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REVIEW ARTICLE

The sitting position in neurosurgery: a critical appraisal

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The use of the sitting or upright position for patients undergoing posterior fossa and cervical spine surgery facilitates surgical access but presents unique physiological challenges for the anaesthetist with the potential for serious complications.[2] This patient position provides optimum access to midline lesions, improves cerebral venous decompression, lowers intracranial pressure (ICP) and promotes gravity drainage of blood and cerebral spinal fluid (CSF).[12] Complications related to the use of this position include haemodynamic instability, venous air embolism (VAE) with the possibility of paradoxical air embolism, pneumocephalus, quadriplegia and compressive peripheral neuropathy.[73] Alternative positions for surgical access to the posterior fossa and the cervical spine include the prone and lateral positions. Prolonged neurosurgical procedures with pin fixation of the head in abnormal positions necessitate extensive patient monitoring to ensure cardiorespiratory homeostasis.

Historical milestones in the adoption of the sitting position in neurosurgical practice were highlighted by Albin and colleagues.[2] The sitting position today is as controversial as when first introduced into clinical practice in 1913 by De Martel.[23] The first reported use of this position was for brain tumour surgery performed under local anaesthesia. Frazier and Gardner reported the use of this position for surgery on the Gasserian ganglion in the USA in 1928.[30] The advent of the operating microscope in neurosurgical practice, new inhalation anaesthetic agents and neuromuscular blocking drugs, and sophisticated cardiovascular and respiratory monitoring equipment facilitated the development of more complicated and technically challenging procedures performed in the sitting position.[82]

Although there have been several studies substantiating the relative safety of the sitting position for neurosurgery, its use remains controversial and appears to be diminishing because of the potential for serious complications and malpractice liability claims. This decline has been observed both in the UK and USA, and appears to be related to successful litigation for neurological consequences after paradoxical air embolism (Michenfelder JD, personal communication). Campkin[17] reported that 19 (53%) of 36 UK neurosurgical centres surveyed in 1981 used the sitting position for posterior fossa surgery and 11 (30%) for cervical spinal surgery. Elton and Howell,[26] based on a postal survey of UK neurosurgical centres, claimed a greater than 50% reduction in the number of neurosurgical centres using the sitting position during 1981–1991. In 1991, patients were normally placed in the sitting position for posterior fossa surgery in eight (20%) of the UK centres surveyed compared with 19 (53%) in 1981. Black and colleagues also reported a major change from the sitting to the horizontal position for patients undergoing posterior fossa craniotomies over the 4-yr period from 1981 to 1984 at the Mayo Clinic.[14] Posterior fossa craniotomies performed in the sitting position in that institution declined from over 110 per year in the early 1980s to less than 50 by the mid-decade.

The objective of this review is to provide a risk–benefit analysis of the present day use of the sitting position for patients undergoing posterior fossa and cervical spine surgery.

Surgical considerations

The 1960s and 1970s were the heyday for the popularity of the sitting position for surgical procedures involving the cervicodorsal spine, and posterior and lateral cranial fossae. A four-part series of review articles outlining patient management for these procedures at the Mayo Clinic were featured in *Anesthesia and Analgesia*.[53–55]

Accumulated blood drains out of and away from the operative site in the sitting position. This allows more rapid access to bleeding points, a cleaner surgical field and a

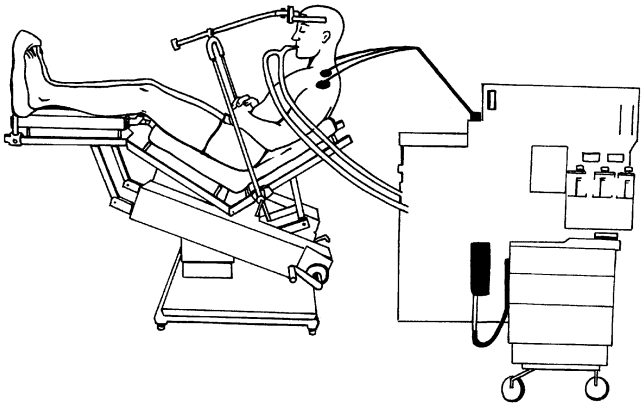

Fig 1 The sitting position for neurosurgery.

technically easier procedure than is possible in the prone position.[34] In addition, the sitting position provides an unobstructed view of the patient's face, permitting observation of motor responses to cranial nerve stimulation. In some procedures, notably supracerebellar, infratentorial approaches to the pineal gland, the sitting position minimizes the amount of cerebellar retraction needed to gain access to deeper structures.

Suitable head holders for the sitting position remove the hazard of pressure on the orbit posed by horseshoe-shaped face pieces, still used occasionally for prone position surgery. Because there is access to the anterior chest wall, resuscitative measures may be instituted rapidly if the patient experiences acute cardiovascular collapse (Fig. 1).

Physiological changes

Use of the operative sitting position is associated with several characteristic physiological changes which are outlined below.

Cardiovascular system

The haemodynamic effects of anaesthesia in the sitting position may be influenced by the choice of ventilatory technique. Spontaneous ventilation with a potent inhalation anaesthetic agent in nitrous oxide–oxygen was popular in the 1960s to provide signs of surgical encroachment on vital medullary and pontine structures.[2] In a retrospective, unblinded study not subjected to statistical analysis, Millar reported lower systolic arterial pressure (SAP) and greater vasopressor requirements when intermittent positive pressure ventilation, rather than spontaneous ventilation, was used.[60]

The problems of cardiovascular instability and arterial hypotension associated with the upright position may be aggravated by the depressant effects of i.v. induction and volatile agents on myocardial contractility during general anaesthesia and changes in venous return after intermittent positive pressure ventilation. The hydrostatic effect of gravity not only permits drainage of blood and cerebrospinal fluid away from the surgical field, but may also produce a decrease in systemic arterial pressure because of venous pooling in the lower extremities.[60] The volume of blood accumulating in the venous system may be influenced by patient factors (i.e. body mass index, intravascular volume status, pre-existing hypertension) and factors related to anaesthesia (e.g. mode of ventilation). As much as 1500 ml may be sequestered in the venous system of the lower limbs because of the effect of gravity[58] and increased diffusion through the capillary walls and venous dilatation associated with the use of potent inhalation anaesthetic agents.[10]

The incidence of hypotension reported in association with the sitting position has varied, depending on definition, in absolute and relative terms (Table 1).

