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Effects of Positioning on Cerebral Oxygenation

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Abstract Postural change during anesthesia has a complex effect on the systemic and cerebral circulations which can potentially decrease cerebral blood flow and oxygenation. Cerebral oximetry is emerging as a monitor of cerebral perfusion with widespread application in many types of surgery. The technology is based on the differential absorption of oxygenated and deoxygenated hemoglobin to near-infrared light. However, the dynamic coupling that exists between cerebral arterial, venous and cerebrospinal fluid volumes may influence oximetric readings during postural change. Interpretation of cerebral oxygen saturation measurement must account for these changes in cerebral physiology if monitoring is to predict neurological outcome.

Keywords Sitting position · Beach chair surgery · Cerebral hemodynamics · Cerebral ischemia · Cerebral oxygen saturation · Neurological outcome

Introduction

> Optimal hypotension can be attained by a postural drainage of blood into the lower limbs. This postural hypotension is safe if carefully controlled to a pressure not lower than 55–65 mmHg. at heart level… (Hale Enderby, Royal College of Surgeons, 1952).

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Surgery is usually performed with the patient supine, though specific operations require the patient to be placed prone, lateral, head-down or into a sitting position, each of which carry specific risk [1]. The neurosurgical sitting position, in particular, presents a major physiological challenge for the anesthetist, and in recent years it has diminished in use due to the risk of complication [2, 3]. However, this position is widely used by orthopedic surgeons for routine shoulder arthroscopy and cuff repair without any form of cerebral monitoring [4]. Of concern is that the sitting position can dramatically affect the circulation with arterial hypotension increasing the risk of cerebral hypoperfusion and ischemia [5, 6]. Several case reports from Pohl and Cullen [6] raised serious concerns about the safety of the sitting position and focused awareness on its physiological effects and management [7]. Even though the incidence of severe neurological injury in sitting patients is reportedly low [8], an awareness of its physiological effects is necessary to ensure patient safety during general anesthesia [9]. With each sitting patient, the primary intraoperative concern is whether arterial pressure remains adequate for cerebral perfusion.

In 1954 Hale Enderby described the technique of *postural ischemia*, where surgical bleeding was reduced by placing the patient into a steep reverse Trendelenburg tilt [10]. This head-up tilt, in association with volatile anesthesia and ganglionic blocking drugs, caused significant hypotension with systolic arterial pressure often decreasing to below 70 (and in some cases below 50) mmHg [11]. Importantly, Enderby carefully monitored these low levels of arterial pressure with the use of an oscillometer. Today, hemodynamic monitoring remains a fundamental aspect of anesthetic practice and the technique of induced hypotension remains an established practice though its use in the sitting position has been questioned [12].

Near-infrared spectroscopy (NIRS), a non-invasive optical technology, has emerged as a monitor of cerebral perfusion, and been recently used for surgery performed in the sitting position. Regional cerebral oxygen saturation (ScO_2) of the frontal cortex is determined by comparing the specific absorbance patterns of oxygenated and non-oxygenated hemoglobin to near-infrared light [13]. As cerebral blood flow (CBF) decreases, tissue oxygen extraction will increase to maintain cerebral metabolism with an eventual decrease in hemoglobin saturation. In the presence of a stable metabolic rate, ScO_2 is therefore an indirect measure of CBF and provides an "index" of organ ischemia [14]. Monitoring ScO_2 allows clinically silent episodes of cerebral ischemia to be detected, and the technology has been used extensively, ranging from neonatal intensive care to adult surgery. However, many factors may influence oxygen transport and cerebral oxygen saturation including hematocrit, hemoglobin-O_2 binding affinity (P_{50}), inspiratory oxygenation and ventilation. In addition, CBF can be influenced by posture, head positioning and anatomical variation [15, 16].

Neurological monitoring with cerebral oximetry can potentially redefine the management of intraoperative hypoxia and hypotension [17•]. Intraoperative ScO_2 measurement provides a therapeutic endpoint for clinical intervention, such as the use of vasopressor therapy to increase cerebral perfusion pressure (CPP). The concept of CPP is central to cerebral protection, and states that CBF is determined primarily by the pressure gradient across the cerebral vascular bed. The driving force for flow equates to the difference between arterial mean pressure and intracranial pressure (ICP) or central venous pressure (CVP). The head elevation debate continues as to whether CBF is more reliant on the perfusion pressure at head level, conceptually viewed as a declining or *waterfall gradient;* or alternatively, whether flow is best viewed as a continuous column of blood, *siphoned* into the low-pressured superior vena cava. In clinical practice these views may influence the threshold for intervention when managing hypotension. However without direct measurement of cerebral perfusion, the perception that blood pressure control alone is sufficient to maintain cerebral perfusion in all patients is limited. The clinical assumption that CBF simultaneously increases with arterial pressure elevation cannot be made.

