in table 2.

Table 2. Common Complications of Posterior Fossa Surgery in Sitting Position

1. Hypotension
2. Venous Air Embolism (VAE)
3. Airway Problems
4. Tension Pneumocephalus
5. Neurological Complications
   - Peripheral Nerve Injuries
   - Upper Cervical Quadriplegia
   - Ischemic Cerebral Damage

***Hypotension*:** Peripheral pooling of the blood in the dependent areas due to vasodilation during anaesthesia may result in hypotension at the time of positioning. The incidence of this complication does not seem to be different between patients with and without cardiac disease (6). Prevention of this complication depends on ensuring normovolemia before positioning, gradual positioning, usage of compressive stockings for legs and adoption of a *lounging position* rather than a strict sitting position. Hypotension during the course of surgery is related most often to surgical complications such as blood loss, air embolism and brainstem disturbances than to the sitting position *per se*.

Acute hypotension at the time of positioning normally responds to intravenous administration of fluids. If the hypotension persists despite adequate fluid administration, the operating table should be tilted backwards to aid venous return to the heart. If the hypotension is not corrected by these simple measures, it is advisable to return the patient to supine position. Administration of vasopressors to treat the hypotension during positioning is not recommended except in life-threatening situations. Blood loss is very poorly tolerated in sitting position and it is essential to ensure adequate intravascular volume throughout the procedure.

***Venous Air Embolism (VAE)**:* Air embolism occurs during posterior fossa surgery in sitting position when there is an open non-collapsible vein and the pressure at the site of opening is subatmospheric. The reported incidence of this complication varies with the sensitivity of the monitoring device employed for its detection. With the advent of precardial Doppler, most studies report an incidence varying between 30-45% (7). Microbubbles of air appeared in the right atrium in all patients monitored by transesophageal echocardiography in one study (8). Despite such high rate of detection, the actual risk of clinically severe air embolism seems to be very low.

The clinical consequences of venous air embolism depend on the volume of air entrained, the rate of entrainment of air and the presence of patent foramen ovale. Continuous entrainment of air results in an increase in the pulmonary arterial pressure as a result of mechanical blockade of the pulmonary vasculature and also reflex pulmonary vasoconstriction. Microbubbles in the

pulmonary circulation may also initiate pulmonary vascular endothelial injury leading to hypoxemia and hypercapnia. Bronchoconstriction may occur in some patients. The consequences of massive air embolism are decrease in cardiac output, hypotension and cardiovascular collapse. Other complications of VAE include pulmonary oedema and ARDS.

Paradoxical air embolism (PAE) is a condition in which the air enters the systemic circulation from the right heart through a patent foramen ovale or a ventricular septal defect (9). It is estimated that the incidence of PFO in normal population is around 27%. PFO may be diagnosed preoperatively by Valsalva manoeuvre combined with contrast transosophageal echocardiography (TEE). A recent study suggests that ventilation manoeuvre with a peak pressure of 30 cmH2O in an anesthetized patient is superior to the conventional TEE (**10**). Air may also be forced through the pulmonary capillary bed, especially in the presence of pulmonary vasodilatory drugs. Paradoxical air embolism is facilitated by hypovolemia, which causes a reduction of both central venous pressure and systemic arterial pressure. Embolisation of coronary circulation due to PAE may lead to arrhythmia and cardiac arrest; cerebral embolisation may lead to cerebral infarction.

Emergency management of venous air embolism depends on accurate and speedy detection of the complication. The rate of detection of air-embolism has increased ever since the introduction of precardial Doppler. Detection of air embolism based on clinical findings such as hypotension, hypoxia, cardiac arrhythmia, mill-wheel murmur and gasping respiration is quite late. Increased airway pressure has also been reported as an additional sign to detect VAE. Precardial Doppler can detect even small quantities of air (0.5 ml/kg). Spectral analysis of the precardial Doppler signal to obtain quantitative information has been attempted. To date, TEE remains the most sensitive method to detect VAE. Using a 3.5-5.0 Hz oesophageal probe, it is possible to measure volumes as low as 0.02 ml/kg. One of the advantages that TEE offers over precardial Doppler is that it can detect incidents of paradoxical air embolism; in fact, risk of paradoxical air embolism is a definite indication for the use of TEE. Routine use of TEE is limited by the cost and the need for specialised training. Change in end tidal CO_2 pressure (PetCO2) occurs later than Doppler change but earlier than cardiovascular changes. PetCO2 provides an estimate of the physiologic derangement; the change, however, is not specific for VAE. Pulmonary arterial and central venous pressure monitoring may also be helpful to detect VAE, but both these methods are less sensitive than Doppler, TEE and PetCO2.