Hypotension, defined as a reduction in systolic arterial pressure of 10 mm Hg or greater, was reported in 10% of patients during induction of anaesthesia and positioning.[83] One patient in this series, who experienced a decrease in mean arterial pressure (MAP) of 30 mm Hg, subsequently sustained silent myocardial infarction. Hypotension in this study was not related to ASA status. In contrast, Albin and colleagues reported a 32% incidence of a 10–20 mm Hg reduction in SAP in the sitting position in a series of 180 patients undergoing neurosurgical procedures. A relationship between ASA status and incidence of hypotension was noted in this study.[2]

Black and colleagues defined hypotension as a decrease of 20% or more in SAP. These investigators observed similar reductions in SAP when patients were placed in the horizontal and sitting positions.[14] The extent of SAP changes induced by patient positioning are influenced by study method, technique of patient positioning and use of ancillary equipment to minimize gravitational effects on intravascular volume. Positioning of the patient with flexion of the hips, elevation of the knees to the level of the heart and wrapping of the lower extremities from the toes to the groin has also been shown to minimize patient position-associated hypotension.[52] The addition of lower extremity compression bandages was associated with a 5% incidence of a 20% reduction in SAP in one study.[57] In four of these patients, morbidity or mortality was possibly related to intraoperative hypotension.

Several studies have evaluated the haemodynamic changes associated with patient transfer from the supine to the sitting position. Ward and colleagues[79] reported a 3% decrease in MAP in healthy subjects when changed from the supine to the sitting position. Heart rate and systemic vascular resistance (SVR) increased by 19% and 10%, respectively, while stroke volume (SV) and cardiac index (CI) decreased by 21% and 10%.

The extent of head-up tilt influences haemodynamic changes. Albin and colleagues, in a series of awake and anaesthetized patients, observed clinically insignificant haemodynamic changes imposed by 45° head-up tilt.[3] One hour after placement in the sitting position at an angle of 90°, MAP increased by 38%, SVR increased by 80%, while a reduction of 20% in CI was noted.

Table 1 Incidence of hypotension after induction of anaesthesia and patient positioning

Ref. (year of publication)	Author(s)	No. of patients	Definition of hypotension	Incidence (%)
1976[2]	Albin *et al.*	180	>10% decrease in MAP	32
1985[57]	Matjasko *et al.*	554	20% decrease in SAP	5
1986[83]	Young *et al.*	225	>10 mm Hg decrease in MAP	12
1988[14]	Black *et al.*	333	>20% decrease in SAP	19

Table 2 Cardiovascular changes after patient positioning and general anaesthesia (data from studies cited in text[2 3 52 79])

Mean arterial pressure	Decreased
Systolic arterial pressure	Decreased
Heart rate	Increased
Stroke volume index	Decreased
Cardiac index	Decreased
Pulmonary capillary wedge pressure	Decreased
Systemic vascular resistance	Increased

Intravascular volume status and surgical stimulation may attenuate adverse haemodynamic changes associated with patient position. When conscious subjects were placed in the seated position, the following physiological changes were observed: heart rate and SVR increased by 12%, resulting in an 11% increase in MAP; stroke volume indices and PCWP simultaneously decreased by 22% and 11%, respectively. Choice of anaesthetic agent and technique may have a significant influence on cardiovascular performance in sitting patients.[52] All patients in this study were subsequently anaesthetized with thiopental (thiopentone) 3 mg kg^{-1}; tracheal intubation was facilitated with pancuronium 0.1 mg kg^{-1}. Anaesthesia was maintained with 60% nitrous oxide in oxygen and supplemented with one of four regimens. Patients were allocated randomly to one of four groups of six subjects according to the anaesthetic technique used: 0.75% end-tidal enflurane; 0.4% end-tidal halothane; Innovar 0.1 ml kg^{-1} and morphine 0.5 mg kg^{-1}. The morphine–nitrous oxide technique resulted in least impairment of cardiovascular performance when patients were placed in the seated position before surgical stimulation. Cardiovascular homeostasis was disturbed more after induction of anaesthesia with thiopental 3 mg kg^{-1} and pancuronium 0.1 mg kg^{-1} than by patient positioning. Surgical stimulation resulted in a 15% increase in SAP and a 62% increase in SVR while CI remained 20% below baseline values in this series of 24 healthy patients undergoing elective neurosurgical procedures.[52]

The effects of patient position and anaesthesia on right and left heart filling pressure have been investigated using pulmonary artery flotation catheters (PAC)[66] (Table 2).

Placement of the patient in the seated position, when awake, resulted in a significant decrease in pulmonary capillary wedge pressure (PCWP) but no change in right atrial pressure (RAP). Similar changes in PCWP and RAP were found after induction of anaesthesia in the supine position. However, anaesthesia in the seated position significantly reduced PCWP 1 h after incision. PCWP decreased further and RAP then exceeded PCWP. The investigators attributed this acute reduction in PCWP when patients were placed in the seated position to a decrease in pulmonary blood volume. Unchanged mean pulmonary pressure with postural changes was thought to reflect the considerable compliance of the pulmonary circulation. However, the pulmonary artery catheter tip might become lodged in a lung region where zone 1 conditions prevail (alveolar pressure exceeding pulmonary artery and venous pressure).[80] In such a situation, PCWP reflects airway pressure rather than left atrial pressure (LAP). If, however, the normal left to right inter-atrial pressure gradient becomes reversed during anaesthesia in the sitting position, the neurosurgical patient is predisposed to the risk of paradoxical air embolism.