In cardiac surgery, ScO_2 monitoring has proven to be a valuable tool in the management of cerebral perfusion during cardiopulmonary bypass, and is predictive of clinical outcome [18]. However in non-cardiac surgery, the role of ScO_2 monitoring remains to be validated, especially in surgical patients positioned upright. Upright positioning is known to cause reflex changes in ICP, cerebrovascular resistance (CVR), intracranial compliance (ICC), as well as global and regional changes in CBF. The interpretation of ScO_2 measurement must, therefore, account for any physiological change that may alter photon penetration, absorbance or scatter.

Physiological Effects of Posture

In awake individuals, a change from supine to standing rapidly activates neuroendocrine reflexes that regulate blood pressure and maintain adequate cerebral perfusion [19, 20]. The arterial baroreflex responds rapidly—within seconds—to postural change inducing cardiac acceleration and peripheral vasoconstriction. Cerebral autoregulation responds simultaneously with cerebral vasodilation regulating CBF [21]. The coordination between these two responses is not fully understood. With aging, the homeostatic response to postural change declines, and in cardiovascular disease such as hypertension and possibly diabetes, regulation is impaired [22]. Cerebral blood vessels are also richly innervated with adrenergic nerve fibres [23] and recent evidence indicates that extracranial sympathetic nerve activity, intrinsically related to baroreceptor function [24], may influence CBF [25]. Hypotensive-bradycardic episodes (HBE), known to occur in upright shoulder surgery [26], are mediated by autonomic activation via a common mechanism responsible for vasovagal syncope, carotid sinus hypersensitivity and orthostatic syncope [27]. Importantly patients with orthostatic hypotension respond to postural change with larger swings in arterial pressure.

Posture and the Systemic Circulation

Marked physiological change occurs when an anesthetised patient is tilted from a supine to upright position (Fig. 1). Gravity causes blood to pool into the lower extremities and a shift in intrathoracic blood volume to the extrathoracic space occurs. Venous return is decreased resulting in a significant decrease in cardiac output, systemic vascular resistance and arterial pressure [28]. Echocardiography has demonstrated a significant reduction in left ventricular end-diastolic dimension and middle cerebral artery velocity [29]. Central venous and left-atrial pressure both decrease though anesthetic technique may influence this [30, 31]. Volume loading with crystalloid or colloid fluids do not fully prevent the decrease in cardiac output [31]. Correct positioning, with elevation of the knees to heart level and use of compression stockings [32], can facilitate venous return. Sequential compression of lower limbs significantly reduces hypotension and can support the mean arterial pressure (MAP) and the stroke volume index [33]. Inflation of medical anti-shock trousers (MAST) is a strategy used to increase CVP and jugular venous bulb pressure to reduce the risk of venous air embolism [34]. Anesthetic induction

dose, volatile agents and intermittent positive pressure ventilation (IPPV) all aggravate this effect on preload [32]. In sitting patients, systolic blood pressure is reported to decrease by 47 % [3, 35]. In his series of upright positioning, Enderby reported that for each 1.25 cm above the level of the head, local arterial pressure is reduced by ~1 mmHg, resulting in a decrease in CPP approximating 25 mmHg [10].