Prevention of VAE depends on attention to meticulous haemostasis at every stage during surgery, especially during the dissection of muscle planes and craniectomy; VAE may also occur during release of PEEP and repositioning of the patient into the supine position. Therefore, continuous monitoring should be carried out until the patient is returned to supine position (11). Appropriate placement of central venous catheter with the tip at the junction of superior vena cava and right atrium aids recovery of air in the event of air embolism. Preoperative TEE examination for PFO would forewarn the risk for PAE.

When air embolism is detected, the operating surgeon must be informed about the event; efforts must be made to secure haemostasis. If the bleeding point is not evident immediately, applying Valsalva maneuver might help in its detection. Flooding the operative field with saline prevents further entrainment of air. Discontinuation of nitrous oxide from the anaesthetic obviates the risk of enlargement of the air bubbles. Application of PEEP is advocated by some authors to decrease air embolism. There is not enough evidence to support the view that PEEP increases the intracranial venous pressure so as to decrease the incidence of VAE. On the other hand, there are suggestions that in susceptible individuals, it may enhance the risk of paradoxical air embolism by reversing the pressure gradient between right and left atrium. Application of pressure on the jugular veins aids detection of the bleeding point; this maneuver may entail the risk of acute brain swelling. Though military antishock trousers (MAST) have been shown to increase the venous pressure, the elevation is not sustained for longer than 30 min and they do not seem to offer any clinical benefit.

***Airway Complications**:* Acute flexion of the neck in sitting position may cause a kink of the endotracheal tube. As the patient's trachea is intubated in supine position with the neck in extension, after assumption of the sitting position with acute neck flexion, there is a potential for the endotracheal tube to migrate into one of the main bronchi or to slip out of the larynx. Acute neck flexion in sitting position has also been associated with massive swelling of the face and tongue and ischaemic necrosis of the tongue necessitating postoperative tracheostomy (12,**13**). Avoidance of extreme flexion and ensuring adequate space between the chin and the sternum prevents kinking of the endotracheal tube. Avoiding Guedel's airway has been suggested to prevent swelling of the tongue. Repeated auscultation of the chest during positioning and monitoring the airway pressure, end-tidal carbon dioxide and oxygen saturation help to detect the airway problems at the earliest before they lead to serious consequences.

***Tension Pneumocephalus**:* Tension pneumocephalus is a complication reported more frequently in sitting position, though it may occur in any craniotomy where a large empty subdural space is created due to excessive shrinkage of the brain or a large empty space is left after decompression of a big tumour (14). During surgery in sitting position, a combination of hyperventilation, mannitol and CSF drainage reduces the brain volume. The potential subdural space above the brain surface is occupied by air when the dura is open. At the end of surgery after the closure of the duramater, re-expansion of the brain and restoration of normal PaCO2 (restoration of normal cerebral blood volume) increase the pressure within the trapped air. The volume of air may increase if administration of nitrous oxide is continued after dural closure. Irrigation of the subdural space with saline, discontinuation of nitrous oxide and hyperventilation after dural closure may prevent this complication.

Delayed recovery is a common manifestation of tension pneumocephalus. In less severe forms the patient may complain of persistent headache. In more severe cases, the patient may show signs of severe rise in intracranial pressure with imminent herniation. A twist-drill aspiration of air rapidly improves the neurological state. Supine position with administration of 100% oxygen may facilitate absorption of air in less severe cases.

