ANAESTHESIA FOR SITTING CRANIOTOMY

SURESH KUMAR GNANASEKARAN
Department of Anaesthesiology,
MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL

Abstract: Most explorations of the posterior cranial fossa can be performed with the patient in either a modified lateral or prone position, but the sitting position may be preferred by some surgeons because neurosurgery in the sitting position offers certain advantages. This position provides optimal surgical exposure for posterior fossa surgery because tissue retraction and risks of cranial nerve damage are reduced. Cerebrospinal fluid and cerebral venous drainage is enhanced by gravity producing a less tense brain which also improves surgical exposure and reduces blood loss. However, these advantages must be balanced against the risks which include high incidence of venous air embolism, paradoxical arterial air embolism, pneumocephalus, cerebral and myocardial ischaemia secondary to hypotension caused by venous pooling of blood in lower limbs. Other particular concerns to the anaesthesiologist are airway management, avoidance of pressure injuries, and the risk of upper airway edema, and rarely quadriplegia due to compression of the cervical spinal cord. Overall the benefits of the sitting position outweighs the risks if the patients are carefully selected and intraoperative management is conducted with care. We present a case of cerebellar space occupying lesion in which the excision was done in sitting position under general anaesthesia.

Keyword: posterior, fossa, sitting, craniotomy, venous, air, embolism

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INTRODUCTION

Sitting craniotomy is commonly done for the excision of posterior cranial fossa tumours and vascular anomalies. This position provides optimal surgical exposure for posterior fossa. Cerebrospinal fluid and cerebral venous drainage are enhanced by gravity producing a less tense brain and decreased pooling of blood in the operating field which also improves surgical exposure. In spite of these advantages it is rarely used because of the
complications like high incidence of venous air embolism (25%–50%)\(^2\), paradoxical arterial air embolism, pneumocephalus, cerebral and myocardial ischemia secondary to hypotension and nowadays surgeons are comfortable with other positions like park bench position. Other particular concerns to the anaesthesiologist are airway management, avoidance of pressure injuries, and the risk of upper airway edema, and rarely quadriplegia due to compression of the cervical spinal cord.

**CASE DESCRIPTION**

A 55-year-old lady came to the neurosurgery outpatient department with the complaints of giddiness and headache for the past 3 months. In her evaluation, magnetic resonance imaging revealed a midline cerebellar space occupying lesion with obstructive hydrocephalus. Other investigations were normal. She underwent emergency ventriculoperitoneal shunt on the left side, under general anaesthesia in supine position, which was uneventful. Elective craniotomy and excision of the space occupying lesion was planned. The neurosurgical team preferred sitting position for the craniotomy. The disadvantages and complications of craniotomy in sitting position and alternate positions possible, were discussed with the neurosurgical team. Because of the neurosurgical team’s persistence, sitting position was decided. She was diagnosed to have hypertension and was given T.Amlodipine for three weeks. After three weeks, her blood pressure was found to be normal. Her haemoglobin was 12.3 gm%. Other routine investigations were normal. Her chest x ray was normal. She underwent an echocardiography which was also normal. The nature of the procedure planned, the complications that can arise and the expected outcome was explained to the patient and her attenders and an informed consent was obtained from her. Four units of packed cells were cross matched and kept ready. A ventilator was checked and kept ready for postoperative mechanical ventilation, if the need arises.