Posture and the Cerebral Circulation

The brain is critically dependent on a continuous substrate and oxygen supply, requiring 15 % of cardiac output, and accounting for 20 % of total body oxygen consumption and 25 % of total body glucose utilization [36]. In their classic study, Kety and Schmidt measured CBF as 54 ml min^{-1} 100 g^{-1}, equating to a total CBF of 702 ml min^{-1}. Though subsequent studies have confirmed this value, significant individual variation occurs [37, 38]. With age CBF decreases by 3 ml min^{-1} per year [39]. The cerebral circulation is controlled by several mechanisms, with autoregulation maintaining a constant blood flow when arterial pressure and CPP are changing [40, 41]. Regional CBF is primarily coupled to metabolic rate, with vascular tone influenced by changes in neural activity, associated substrate production and pH. An increase in arterial carbon dioxide partial pressure ($PaCO_2$) can induce vasodilatation, increasing CBF by 6 % per mmHg change in $PaCO_2$, whilst hypocapnia decreases CBF 3 % per mmHg change in $PaCO_2$ [42]. Control of CBF by sympathetic innervation has been demonstrated but its importance remains debated [43]. Activation is believed to protect the cerebral microcirculation from excessive increases in perfusion pressure and flow and influence cerebral capacitance vessels to regulate ICP and cerebral blood volume [44]. Postural change, including the 30° head-up tilt is commonly used in critical care, to reduce ICP and facilitate cerebral perfusion [45], often in combination with hyperventilation [46].

The brain is supplied by the internal carotid (ICA) and vertebral arteries (VA), which anastomose to form the ring-like arterial Circle of Willis. The anterior ICA circulation contributes 76 % of total flow, and the posterior vertebral system 24 % [37]. Anatomical variation of the cerebral circulation is common, with an incomplete circle of Willis reported in about 50 % of the population, usually involving at least one absent or hypoplastic communicating artery [47]. Congenital variations in the VA have also been described [48]. These variations are associated with asymmetrical flow [49] and in patients with severe ICA stenosis, a risk of stroke and transient ischemic attack is also present [50]. Delayed awakening and hemiparesis following shoulder surgery in the sitting position has been attributed to such variant anatomy [51].

Cerebral Blood Volume

The cerebral blood volume comprises three components: arterial, capillary and venous blood volumes. The arterial: venous blood volume ratio is ~0.3 [52] and capillary blood volume is considered negligible [53]. In healthy subjects, the effect of posture on intracranial physiology has been studied using magnetic resonance imaging (MRI) [54•]. With head elevation there is hydrostatic displacement of cerebrospinal fluid (CSF) from the cranial cavity to

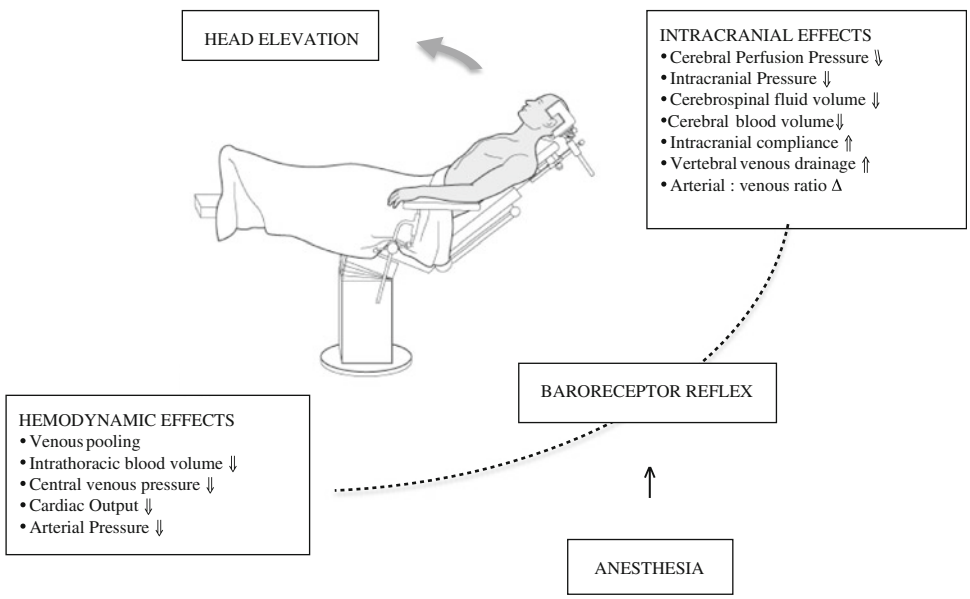

Fig. 1 Hemodynamic and intracranial changes associated with the sitting beachchair position during anesthesia. Source: AO Surgery Reference, www.aosurgery.org. Copyright and permission granted by AO Foundation, Switzerland

the spinal subarachnoid space. A dynamic reduction in cerebral venous blood volume occurs, with a resultant increase in ICC, a decrease in venous outflow pulsatility and an increase in cerebral transit time. These postural changes lower ICP and facilitate cerebral perfusion [55]. Further, hypercapnia can induce vasodilatation and increase arterial blood volume without necessarily a change in venous blood volume [56]. Hypercapnia also affects the circulation regionally with the temporal-occipital cortices tending to vasoconstrict and frontal cortices tending to vasodilate [57].