Compensatory mechanisms, perhaps mediated by the renin–angiotensin–aldosterone system or the sympathetic nervous system, may be operative in the awake or anaesthetized state to attenuate adverse haemodynamic changes associated with the sitting position. In unconscious humans, the renin–angiotensin system plays an important role in preventing postural hypotension when the upright position is attained[63] Plasma renin activity was measured in patients in the supine position and also when seated for 5 min while awake and again after induction of anaesthesia.[52] As patients in this study had been fasting for 8–15 h, concentrations of plasma renin activity varied widely, both in the conscious and anaesthetized states. These wide variations precluded finding statistically significant changes in plasma renin activity after patients were placed in the seated position. Also, wrapping of the legs and positioning of the knees at right heart level, as practised in this study, may have contributed to the lack of significant changes in renin release which remained constant after induction of anaesthesia or change in position, but increased with surgical stimulation.

Respiratory system

The early postoperative period is universally characterized by small airway closure and a reduction in functional residual capacity (FRC) associated with a decrease in arterial oxygen saturation. Adoption of the operative sitting position results in an increase in FRC but the associated reduction in perfusion may obviate the expected benefits in oxygenation. Among the advantages claimed by proponents of the operative sitting position is superior access to the chest wall and airway in this position. Ventilation is unimpeded as diaphragmatic excursion is greater than in

the horizontal position and consequently airway pressures are lower.[2 10]

Lung function has been studied in awake and anaesthetized subjects in different postures. Lumb and Nunn found that lung function test values obtained in standing subjects correlated with data obtained from the same subjects in the sitting position.[48] Forced expiratory volume in 1 s ($FEV_{1)}$ was unchanged while FRC and forced vital capacity (FVC) increased in the seated patients. A significant reduction in arterial oxygen tension (Pa_{O_2}), associated with a decrease in cardiac output, was documented in anaesthetized subjects on changing from supine to sitting.[22] Arterial carbon dioxide tension Pa_{CO_2} was unchanged, suggesting physiological deadspace did not increase significantly. Similarly, Albin and colleagues noted a decrease in oxygen transport (the product of cardiac output and arterial oxygen content) after transfer to the sitting position.[3] Courington and Little, based on an extensive literature review, concluded that the sitting position offered least restriction to movement of the diaphragm, ribs and sternum.[18]

Rehder, Sessler and Rodarte studied regional intrapulmonary gas distribution in awake and anaesthetized subjects. The sitting position in anaesthetized and paralysed subjects was associated with minimal impairment of FRC compared with the supine or lateral decubitus position. The distribution of ventilation was less uniform in anaesthetized than awake subjects in the sitting position but this improved with increasing tidal volumes.[68]

Hunter, as early as 1962, claimed that intermittent positive pressure ventilation increased the risk of VAE[42] but subsequent investigators have not substantiated this claim.[12] The sole advantage of spontaneous respiration in this setting was the provision of an index of surgical encroachment on medullary centres[2]; the disadvantages included hypercapnia and the possibility of venous engorgement of the operative field.[6] Controlled ventilation allows the provision of light anaesthesia, without the risks of coughing and straining, and without hypotension associated with high volatile anaesthetic concentrations. Electrocardiographic changes have been shown to provide an equally sensitive index of medullary stimulation, and modern monitoring, including beat-to-beat arterial pressure measurement, allows early detection of brainstem compromise.

The incidence of postoperative pulmonary dysfunction does not appear to relate to the operative sitting position. Black and colleagues noted a similar incidence of respiratory complications in patients in whom surgery was performed in the sitting and supine positions (2% and 3%, respectively).[14] In addition, the occurrence of VAE was not associated with an increased incidence of pulmonary dysfunction. Standefer, Bay and Trusso noted a similarly low incidence of postoperative complications (2.5%).[73] Young and colleagues observed that postoperative pulmonary dysfunction was more likely in patients with pre-existing lung disease or after pulmonary aspiration in the postoperative period.[83]

Table 3 Factors influencing cerebral perfusion pressure (CPP)

Mean cerebral arterial pressure
Mean arterial pressure
Cerebral venous pressure
Central venous pressure
Intracranial pressure (dura intact)
Surgical retraction

Cerebral perfusion and intracranial pressure

Inadequate cerebral perfusion after placement of anaesthetized patients in the sitting position is a well recognized hazard. Global cerebral blood flow (CBF) ranges from 45 to 55 ml 100 g^{-1} min^{-1}.[25] At rest, the brain consumes approximately 3.5 ml of oxygen per 100 g of brain tissue per minute. Autoregulation refers to the intrinsic capacity of the cerebral circulation to adjust its resistance to maintain CBF constant over a wide range of MAP values. In normal subjects, the limits of autoregulation are approximately 50 and 130 mm Hg. Above and below this range, CBF is pressure-dependent and varies linearly with cerebral perfusion pressure (CPP). CPP is determined by the difference between mean cerebral arterial pressure and mean central venous pressure (Table 3).

In the supine position, MAP approximates cerebral arterial pressure while cerebral venous pressure approximates intracranial pressure.[62]

Several factors produce changes in cerebral vascular resistance which influences cerebral blood flow. These include changes in cerebral metabolic rate for oxygen ($CMRO_2$), Pa_{CO_2} and Pa_{O_2}. Intracranial pressure is determined by compliance of the cerebral spinal fluid space and the resistance to CSF absorption. When the dura is opened, ICP decreases to zero and MAP becomes the principal determinant of CPP. However, surgical stimulation may produce local increases in ICP and consequently cerebral ischaemia in the setting of reduced arterial pressure. Positioning of the arterial pressure transducer at the level of the mid-cerebrum allows estimation of CPP.