After shifting her to the operating room, pulseoximeter, noninvasive blood pressure monitor, electrocardiography were connected. An intravenous access was obtained in left forearm with 18G cannula. She was given inj midazolam 2mg, inj glycopyrrolate 0.2mg and inj fentanyl 100 microgram intravenously on the operating table. Preoxygenation was done for 3 minutes. As the patient was on T.Amlodipine in the preoperative period, hemodynamic instability was anticipated during induction and positioning. So inotropes were kept ready, patient was prehydrated with 500ml of normal saline. Induction was done slowly with 200mg of thiopentone sodium and intubated with 7.0 mm size cuffed endotracheal tube, facilitated by vecuronium 6mg. Inj lidocaine 60mg was given i.v. before laryngoscopy to blunt the hemodynamic response. End tidal carbondioxide monitoring was started after intubation. Sevoflurane 2% was given in 40% oxygen and 60% nitrous oxide for maintenance. Fentanyl 20 mic was repeated every hour and vecuronium 1mg every 30 mins. A nasogastric tube was passed, and the bladder was catheterized. Throughout the procedure, urine output was monitored, which was 75ml/hour. Temperature monitoring could not be done due to technical problem with the monitors. Right internal jugular vein was cannulated (shunt is on the left side) with a triple lumen central venous catheter.
Compression dressing was applied to the lower limbs to prevent venous pooling of blood. 1000ml of normal saline was infused before the gradual elevation of the head began. The head was elevated in 20 degree increments every 10 minutes and hemodynamic parameters were monitored continuously. Positive pressure ventilation was adjusted to maintain the end tidal carbon dioxide at around 30mmHg. Mean blood pressure was maintained between 65-75 mmHg. When the tumor was removed, hemodynamic parameters were closely monitored, and there was no hemodynamic instability. No untoward events were noted during the intraoperative period. The duration of surgery was 3 hours, and blood loss was 300ml, which was replaced by equal volume of colloid. After the procedure was over, supine position was attained gradually. Anesthesia was maintained till the head dressing was given. As there was no significant hemodynamic instability, no significant blood loss and as the duration of the surgery was reasonably short, it was planned to extubate the patient on the table, provided she recovered well and there was no neurological deficits. Ventilation was assisted till spontaneous breathing returned and normocapnia was ensured. Inj. esmolol 25mg was given to attenuate the hemodynamic responses, as the patient regained consciousness. Reversal was done with inj.glycopyrrolate 0.4mg inj. neostigmine 2mg. 60mg of lidocaine was given. Once the patient was found to be oriented and there was no signs of any neurological deficit, she was extubated. She was transferred to neuro I.C.U. There were no postoperative complications and was shifted to general ward after 48hrs. She was discharged after one week.

DISCUSSION:
The patient would be actually in a semirecumbent in the standard sitting position. The back is elevated to 60°, and the legs are elevated and the knees are flexed. The head is fixed in a three-point holder with the neck flexed, the arms remain at the sides with the hands resting on the lap.

PREOPERATIVE ASSESSMENT:
The primary objective is to identify the contraindications to the sitting craniotomy such as age more than 70yrs, patent foramen ovale, uncontrolled hypertension, COPD. Cardiovascular and cerebrovascular status should be assessed and therapy should be optimized.

MONITORS:
Of the mandatory monitors, ETCO2 monitor is of paramount importance. Urine output needs to be monitored continuously. Usage of Trans esophageal echo...
and Trans thoracic Doppler are advised for early detection of venous air embolism.

**INDUCTION AND MAINTENANCE:**
As with any craniotomy the choice of particular agents is not important, but stable anaesthesia has paramount importance. The effects of a particular drug on circulatory hemodynamics should be considered. Central venous access frequently allows aspiration of entrained air. Many clinicians consider right atrial catheterization mandatory for sitting craniotomies. Optimal recovery of air following venous air embolism is provided by a multiorificed catheter positioned high in the atrium at its junction with the superior vena cava. Placing the anesthetized patient in the sitting position conveys some risk of impaired cardiovascular function, in particular, hypotension. Pressor administration is required in some patients. Measures to minimize hypotension associated with the sitting position include a slow, staged positioning over 10-20 min and use of the 'G suit' inflated with compressed air applied to the lower extremities and pelvis.