In supine subjects, venous outflow is primarily via the internal jugular veins. In the sitting position, flow shifts to the vertebral, epidural and deep cervical venous pathways, possibly as a result of the decrease in CSF volume. Postural change results in complete or partial collapse of the internal jugular vein. Venous outflow, in conjunction with CBF, decreases by 12 %. Spontaneous ventilation with inspiratory subatmospheric pressure facilitates cerebral venous return, but this mechanism is attenuated by positive pressure ventilation.

Cerebral Perfusion Pressure

Autoregulation maintains CBF at a constant level in response to changes in CPP. At the lower limit of autoregulation (LLA), maximal cerebral vasodilatation occurs below which CBF becomes pressure passive, decreasing as pressure falls. The threshold for LLA is debated, primarily because significant individual variation exists. In hypertensive patients, LLA is shifted to the right, with higher a CPP required to ensure adequate cerebral perfusion. With ageing, the vascular responsiveness of cerebral arterioles to $PaCO_2$ diminishes [58] and the autoregulatory range also becomes narrowed. Studies in conscious patients have demonstrated ischemic symptoms to occur when arterial pressure decreases to 48 mmHg, but significant individual variation shows the onset of symptoms ranging from 29 to 89 mmHg [59]. Drummond has emphasized that the LLA is significantly higher than a conventional value of 50 mmHg in most patients and should be modified upward to reflect a range of values from 70 to 93 mmHg with a mean value of 80 mmHg [60]. Estimation of LLA should be based on individual resting MAP, with a 25 % decrease proposed as threshold, and a 40–50 % decrease indicative of ischemia [59].

The effective cerebral perfusion pressure (eCPP) is a concept that relates cerebral perfusion to the *downstream* resistance to flow within the cerebral vasculature [61]. Cerebral resistance vessels collapse when transmural pressure is affected by changes in both ICP and arterial pressure occurring with upright positioning. With small vessel closure, the critical closing pressure (CCP) is defined as the arterial pressure at which CBF becomes zero, and can be can be determined using transcranial Doppler. Simultaneous measurement of middle cerebral artery blood flow velocity (MCAv) and arterial waveforms provides an estimate of eCCP (eCPP = MAP−CCP). Estimates of CCP have been shown to be influenced by arterial $PaCO_2$, ICP, cerebral autoregulation, intra-thoracic pressure, and MAP [62]. In patients undergoing shoulder surgery, McCulloch [35] found the sitting positioning to decrease MAP by 47 % and MCAv by 22 %. Cerebral vasodilatation was associated with a decrease in both the resistance area product (RAP), a measure of cerebrovascular resistance, and the apparent zero flow pressure (AZFP), an estimate of the critical closing pressure. They hypothesized that the decrease in MCAv was due to the dual effect of autoregulatory cerebro-vasodilatation, and a decrease in transmural wall pressure, caused by a lower ICP during beachchair positioning. Such small vessel collapse is considered to partly undermine the "siphon" hypothesis of CBF during upright positioning.

Principles of Cerebral Oximetry

Most biological tissues including bone are transparent to infrared light, and as light penetrates tissue, absorption will occur primarily by hemoglobin and cytochrome molecules. Within the NIR wavelength range 700–1,000 nm, the absorption spectra of oxyhemoglobin and deoxyhemoglobin differ, causing an alteration in the reflected NIRS signal. The ratio of oxygenated to nonoxygenated hemoglobin forms the basis of estimating brain oxygen saturation [63], with measurement derived using an algorithm based on the Beer-Lambert law [64]. Optodes placed on the forehead above the brow, transmit infra-red light, through the skull, to penetrate several centimetres into the frontal cortex [65]. The beam follows a curvilinear path, is subject to absorbance and scatter, and reflected photons are then received by surface detectors. Most devices are continuous wave (CW) monitors with a transmitter-detector distance (usually 4 cm) allowing deeper cortical signals to be separated from superficial extracranial signals [66]. The CW devices measure hemoglobin saturation from the red: infrared ratio, without measuring absolute values of oxy- and deoxy hemoglobin. Increased spatial resolution is achieved by optimising emitter-to-detector separation, using multiple wavelengths and detectors, or by determining absolute chromophore values. Frequency domain (FW) and time-domain approaches estimate ScO_2 based on absolute values of oxygenated, deoxygenated and total hemoglobin, and are thought to account for variation in cerebral blood volume [67, 68]. The FW-NIRS devices account for individual scatter using a mathematical model based on photon

diffusion theory to calculate tissue oxygen index (TOI) and total hemoglobin index (THI).