The sitting position is associated with a reduction in arterial and venous pressures as a result of the gravitational effect of positioning the head above heart level. Arterial pressure has been reported to be reduced by 0.77 mm Hg for each centimetre gradient above the heart.[32] The extent of mean arterial and intracranial pressure changes depends on patient factors and the anaesthetic technique chosen. Finnerty, Witkin and Fazekas demonstrated symptoms and signs of cerebral ischaemia in awake subjects when cerebral blood flow was reduced by 42% from control levels.[29] Scheinberg and Stead noted a 21% reduction in cerebral blood flow in awake subjects who were tilted 65° head-up from the horizontal.[69]

The anaesthetic technique chosen may influence the potential for cerebral ischaemia. Tindall, Craddock and Greenfield cannulated the common carotid artery in several patients undergoing anaesthesia and surgery in the sitting

Table 4 Incidence of venous air embolism (VAE) in the sitting position

Ref. (year of publication)	Author(s)	No. of patients	Incidence of VAE (%)	Monitoring technique
1972[59]	Michenfelder, Miller, Gronert	69	32	Doppler
1976[2]	Albin *et al*	180	25	Doppler
1983[78]	Voorhies, Fraser, Van Poznak	81	50	Doppler
1984[73]	Standefer, Bay, Trusso	382	7	Doppler
1985[57]	Matjasko *et al*	554	23	Doppler
1988[14]	Black *et al*	333	45	Doppler
1994[65]	Papadopoulos *et al*	62	76	Transoesophageal echocardiography

position and used an electromagnetic flowmeter to continuously measure blood flow.[76] General anaesthesia and induced hypocapnia reduced flow by 34% in the supine position. Assumption of the sitting position under anaesthesia further reduced flow by 14%. The authors attributed the reduction in cerebral blood flow to a reduction in effective arterial pressure. The consequence of such a reduction in cerebral blood flow may be offset by the reduction in $CMRO_2$ and consequent lowering of the ischaemic threshold associated with anaesthesia.[39] In addition, surgical stimulation attenuated the reduction in cerebral blood flow and internal carotid flow.

Complications

Venous air embolism

Venous air entrainment is a positional hazard confronting patients placed in the sitting position for cervical spine or posterior fossa surgery and patients placed with significant head-up tilt for thyroid and head-neck surgery. Conditions favouring venous air entrainment include an open vein, gravitational effect of low central venous pressure and negative i.v. pressure relative to atmospheric pressure and poor surgical technique. These conditions may be encountered in neurosurgical practice with head elevation to promote venous drainage and to optimize surgical access. The vertical distance between the head and heart may range from 20 to 65 cm, depending on the procedure.[1]

The incidence of VAE is difficult to quantify because of significant differences in the sensitivities of various monitoring modalities used and the clinical significance of the findings (Table 4). The incidence has ranged from 25%[2] to 50%[78] in studies using praecordial Doppler monitoring. Other investigators, using the more sensitive transoesophageal echocardiography (TOE) monitoring, have indicated an incidence as high as 76%.[65]

The first fatality as a result of VAE in association with surgery in the sitting position was recorded as early as 1830.[9] The complication occurred during a procedure to remove a facial tumour. Fifty years later, Dr N. Senn from Milwaukee, Wisconsin, felt compelled to draw the attention of readers to VAE, 'one of the most uncontrollable causes of sudden death'.[70] He performed an exhaustive search of the literature available at that time and a series of animal experimental studies involving injection of various quantities of air into the jugular vein. Dr Senn concluded that VAE produces death by 'mechanical overdistension of the right ventricle of the heart.....and asphyxia from obstruction to the pulmonary circulation consequent upon embolism of the pulmonary artery'. To treat VAE, he proposed 'catheterization and aspiration of the right auricle.....thus relieving the overdistension of the right ventricle, and, at the same time, to guard against a fatal embolism of the pulmonary artery'.

Table 5 Monitoring of venous air embolism (VAE); VAE detection techniques in order of decreasing sensitivity

Monitor	Associated clinical signs
Transoesophageal echocardiography	Absent
Praecordial Doppler	Absent
Pulmonary artery pressure	Minor (heart rate, MAP)
End-tidal carbon dioxide	Minor (heart rate, MAP)
Right atrial pressure	Significant
Electrocardiography	Cardiovascular collapse
Oesophageal stethoscope	Cardiovascular collapse

While VAE has been described in association with a wide variety of surgical procedures and positions, it remains the most feared complication of the operative sitting position. Monitoring is therefore directed towards detection and treatment of VAE. The sensitivities of the techniques in detecting intracardiac air, in the absence of measurable cardiopulmonary changes, are variable. Monitoring techniques include praecordial Doppler, right heart catheters, transoesophageal echocardiography, fractional excretion of nitrogen ($F_{E_{N_2}}$), capnography, oesophageal stethoscope and transcutaneous oxygen measurement (Table 5). These techniques are described in detail (see below).

The gradient between the atria is a factor in the pathophysiology of paradoxical air embolism (PAE). Conditions which increase RAP relative to LAP may predispose to PAE when VAE occurs.[66] Placement of patients in the seated position, in some cases, has been shown to result in an RAP greater than PCWP. Application of positive end-expiratory pressure (PEEP) may also increase RAP sufficiently to exceed PCWP.[66] At one time PEEP was advocated to prevent and treat VAE.[78] However, both application and

release of PEEP have been associated with right to left shunting (presumably because of increased RAP and increased right heart venous return, respectively).[44] The use of PEEP is generally not recommended during procedures performed in the sitting position because of the possible increased risk of PAE and inconclusive data concerning its efficacy.

Pneumocephalus

Tension pneumocephalus may follow air entry into the epidural or dural spaces in sufficient volumes to exert a mass effect with the potential for life-threatening brain herniation.[7 28] This complication has been described in association with posterior fossa exploration in the sitting position,[46 77] with an incidence of 3% in one large study.[73]

The development of tension pneumocephalus is a serious and life-threatening emergency.[75] This complication has been attributed to diminution of brain volume secondary to mannitol administration, hyperventilation, removal of a space-occupying mass and contraction of intravascular blood volume associated with acute haemorrhage.[31] Intraoperative drainage of CSF either via a ventriculostomy, ventricular incision or subarachnoid drainage may result in further ventricular collapse. The gravitational effect of the sitting position may increase the likelihood of air entry into the subdural space as CSF leaks through the incision site. Lunsford and colleagues has proposed the 'inverted pop bottle' analogy to describe this phenomenon.[49] As fluid (CSF) pours out, air bubbles to the top of the container (cranium).