Neurological Complications

Quadriplegia: Mid-cervical quadriplegia following operations in sitting position has been described. Elderly spondylotic patients are prone for this complication. The precipitating insult is not clear but may be related to prolonged flexion producing stretch injury of the spinal cord substance or ischemia of the spinal cord (15). Attention to maintenance of adequate perfusion pressure in sitting position and avoidance of excessive flexion prevent this complication.

Subdural Haematoma: Supratentorial subdural haematoma occurs in less than 1% of patients operated upon in sitting position (16). Subdural haematoma is caused by stretching of the bridging veins due to excessive cerebral dehydration and effective CSF drainage through a ventriculoperitoneal shunt. Management of this complication comprises of an emergency CT scanning to confirm the diagnosis and prompt surgical evacuation of the clot.

Ischemic Cerebral Damage: With an increased gradient between the heart and brain, there is a potential risk of reduction of cerebral blood flow in the sitting position. A number of studies examined this issue and it appears that the risk is very low when the intracranial pressure is normal. The cerebral blood flow values are the lowest in sitting position when the intracranial pressure is high (17).

Peripheral Nerve Injuries: Several peripheral nerve injuries have been described in association with sitting position. These include damage to common peroneal nerve, and less commonly recurrent laryngeal nerve injury (4).

COMPLICATIONS COMMON TO PATIENTS OPERATED UPON IN ALL POSITIONS

Cerebellar Swelling

Slack cerebellum is one of the major advantages claimed for the sitting position. However, it is not unusual, during surgery, to encounter cerebellar swelling as a result of excessive retraction, deep-seated haematoma or dilated ventricles. The problem may be further exaggerated by high concentrations of inhalational anaesthetics, venous obstruction due to excessive flexion or rotation of the neck and inappropriate control of $PaCO_2$. Management of this complication in sitting position is difficult as aggressive measures such as controlled hyperventilation, and high-dose barbiturate therapy, that can be applied with reasonable safety in supine position, are fraught with major risks of cerebral ischemia in sitting position.

If the *brain is tense at the time of dural opening*, the position of the head and neck should be verified. If the neck is rotated causing obstruction to jugular venous outflow, appropriate correction should be carried out. Airway obstruction, hypoxia and hypercapnia should be ruled out as the possible causes. Discontinuation of nitrous oxide from anaesthetic may be beneficial. Lowering the concentrations of inhalational agents by substitution with intravenous agents might help reduce the brain tension. Incipient straining could be avoided by ensuring adequate muscle relaxation. If PaCO2 is not in the hypocapnic range, minute ventilation could be increased to bring it down to around 25 mmHg. Caution should be exercised in institution of hyperventilation as reduction of $PaCO_2$ even to 25 mmHg could result in cerebral hypoxia. Mannitol in a dose of 0.5 - 1.0 g/kg in combination with frusemide 0.5 - 1.0 mg/kg reduces brain volume. As all these measures are being undertaken, it is preferable not to open the dura until there are signs of laxity. After the dural opening, a rapid initial decompression of the mass lesion would prevent the vicious cycle of venous obstruction at the margins of craniotomy and further brain bulge. In patients with evidence of ventriculomegaly, establishing ventricular CSF drainage through a supratentorial burr-hole may be helpful to decrease the brain-bulge.

Intraoperative Brainstem Dysfunction

Integrity of the brainstem function may be jeopardised during the posterior fossa surgery due to pressure, vascular compromise or mechanical distortion of the brainstem. Such intraoperative ischemic episodes, when prolonged, may lead to postoperative neurologic deficits, disturbances of spontaneous respiratory function and cardiovascular instability. At present, in most of the institutions, intraoperative monitoring of the integrity of the brain stem function relies on monitoring the heart rate, rhythm and blood pressure. Sinus bradycardia, junctional bradycardia, sinus arrest and ventricular tachycardia may occur during removal of medulloblstomas, intraventricular ependymomas, and brainstem gliomas, dissection of acoustic neurinomas and surgery on vertebrobasilar aneurysms. Manipulations in the root entry zones of cranial nerves (surgery for trigeminal neuralgia, lower cranial nerve schwaanomas) may cause hypertension, bradycardia, ventricular tachycardia and ST segment changes.