Multiple investigations have shown ScO_2 to increase with hyperemia, and decrease with ischemia. Indocyanine green, a dye with similar absorbance spectra to hemoglobin, injected into the ICA during angiography increases ScO_2 whilst ScO_2 was unchanged with external carotid injection indicating detectors received predominately cortical signals [69]. Acute vessel occlusion results in temporal ScO_2 desaturation in human and animal studies [70, 71]. The ScO_2 desaturation correlates with jugular venous O_2 saturation ($SjvO_2$) under conditions of hypoxia and hypercapnia [32, 72, 73]. Functional MRI has shown NIRS measurement to be consistent with changes in deoxyhemoglobin concentration and regional cerebral blood volume [74]. The CBF autoregulation can be assessed using NIRS [75] and an NIRS-based autoregulation index, derived from ICP and blood pressure waveforms, has been developed [76]. Despite these validation studies, NIRS remains fundamentally limited in its application, since a small sample volume of the frontal cortex (1.5 cm^3 at a depth of 1.5 cm) is used to represent global cerebral perfusion. Most cerebral regions including the vertebrobasilar arterial system remain unmonitored.

Clinical Experience

Cerebral oximetry has widespread application as a functional monitor of cerebral oxygen saturation during surgery and in the intensive care unit [14]. It has been used in carotid [77], abdominal [78], thoracic [79] and orthopedic surgery (Table 1). In paediatric patients ScO_2 has been used to monitor sleep apnea [80] and cerebral autoregulation in preterm infants [81]. The ScO_2 monitoring correlates with other monitors of cerebral ischemia, such as transcranial Doppler, somatosensory evoked potentials, and stump pressure during carotid cross-clamp [77, 82–84]. During cardiopulmonary bypass, ScO_2 can provide a more rational means for individualizing MAP, since cerebral autoregulation is impaired in 20 % of patients predisposing them to perioperative stroke [85]. The ScO_2 can identify the threshold for autoregulation, observed as 66 mmHg in a study of 225 patients (range 43–90 mmHg) [86]. Monitoring cerebral ScO_2 during coronary artery bypass can avoid profound cerebral desaturation, and have significantly fewer incidences of major organ dysfunction [87]. In neonates undergoing arterial switch operations, a decreased preoperative ScO_2 is associated with decreased Developmental Quotient scores at 30–36 months [88]. In children undergoing congenital heart surgery, perioperative death was associated with baseline cerebral saturation of <50 % [89]. Similarly in adult patients undergoing on-pump cardiac surgery, baseline ScO_2 is an independent risk factor for 30-day and 1-year mortality [90]. Preoperatively ScO_2 measurement has been used to identify patients with increased risk of cardiac morbidity and autonomic failure [90, 91].

Cerebral ischemia is likely to occur with a regional CBF < 220 ml min^{-1} (<15 ml 100 g^{-1} min^{-1} and $CMRO_2$ < 1.3 ml 100 g^{-1} min^{-1}) [38, 92]. In conscious patients, the normal ScO_2 range is 60–75 %, with a coefficient of variation being 10% and values lower in the elderly [93, 94]. In awake humans, clinical symptoms of pre-syncope occur when cerebral desaturation exceeds 20 % [82, 95] and during carotid endarterectomy, ischemic symptoms reportedly occur with desaturation 13 % from baseline [96]. In cardiac surgery, a prolonged desaturation episode >20 % decline from baseline, or value of <50 %, is associated with a greater risk of postoperative cognitive decline, increased hospital stay [87, 97, 98], and adverse neurological outcome [99]. In animal models of deep hypothermic circulatory arrest, similar thresholds are associated with neurologic outcome. Brain energy depletion is characterized by increased cerebral lactate, and associated ScO_2 values of 40–45 %, with levels below 35 % associated with cellular injury. [84, 100–102]. The threshold for cerebral ischemia is often taken as >20 % desaturation from baseline [77].