Several case reports have highlighted the risk of tension pneumocephalus in patients with hydrocephalus.[11 36 43 47 61 67] Patients who have undergone CSF shunting or drainage procedures for the treatment of hydrocephalus are at increased risk of subdural pneumocephalus if free gravity drainage of CSF is permitted while the cranial vault is open.[36] The cerebral mantle is thin and easily collapsible in patients with marked hydrocephalus. When subjected to negative pressure influences, an opening of the skull outside the cerebral mantle results in excessive CSF drainage and collapse of the ventricles. The slow but continuous gravitational drainage of CSF which occurs in the sitting position likewise can result in accumulation of air in the subdural space.

The role of nitrous oxide in the pathogenesis of tension pneumocephalus remains controversial. Nitrous oxide was implicated as a major contributory factor in the earlier reports of tension pneumocephalus.[11 46 49] However, avoidance of nitrous oxide has not eliminated the problem. [64 77] Nitrous oxide diffuses into an air filled cavity more rapidly than nitrogen diffuses into blood and thus the size of any air-filled space or embolus increases. Friedman, Norfleet and Bedford questioned the role of nitrous oxide in the development of tension pneumocephalus after sitting posterior fossa craniotomy because nitrous oxide was discontinued 30 min before dural closure.[31] Based on his clinical observations, he recommended that nitrous oxide administration should be continued until after dural closure so that an intraoperative pneumocephalus would contain nitrous oxide and would be reabsorbed rapidly after its discontinuation.

Pandit and colleagues evaluated the contribution of nitrous oxide to pneumocephalus formation in a prospective study of five patients undergoing posterior fossa exploration in the sitting position.[64] ICP was monitored after closure of the dura. Nitrous oxide was added to the breathing mixture only after dural closure. Administration of nitrous oxide was associated with a two-fold increase in intracranial pressure, suggesting the presence of intradural air. Intracranial pressure elevation subsided when nitrous oxide was discontinued. Intracranial hypertensive effects were not associated with nitrous oxide administered throughout the case in the two other patients studied, presumably because nitrous oxide in blood had already equilibrated with intracranial gas. Computerized tomography (CT) scans performed 24 h after surgery revealed varying volumes of intracranial air in all cases. The authors concluded that air invariably enters the cranium during craniotomy in the sitting position.

In a prospective study of 30 patients, all were noted to have pneumocephalus after posterior fossa surgery in the sitting position.[24] Thus pneumocephalus is an invariable consequence of intracranial surgery. However, Toung and colleagues reported four cases of tension pneumocephalus complicating posterior fossa exploration in the sitting position where nitrous oxide was not used and in one case where it was discontinued 20 min before dural closure.[77]

A variety of strategies have been adopted to reduce the potential for pneumocephalus after surgical procedures in the sitting position. Flushing the subdural space with normal saline has been recommended but may be an impractical suggestion.[31] Discontinuation of nitrous oxide before dural closure has been recommended but may be ineffective. Efforts to minimize intracranial dehydration secondary to diuretics have been suggested.[75] Adjustment of minute ventilation and avoidance of hyperventilation may facilitate brain expansion as the dura is closed. Ventriculostomy drainage of CSF after major posterior fossa procedures is commonplace. Nitrous oxide should be avoided if surgical re-exploration becomes necessary during the first 14 days after operation because intracranial air is absorbed only slowly.[64 75]

A high index of suspicion is required to ensure prompt diagnosis of tension pneumocephalus after craniotomy. Suggestive clinical features include confusion, headache, convulsions, neurological deficit and failure to regain consciousness. The differential diagnosis of tension pneumocephalus should be considered after procedures performed in the sitting position, in association with extensive brain tumours or hydrocephalus, and in the context of nitrous oxide administration. Early CT scan enables diagnosis and localization of intracranial air or other mass lesions.

Untreated tension pneumocephalus may result in brain

herniation and death, and therefore rapid therapeutic intervention is warranted. Cardiac arrest has been reported in association with this complication.[46] Immediate twist drill aspiration of air through burr holes on either side of the vertex is indicated if a tension pneumocephalus is diagnosed.[77] Rapid evacuation of air should ensure prompt recovery.

Macroglossia

Extreme flexion of the head with the chin resting on the chest and the prolonged presence of an oral airway may promote obstruction of venous and lymphatic drainage of the tongue after procedures performed in the sitting position. Postoperative macroglossia has the potential to produce airway obstruction, hypoxaemia and hypercapnia. Infants may be at particular risk because of the high anterior larynx, small tracheal diameter and relatively large tongue.

Reports of macroglossia after procedures performed in the sitting position highlight swelling of other pharyngeal structures, including the soft palate, posterior pharyngeal wall and tongue.[56] Sustained neck flexion, the use of oral airways and long duration of surgery have all been associated with this condition. Several strategies have been advocated to reduce the possibility of this complication. The oropharyngeal airway may be withdrawn until the tip functions as a bite block when the patient has been positioned.[56] Small diameter transoesophageal echocardiographic probes have been recommended when this monitoring modality is used to avoid trauma to pharyngeal and peri-laryngeal structures.

Quadriplegia

There have been rare, occasional reports of paraplegia in the postoperative period after procedures performed in the sitting position. Hitselberger and House, in an editorial comment in *Archives of Otolaryngology*, referred to five unreported cases of mid-cervical quadriplegia after acoustic neuroma resection performed in the sitting position.[41] Focal pressure on the spinal cord, in conjunction with flexion of the head on the neck, was the postulated mechanism. Wilder claimed knowledge of more than 20 such unreported cases.[81] More recently, Matjasko and colleagues reported a case of quadriparesis associated with the operative sitting position in a patient with severe cervical stenosis.[57] Young and colleagues reported a case of triparesis after resection of an acoustic neuroma in a patient who had a documented episode of VAE.[83] Wilder proposed that acute flexion of the neck in an anaesthetized patient in the sitting position may stretch the cord at the level of the fifth cervical vertebra.[81] Regional cord perfusion may be compromised, especially if mean arterial pressure is reduced. More recently, Ernst, Albin and Bunegin studied an animal model and demonstrated a decrease in spinal cord blood flow in subjects in the sitting position with experimentally induced intracranial hypertension.[27] The decrease in CBF consequent on assumption of the sitting position was not accompanied by a decrease in $CMRO_2$.