**EMERGENCE FROM ANESTHESIA**
Emergence should be timed to coincide not with the final suture, but rather with the conclusion of the application of the head dressing. Administration of lidocaine of 1.5mg/kg body weight is an effective technique for reducing airway responsiveness and the likelihood of coughing and straining as depth of anesthesia is reduced in anticipation of emergence. To prevent hypertension during the last stages of a craniotomy, administration of vasoactive agents, most commonly labetalol and esmolol is done. Other agents, including enalapril, nicardipine, and diltiazem, have been used to good effect. Administration of dexmedetomidine during the procedure also has been reported to reduce the hypertensive response to emergence. Emergence should be smooth. Coughing and straining and arterial hypertension should be avoided. The sudden increases in intrathoracic pressure are transmitted to arteries and veins, producing transient increases in cerebral arterial and venous pressure with the same potential consequences: edema formation, bleeding, and elevation of intracranial pressure. Hypertension can contribute to intracranial bleeding and increased edema formation. It is possible that neurologic events have occurred that would delay recovery of consciousness, or there may be lower cranial nerve dysfunction during the surgery. Some patients may need mechanical ventilation. So it generally is best to wait until the likelihood of the patient's recovery of consciousness is confirmed and until patient cooperation and airway reflexes are likely to have recovered.

**VENOUS AIR EMBOLISM**
Venous air embolism can occur when the pressure within an open vein is subatmospheric. This can happen in any position and during any procedure whenever the wound is above the level of the heart. The incidence of venous air embolism is highest during sitting craniotomies (25–50%). The consequences of venous air embolism depend on the volume as well as the rate of air entry and whether the patient has a probe-patent foramen ovale (10–25% incidence). The latter is important because it can facilitate passage of air into the arterial circulation (paradoxical air embolism). Small bubbles are well tolerated by most patients. When the amount entrained exceeds the rate of pulmonary clearance, pulmonary artery pressure progressively rises. Eventually, cardiac output decreases in response to increases in right ventricular afterload. This leads to
refractory hypotension. Preexisting cardiac or pulmonary disease enhances the effects of venous air embolism; relatively small amounts of air may produce marked hemodynamic changes.

**DETECTION OF AIR EMBOLISM**

- **Precordial Doppler**: Doppler probes can detect emboli as small as 1 ml.
- **Capnography**: Shows a abrupt fall in end-tidal carbon dioxide concentration as air enters the pulmonary circulation. It is less sensitive but is much easier to use than the Doppler.
- **Transoesophageal echocardiography**: It is the most sensitive method. But it is an invasive method of detecting intracardiac air.

**TREATMENT OF VENOUS AIR EMBOLISM**

The surgical field can be flooded with saline and bone wax applied to the skull edges until the entry site is identified.

Nitrous oxide (if used) should be discontinued.

The central venous catheter should be aspirated in an attempt to retrieve the entrained air.

Intravascular volume infusion should be given to increase central venous pressure.

Vasopressors should be given to treat hypotension.

Bilateral jugular vein compression, by increasing cranial venous pressure, may slow air entrainment and cause back bleeding, which might help the surgeon identify the source of the embolus.

Some clinicians advocate PEEP in an effort to increase cerebral venous pressure; however, reversal of the normal transatrial pressure gradient may promote paradoxical embolism.

If the above measures fail, the patient should be placed in a head-down position and the wound closed quickly.

Persistent circulatory arrest necessitates the supine position and institution of resuscitation efforts using advanced cardiac life support algorithms.

**PREVENTION OF AIR EMBOLISM**

Volume loading to raise the central venous pressure reduces the likelihood of air embolism. Raising end-expiratory pressure may reduce the negative pressure in an open vein in the posterior fossa. However, positive end-expiratory pressure reduces venous return and may cause systemic hypotension. Medical anti-shock trousers or compressive dressing to lower limbs help to reduce venous pooling in the lower limbs.

**PARADOXICAL AIR EMBOLISM**

It can happen by the passage of air across the interatrial septum via a patent foramen ovale. It has a very high risk of major cerebral or coronary morbidity. Although the minimal pressure required to open a probe patent foramen ovale is not known, it is thought that the necessary gradient may be 5 mm Hg. PAE occurs only in the context of major air embolic events, suggesting that significant increases in right heart pressures are an important predisposing factor for the occurrence of PAE. The use of positive end expiratory
pressure was shown to increase the and generous fluid administration was shown to reduce the incidence. Some centers have advocated studies preoperatively with precordial echocardiography to identify patients with a patent foramen ovale.