Any unexplained change in the cardiac rate or rhythm or blood pressure, irrespective of its magnitude, should be brought to the notice of the operating surgeon, as it could be a potential indicator of a brain-stem insult. The surgeon, in turn, should examine the possibility of the change being a result of the surgical procedure under progress. It may be worthwhile to change the approach temporarily in order to avoid any further insult to the brainstem. Usage of pharmacological agents to correct these brainstem cardiovascular changes is to be discouraged unless the change is life-threatening. Modification of these parameters by pharmacological agents prevents utilisation of these parameters for further monitoring of the brainstem function.

Evoked potential monitoring is a sensitive technique for detection of intraoperative brainstem dysfunction; short latency somatosensory evoked potentials (SSEP) can be helpful to monitor the cervical cord ischemia as well as the brainstem function. Brainstem auditory evoked potentials (BAEP) are more robust and less affected by anaesthetics than SSEP. A 50% reduction in the height or a 1 msec increase in the latency of N_{20} wave of SSEP or the fifth peak of BAER are considered clinically significant. These two modalities of evoked potentials do not

seem to differ in their ability to predict postoperative neurological deficit (18). They are extensively used to monitor eighth cranial nerve and brainstem function during resection of acoustic nerve tumours and microvascular decompression of cranial nerves. The major limitation of both SSEP and BAER is that they monitor only sensory pathways and damage to motor tracts and cranial nerves can occur with a normal intraoperative SEP study. Secondly, the recordings may be affected by anaesthetics and changes in physiological parameters such as mean arterial pressure, blood gases and body temperature. Motor evoked potentials have been successfully utilised to monitor motor function during posterior fossa surgery. They have been used to guide intraoperative compression and to predict postoperative function in medullary tumours and Chiari malformation.

Neurological Complications

Cranial Nerve Dysfunction
Surgical procedures on cerebellopontine angle lesions could result in postoperative dysfunction of VII, VIII, IX and X cranial nerves. Involvement of IX and X cranial nerves could lead to the risk of postoperative pulmonary aspiration and delay in tracheal extubation. Significant obtundation of pharyngeal and laryngeal reflexes may necessitate a tracheostomy.

Cerebellar Mutism
Cerebellar mutism is a condition typically found only in children following operations requiring entry into the cerebellum, especially the vermis. The cause is not certain, but proposed to be due to injury to deep cerebellar nuclei. The mutism is usually transient resolving in a few months.

Respiratory failure
Some of the posterior fossa lesions like Arnold Chiari malformation and cervicomedullary junction tumours may be associated with increased risk of postoperative respiratory failure due to central sleep apnoea and hypoventilation (19).

Anaesthetic Technique
The goals of anaesthetic management in posterior fossa surgery are maintenance of haemodynamic stability, cerebral perfusion pressure and oxygenation, facilitating brain retraction, monitoring for air embolism, and choosing an anaesthetic technique compatible with electrophysiological monitoring, where it is used. No single anaesthetic technique meets all the requirements. Nitrous oxide is best avoided in situations associated with increased risk of air embolism and tension pneumocephalus. Volatile anaesthetics may help to achieve smooth control of blood pressure; they may however, increase the risk of hypotension and reduction of cerebral perfusion pressure in patients with raised ICP and may interfere with interpretation of evoked potentials. Use of controlled ventilation with narcotics, propofol and muscle relaxants provides slack brain. Some reports have indicated the usefulness of an anaesthetic technique with spontaneous respiration to monitor brainstem function intaoperatively. Respiratory changes seem to occur much earlier and more frequently than cardiovascular and even evoked potential changes (20). But the technique may be associated with progressive atelectasis, hypercapnia and hpoxia, all of which increase the risk of brain swelling. "Gasp response" has been noticed during venous air embolism in awake individuals. A similar response during surgery under spontaneous ventilation might increase the volume of air entrained.

In conclusion, anaesthetic management of posterior fossa lesions requires, in addition to the general principles followed in any intracranial surgery, an understanding of the primary pathology of the surgical lesion, institution of measures to deal with the adverse consequences of the surgical positions and monitoring for and prevention of intraoperative brainstem dysfunction.