Somatosensory evoked potential monitoring has been proposed as an indicator of the adequacy of regional spinal cord perfusion in these cases.[81] The sitting position may be relatively contraindicated in patients with degenerative disease of the cervical spine as spondylolistheses may produce compromised local spinal cord perfusion.[41] Furthermore, the sitting position may be potentially hazardous in patients with significant cerebrovascular disease.[12]

Neurological sequelae—peripheral nerve injuries

Several peripheral nerve injuries have been reported in association with the sitting position for neurosurgery. These include damage to the common peroneal nerve, resulting in foot drop, and less commonly, recurrent laryngeal nerve palsy.

The association between the operative sitting position for neurosurgical procedures and common peroneal neuropathy was first noted by Keykhah and Rosenberg, who described a case of bilateral foot drop after posterior fossa exploration.[45] The incidence of this complication in a series of 488 patients was less than 1%.[73] All four patients in this series sustained a common peroneal nerve injury. None of the patients had identifiable predisposing factors (e.g. diabetes mellitus, vascular disease or peripheral neuropathy).

A possible pathogenesis of common peroneal palsy after procedures performed in the sitting position was advanced by Garland and Moorhouse, based on the experience of 20 such cases.[33] Local nerve ischaemia may develop secondary to nerve compression as it courses around the neck of the fibula. Nerve injury resulting from bandages wrapped around the knee carried the poorest prognosis with respect to recovery of nerve function.[45] A common peroneal palsy may also occur as a result of injury to the peroneal division of the sciatic nerve. It has been suggested that foot drop after neurosurgery in the sitting position may be related to hyperflexion of the thigh and consequent compression or stretching of the sciatic nerve.[23 27]

Recurrent laryngeal nerve palsy has been described in association with the use of transoesophageal echocardiography in two of a series of 15 patients who underwent craniotomy in the sitting position.[20] The authors postulated that the rigidity and large size of the probe combined with neck flexion and tracheal intubation were contributing factors.

Recurrent laryngeal nerve palsy has been described in association with tracheal intubation,[38] and direct compression of the nerve endings between the cuff of the tube and the tracheal rings was a suggested mechanism. Irregular or over-inflation of the cuff or positioning of the cuff just below the vocal cords may be implicated. Nitrous oxide distension of an air containing cuff may also be contributory. Individual anatomical variation such as the presence of a cervical rib, size of the cervico-axillary canal, shape of the first rib and slope of the shoulder may all play a part in

Table 6 Contraindications to use of the operative sitting position

Absolute
Patent ventriculo-atrial shunt
Right atrial pressure in excess of left atrial pressure
Patent foramen ovale
Cerebral ischaemia when upright and awake
Relative
Extremes of age
Uncontrolled hypertension
Chronic obstructive airways disease

Table 7 Summary of anaesthetic management

History/physical examination	
(to determine absolute/relative contraindications)	
Age >70 yr	
Untreated hypertension	
Chronic obstructive lung disease	
Cerebrovascular disease	
Echocardiography	
(to determine presence of patent foramen ovale)	
Anaesthetic management	
Induction	
Fentanyl 2–5 μg kg^{-1}	
Lidocaine 1 mg kg^{-1}	
Thiopental 3–5 mg kg^{-1} or etomidate 0.2–0.3 mg kg^{-1}	
Vecuronium 0.2 mg kg^{-1}	
Maintenance	
O_2–air and 1–1.5% isoflurane	
Incremental bolus fentanyl	
or	
Propofol target-controlled infusion	
Ventilation	
Intermittent positive pressure ventilation–low-normal $PaCO_2$	
Insertion of pulmonary artery flotation catheter	
Inflation of anti-gravity suit	
Slow stage patient positioning	
Placement of praecordial Doppler probes	
Placement of transducers at heart level	
Monitoring	
General	Electrocardiogram
	Non-invasive arterial pressure
	Temperature
	Neuromuscular function
	Urine output
Specific	End-tidal CO_2
	Praecordial Doppler
	Oesophageal stethoscope
	Pulmonary artery pressure
Postoperative evaluation	
Upper airway assessment/cranial nerve function	

rendering nerves which have a cervico-thoracic course vulnerable to stretch injury.[8] This is compounded further by neck flexion in the sitting position.

Contraindications

Black and Cucchiara suggest several conditions which may warrant avoidance of the operative sitting position (Table 6). They note, however, that there are few objective data to support this suggestion.[12]

Certain pre-existing conditions may place patients at increased risk of vascular air embolism (i.e. presence of a patent ventriculo-atrial shunt, demonstrable pressure gradient from the right to the left heart or presence of a patent foramen ovale). Patients who experience cerebral ischaemia whenever they assume the upright position as a result of cardiovascular and/or cerebrovascular disease are at increased risk of inadequate cerebral perfusion under anaesthesia in the operative sitting position. Relative contraindications may include extremes of age, uncontrolled hypertension or chronic obstructive airways disease.

Anaesthetic management

Preoperative evaluation

The objectives of preoperative evaluation are to use the history and physical examination to identify absolute and relative contraindications to the sitting position (i.e. age >70 yr; untreated hypertension; chronic obstructive lung disease; cerebrovascular disease and transthoracic echocardiographic-detected patent foramen ovale). There are no outcome data to support the claim of superiority of either the sitting or horizontal position.[14] Assessment of the risk–benefit ratio, based on individual physical status and the surgical implications of the particular intracranial pathology, is of paramount importance. Cardiovascular and cerebrovascular status should be assessed before operation and therapy optimized. The presence of intracranial hypertension has implications for the anaesthetic technique used. A summary of the anaesthetic management used by the authors is included in Table 7.