**TRANSPULMONARY PASSAGE OF AIR**

Air can traverse the pulmonary vascular bed to reach the systemic circulation, which presents as paradoxical air embolism. It is more likely to occur when large volumes of air enters the pulmonary vascular filter.

**PNEUMOCEPHALUS**

The incidence of pneumocephalus is maximum with posterior fossa craniotomies performed with a head-up posture. During these procedures, air may enter the supratentorial space. Depending on the relationship of the brainstem and temporal lobes to the incisura, the pressure in the air collection may or may not be able to equilibrate with atmospheric pressure. This phenomenon has relevance to the use of nitrous oxide because any nitrous oxide that enters a trapped gas space would augment the volume of that space. But usage of nitrous oxide is not absolutely contraindicated because, before dural closure, intracranial gas is not trapped. Nonetheless, attention to this possibility is important when one is presented with the problem of an increasingly “tight” brain during a posterior fossa craniotomy. The use of nitrous oxide up to the point of dural closure may represent a clinical advantage, because the gas pocket can be expected to shrink more rapidly owing to the presence of nitrous oxide because it diffuses out much more quickly than nitrogen after closure. Tension pneumocephalus is one of the causes of delayed awakening after posterior fossa procedures. It occurs because air enters the cranium while the patient is in a head-up position at a time when the volume of the intracranial contents has been reduced as a result of combination of hypocapnia, good venous drainage, osmotic diuresis, and CSF loss from the operative field. When the cranium is closed, and the patient is returned to the supine position, CSF, venous blood volume, and extracellular fluid return or reaccumulate, and the air pocket becomes an unyielding mass lesion. This mass may cause delayed recovery of consciousness or severe headache. The diagnosis of pneumocephalus is confirmed by CT scan. If left untreated, it can result in coning of brain. The treatment is a twist-drill hole followed by needle puncture of the dura.

**MACROGLOSSIA**

After posterior fossa procedures, swelling of pharyngeal structures, including the soft palate, posterior wall, pharynx, and base of the tongue, has been observed especially in sitting position. It is attributed to edema formation following ischemia occurring as the result of foreign bodies (usually oral airways) causing pressure on these structures in the circumstances of lengthy procedures with sustained neck flexion. Incidence of macroglossia is increased, if transesophageal echocardiography is used. So for sitting craniotomy, small diameter probes are used to avoid trauma to pharyngeal and perilaryngeal structures.

**QUADRIPLEGIA**

The sitting position per se has been implicated as a cause of rare instances of unexplained postoperative paraplegia. It is attributed to the neck flexion needed in the sitting position which may result in stretching or compression of the cervical spinal cord. This possibility may represent a relative contraindication to the use of this position in patients with significant degenerative disease of the cervical spine, especially when there is evidence of
associated cerebrovascular disease. In such patients, somatosensory evoked response monitoring during the positioning phase of a sitting procedure becomes essential. Surgery near the roots of nerves VII–X may lead to loss of airway reflexes, dysphagia and dysphonia. Rarely, recurrent laryngeal nerve is injured by extreme neck flexion. The most common peripheral nerve injured is common peroneal nerve resulting in foot drop. It is caused by tight wrapping of bandages around the knee.

REFERENCES:
1 Voorhes RM, Fraser AR, Van Poznak. Prevention of air embolism with positive end expiratory pressure. Neurosurgery 1983; 12;503-6


3. Lange Anesthesiology Section IV. Physiology, Pathophysiology, & Anesthetic Management; Chapter 26. Anesthesia for Neurosurgery


7 Philippa Newfield, James E. Cottrell Handbook of neuroanaesthesia


9 Michenfelder WK, Miller RH, Gronert GA The evaluation of Doppler