References

1. Black S, Mazzi DA, Nishimura RA, Cucchiara RF. Preoperative and intraoperative echocardiography to detect right to left shunt in patients undergoing neurosurgical procedures in sitting position. Anesthesiology 1990:72;436-8
2. Elton RJ, Howell RSC. The sitting position in neurosurgical anaesthesia: a survey of British practice in 1991. Brit J Anaesth 1994; 73:247-248
3. Harrison EA, Mackersie A, McEwan A, Facer E, The sitting position for neurosurgery in children: A review of 16 years' experience. Br J Anaesth 2002; 88:12-7
4. Porter JM, Pidgeon C, Cunningham AJ. The sitting position in neurosurgery: a critical approach. Br J Anaesth 1999; 82:117-28
5. A. Schaffranietz L, Grothe A, Olthoff D. Use of the sitting position for neurosurgery. Results of a 1998 survey in Germany. Anaesthesist 2000; 49:269-74
6. Black S, Ockert DB, Oliver WC, Cucchiara RF. Outcome following posterior fossa craniotomy in patients in the sitting and horizontal position. Anesthesiology 1988; 69:49-56.
7. Porter SS, Sanan A, Rengachary SS. Surgery and anaesthesia of the posterior fossa In: Text book of neuroanaesthesia (ed) Albin M.S. McGraw-Hill, New-York, 1997; pp:971-1008.
8. Mammoto T, Hayashi Y, Kuro M. Incidence of venous and paradoxical air embolism in neurosurgical patients in the sitting position; detection by transoesophageal echocardiography. Acta Anaesthesiol Scand 1998; 42: 643-7
9. Bedell EA, Berge KH, Losasso TJ. Paradoxical air embolism during venous air embolism: Transoesophageal echocardiac evidence of transpulmonary air passage. Anesthesiology 1994; 80:947-50.
10. Greim CA, Trautner H, Kra¨mer K, Zimmermann P, Apfel CC, Roewer N. The Detection of Interatrial Flow Patency in Awake and Anesthetized Patients: A Comparative Study Using Transnasal Transesophageal Echocardiography. Anesth Analg 2001;92:1111–6
11. Schmitt HJ, Hemmerling TM. Venous air emboli occur during release of positive end expiratory pressure and repositioning after sitting position. Anesth Analg 2002; 94:400-3
12. Narayan VB, Umamaheswara Rao GS. Unilateral facial and neck swelling after infratentorial surgery in the lateral position. Anesth Analg. 1999;89:1290-1
13. Teeple E, maroon J, Reuger R. Hemimacroglossia and unilateral ischemic necrosis of the tongue in a long duration neurosurgical procedure. Anesthesiology 1986; 64:845.
14. Grundy BL, Spetzler RF. Subdural pneumocephalus resulting from drainage of cerebrospinal fluid during craniotomy. Anesthesiology 1980; 52:269-71
15. Wider BL. The etiology of midcervical quadriplegia after operation with the patient in sitting position. Neurosurgery 1982; 11:530-1
16. Seiler RW, Zurbrugg HR. Supratentorial intracerebral haemorrhage after posterior fossa operation. Neurosurgery 1986; 18:472-4
17. Ernst PS, Albin MS. Intracranial and spinal cord haemodynamics in the sitting position in dogs in the presence and absence of increased intracranial pressure. Anesth Analg 1990; 70:147-153.

18. Manninen PH, Patterson S, Lam AM, Gelb AW, Nantau WE. Evoked potential monitoring during posterior fossa aneurysm surgery: a comparison of two modalities. Can J anaesth 1994; 41:92-7
19. Williams DL, Umedaly H, Martin H, Boulton A. Chiari type I malformation and postoperative respiratory failure. Can J Anaesth 2000; 47:1220-3
20. Manninen PH, Cuillerier DJ, Nantau WE, Gelb AW. Monitoring of brainstem function during vertebral basilar aneurysm surgery. The use of spontaneous ventilation. Anesthesiology 1992; 77:681-5