Preoperative contrast echocardiography may be used as a screening technique to detect the population at risk of PAE as a result of the presence of a patent foramen ovale (PFO). Praecordial or transoesophageal echocardiography involves i.v. injection of saline agitated with air and application and release of a Valsalva manoeuvre.[21] The overall incidence of asymptomatic probe-patent PFO in autopsy specimens has been reported to be as high as 27.3%.[37] The mean diameter of the defect was 5–6 mm and this tended to increase with increasing age.

Black and colleagues noted that the incidence of PFO on echocardiography, in a series of 101 neurosurgical patients, was less than expected in the general population.[13] A PAE complication occurred in one patient in the absence of PFO on preoperative testing. The authors concluded that the usefulness of preoperative echocardiography as a screening test may be limited. This finding was corroborated by a study in which universal preoperative screening for the presence of a PFO with TOE failed to identify two patients in whom air was found in the left heart during operation.[65] However, the preoperative identification of a PFO has major implications for subsequent patient management.

Anaesthetic technique

Specific aspects of the anaesthetic technique appropriate for patients undergoing neurosurgical procedures performed in the sitting position are well established.[52] Thiopental 3–5 mg kg^{-1} and etomidate 0.2–0.3 mg kg^{-1} supplemented with an opioid such as fentanyl 1–2 μg kg^{-1} have been

advocated to induce anaesthesia, and a non-depolarizing neuromuscular blocking agent such as vecuronium can be used to facilitate tracheal intubation. Adjustment of fresh gas flow and intermittent positive pressure ventilation are used to ensure normocapnia. Pharmacological measures (lidocaine/beta blockers) may be used to attenuate hypertensive responses to laryngoscopy and intubation in this patient population. Maintenance of anaesthesia include air–oxygen supplemented with potent inhalation agents and incremental opioid administration. Nitrous oxide has been implicated as a major contributory factor in reports of tension pneumocephalus which preclude its use in this patient population. Alternatively, a propofol-based total i.v. anaesthesia or target-controlled infusion technique should have some beneficial effects, including the ability to monitor facial nerve function, without supplementary administration of neuromuscular blocking agent, and possibly enhanced haemodynamic stability.

Measures to minimize hypotension include slow staged positioning over a 20-min period and the use of neuroleptanaesthetic agents. Gardner and Dohn devised an easily applied 'G-suit' (anti-gravity suit) in 1956 to obtund extreme haemodynamic responses to postural changes.[55] This consisted of a double layer of heavy plastic which was wrapped around the patient and inflated with compressed air to a pressure of 30 mm Hg. Based on limited clinical evaluation, Martin recommended its use in patients undergoing neurosurgical procedures in the sitting position to prevent pooling of blood in the dependant lower extremities and pelvis.[53] However, inflation of the suit to 30 mm Hg for prolonged periods failed to maintain venous pressure and may therefore be ineffective as a prophylaxis against VAE.

The decision to reverse neuromuscular block and extubate the trachea at the end of surgery is influenced by the extent and severity of the surgical insult, preoperative neurological status, duration of surgery, extent of intraoperative blood loss, and patient factors such as haemodynamic stability and relative normothermia. As posterior fossa surgery may be complicated by cranial nerve palsies and recurrent laryngeal nerve palsy, careful assessment of cranial nerve function would be prudent before the trachea is extubated at the end of surgery.

Monitoring

Standard intraoperative monitoring for patients undergoing complex neurosurgical procedures in the sitting position include the electrocardiogram to assess heart rate and rhythm, non-invasive arterial pressure monitoring, oesophageal stethoscope to auscultate heart and breath sounds, neuromuscular function monitoring and a catheter to quantify hourly urine output. Invasive arterial monitoring is indicated to provide continuous beat-to-beat measurement of arterial pressure and frequent blood-gas analysis. The transducer should be zeroed at skull level and cerebral perfusion pressure should be calculated.[20]

Venous air embolism, however, remains the most feared complication of operative procedures performed in the sitting position. Supplementary monitoring is directed towards prompt detection and early treatment of VAE. Monitoring techniques include praecordial Doppler, right heart catheters, transoesophageal echocardiography, fractional excretion of nitrogen ($F_{E_{N_2}}$), capnography, oesophageal stethoscope and transcutaneous oxygen measurement. Transoesophageal echocardiography is the most sensitive monitor to detect air in the right atrium and paradoxical embolization of air to the left atrium through a patent foramen ovale.[21] TOE is capable of detecting a single air bubble as an echo dense structure.[71] However, TOE is not specific for VAE. Fat emboli and blood microemboli will also be detected.

Doppler ultrasonography is the most sensitive of the generally available monitors capable of detecting intracardiac air.[12 35] Commercially available Doppler systems generate and detect an ultrasonic signal (approximately 2.0–2.5 mm Hz), which is reflected from moving erythrocytes and cardiac structures. The reflected signal is converted to audible sound. Air is an excellent acoustic reflector. Its passage through the heart is heralded by a change from previously established regular swishing signal to an erratic roaring noise. Praecordial Doppler has been compared with TOE in patients undergoing anaesthesia and neurosurgery in the sitting position. TOE and Doppler were found to be equally sensitive with respect to air detection, and TOE provided the added benefit of localization of intracardiac air within a specific cardiac chamber.[20] Thus TOE also allows recognition of inter-atrial passage of air before it reaches the systemic circulation. Early Doppler detection and immediate aspiration from a right atrial catheter has been shown to decrease mortality from VAE.[59]

The progressive decrease in lung perfusion caused by air trapped within the pulmonary circulation leads to increased physiological deadspace which is reflected by a decrease in end-tidal carbon dioxide concentration and an increase in end-tidal nitrogen. Continuous monitoring of end-tidal carbon dioxide concentration and $F_{E_{N_2}}$ may provide a semi-quantitative estimate of the volume of VAE. These forms of monitoring offer intermediate sensitivity with respect to VAE detection. Similarly, transcutaneous P_{O_2} is also of intermediate clinical application.[12]

The routine use of a central venous catheter (CVC) is recommended by several workers.[12 40 70 74] A CVC tip positioned close to the superior vena caval junction with the right atrium provides a measure of intravascular volume status and can be used to confirm the presence of intravascular gas in addition to a means for evacuation of air.[19 74] CVC may be percutaneously inserted via a cubital vein. Trendelenburg tilt, neck rotation and the possibility of haematoma formation limit the traditional internal jugular vein catheter placement.

Intravascular electrocardiography may be used to accurately localize the catheter tip[54]; the P wave should be large and negative with no positive component. *In vitro*

experimental data suggest that the caval–atrial junction is the most efficacious site for air removal[19] and multi-orifice catheters have been designed to enhance bubble recovery.[15] The CVC may also be used to verify Doppler probe placement using rapid injection of saline.[12]

The PAC has been evaluated as a diagnostic and therapeutic tool.[51] A prospective clinical study compared the PAC with praecordial Doppler. An increase in PAP provided a semi-quantitative estimate of the volume of intravascular air. PAP changes preceded systemic cardiovascular changes. Entry of air into the pulmonary circulation causes an increase in PAP. A subsequent decrease in PAP and clearing of the Doppler signal suggested onward movement of air distally into the pulmonary circulation. Only small quantities of air could be aspirated from the PA or central venous ports of the PA catheter.

The older classic method of monitoring for intracardiac air with an oesophageal stethoscope for a change in heart sounds or a 'mill-wheel' murmur is dependant on large amounts of intracardiac air and provides little advance warning of impending cardiovascular collapse.[50] Because of its non-invasive nature and mechanical simplicity, the oesophageal stethoscope should be retained in monitoring procedures designed to detect air embolism.

Cardiovascular changes occur late and include hypotension, elevation of CVP and ECG changes. VAE may increase airway pressures during mechanical ventilation as a result of bronchoconstriction and reduced pulmonary compliance.[73] As no one monitor is completely reliable, many workers recommend more than one form of monitoring for VAE. Black and Cucchiara recommend a minimum of three monitoring techniques.[12] Doppler ultrasound is advocated as the basic monitoring device to minimize complications caused by VAE. It is non-invasive, sensitive and capable of detecting 0.015 ml kg^{-1} of air.[35] The probe position over the right heart (usually in the third to sixth intercostal spaces to the right of the sternum) can be verified by rapid injection of saline into a central venous cannula and its signal is audible to both surgeon and anaesthetist. The performance of the Doppler is generally related to correct placement of the transducer and unusual chest configurations; variable cardiac position, breast tissue and obesity may preclude accurate positioning of the transducer. In addition, a correctly positioned probe may shift during positioning changes. For these reasons, simultaneous application of other VAE detection methods is advocated.

The risk of VAE is not confined to neurosurgical procedures performed in the sitting position,[5 14] nor is it eliminated by placing the patient horizontal.[4 14] Avoidance of techniques which enhance air entrainment or increase bubble size is imperative, as is identification of the population at risk of its devastating sequel paradoxical air embolism.

Children appear to represent a subpopulation at increased risk of VAE as the reported incidence of VAE is significantly higher compared with adults (73% compared with 37%)[57] and is associated with more profound hypotension.[19] Intermittent positive pressure ventilation has been advocated to prevent the reflex gasp which occurs with an air embolus which may cause a bolus of air to be sucked into an open vein.[35 74] Initial exposure of the posterior fossa, when air may enter the diploic and emissary veins or the dural sinuses,[84] is the time of greatest concern for VAE.

Sources of VAE are often not identified but careful surgical technique is paramount. Bone was identified as a source of VAE in 16% of cases in one study[57] and hence the recommendation that all bone edges be waxed. Pin-type headholders have also been implicated and these pins should be wrapped with gauze impregnated with petrolatum or bismuth tribromophenate.[16 57]

Summary

The potential for serious complications after venous air embolism and successful malpractice liability claims are the principle reasons for the dramatic decline in the use of the sitting position in neurosurgical practice. Although there have been several studies substantiating the relative safety compared with the prone or park bench positions, its use will continue to decline as neurosurgeons abandon its application and trainees in neurosurgery are not exposed to its relative merits.

How can individual surgeons continue to use this position? Will individual, difficult surgical access cases be denied the obvious technical advantages of the sitting position? Limited use of the sitting position should remain in the neurosurgeon's armamentarium. However, several caveats must be emphasized. Assessment of the relative risk–benefit, based on the individual patient's physical status and surgical implications for the particular intracranial pathology, is of paramount importance. The patient should be informed of the specific risks of venous air embolism, quadriparesis and peripheral nerve palsies. Appropriate charting of patient information provided and special consent issues are essential.

An anaesthetic input into the decision to use the sitting position is a *sine qua non.* The presence of a patent foramen ovale is an absolute contraindication. Preoperative contrast echocardiography should be used as a screening technique to detect the population at risk of paradoxical air embolism caused by the presence of a patent foramen ovale. The technique involves i.v. injection of saline agitated with air and a Valsalva manoeuvre is applied and released.

Use of this position necessitates supplementary monitoring to promptly detect and treat venous air embolism. Doppler ultrasonography is the most sensitive of the generally available monitors to detect intracardiac air. The use of a central venous catheter is recommended, with the tip positioned close to the superior vena cava junction with the right atrium, to aspirate intravascular gas. Measures to minimize hypotension associated with the sitting position include a slow, staged positioning over 5–10 min and use

of the 'G suit' inflated with compressed air applied to the lower extremities and pelvis.

Use of the sitting or upright position for patients undergoing posterior fossa and cervical spine surgery presents unique challenges for the anaesthetist. With appropriate patient selection and preparation, and using prudent intraoperative monitoring and anaesthetic techniques, selected patients should still benefit from the optimum access to mid-line lesions, improved cerebral venous decompression, lower intracranial pressure and enhanced gravity drainage of blood and CSF associated with the sitting position.

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