Validity of ScO_2 with Postural Change

The effect of posture on cerebral blood volume and venous composition calls into question the accuracy of ScO_2, since readings are primarily a measure of venous hemoglobin saturation (Fig. 2). Positive emission tomography has demonstrated the venous: arterial blood volume ratio, approximating 70 %, to correlate with ScO_2 measurement [103]. However this absolute ratio may vary between individuals [43, 104], with hypoxia [105], hemodilution [106] and with changes in $PaCO_2$ [107•]. Importantly head elevation causes a dynamic reduction in cerebral blood volume, with compensatory changes in vascular tone and arterial inflow. This relationship was demonstrated in patients placed in a head up tilt, where CBV and ScO_2 both simultaneously decreased (0.07 ml 100 g^{-1} and 3.5 % respectively). Hyperventilation had a similar effect (0.06 ml 100 g^{-1} and 2.6 % respectively), presumably from a reduction in arterial inflow. This effect on ScO_2 and CBV had no correlation to changes in MAP or cardiac output resulting from intravenous (not volatile inhalational) anesthesia. Similarly, the head down position with pneumoperitoneum during laparoscopy is associated with cerebral desaturation, likely due to changes in ICP, CBF and volume. This effect was more pronounced with propofol compared to sevoflurane [108].

The CW-NIRS devices incorporate algorithms that may not account for changes in venous content or optical pathlength, with resultant overestimation of desaturation

Table 1 The incidence of cerebral desaturation episodes (DSE) during orthopedic beachchair surgery

Study	Patients	Anaesthesia	Endpoint	Protocol	Comments
Koh et al. [135]	60	ISB and SEDATION ISB and IPPV, SEVO NIBP	DSE incidence	BL preinduction air DSE > 20 % BL	DSE incidence: 57 % GA, 0 % sedation
Salazar et al. [128]	50	ISB, SV, SEVO NIBP	Patient factors Neurocognitive	BL preinduction DSE > 20 % BL	DSE incidence 18 % Obesity risk No POCD
Soeding et al. [29]	34	ISB, IPPV, SEVO IAP	Neurocognitive Vasopressor study	BL preinduction air DSE > 20 % BL	DSE incidence 0 % PE 18 % decrease No POCD
Moerman et al. [130]	20	IPPV, SEVO NIBP	DSE incidence	BL post induction DSE > 20 % BL	DSE incidence 80 % One third < 55 %
Jeong et al. [131]	56	PRO/REMI SEVO/N_2O IAP	DSE incidence Correlation with $SjvO_2$	BL post-induction DSE > 20 % BL $DSjvO_2$ < 50 %	DSE incidence 30 % $DSjvO_2$ Incidence 41 %
YaDeau et al. [125]	99	ISB SEDATION NIBP	DSE incidence Relation to MAP	BL pre surgery DSE > 20 % BL	DSE incidence 0.77 % Hypotension 76 %
Lee et al. [132]	28	IPPV, SEVO IAP	Neurocognitive	BL preinduction DSE > 20 % BL MAP 60–65 mmHg	DSE incidence 7 % No POCD
Murphy et al. [133]	124	IPPV, SEVO, ISB 8 % NIBP	DSE incidence Compare LDC	BL Preinduction, O_2 DSE > 20 % BL	DSE incidence 80 % LDC incidence 0 %
Tange et al. [134]	30	IPPV SEVO NIBP	DSE incidence	BL preinduction air	DSE incidence 0 %

DSE incidence: each study protocol defined a baseline from which significant change was measured. Anaesthestic technique: *PRO* propofol, *REMI* remifentanil, *SEVO* sevoflurane, *PE* phenylephrine, *ISB* interscalene anesthesia, *SV* spontaneous ventilation, *IPPV* intermittent positive pressure ventilation. Arterial pressure measurement: *MAP* mean arterial pressure, *NIBP* non-invasive blood pressure, *IAP* intra-arterial blood pressure, *LDC* lateral decubitus position, *$SjvO_2$* jugular venous oxygen saturation, *$DSjvO_2$* desaturation of jugular venous oxygen saturation, *POCD* post operative cognitive dysfunction

values [109]. However CW-ScO_2 monitoring has been demonstrated to remain constant with minor head elevation (20°) [110]. The FD-NIRS uses a quantitative approach where resolution of photon scattering enables "absolute" values of TOI and THI to be derived. In head injured patients, TOI and THI both respond to changes in MAP and ICP, and correlate with changes in brain tissue oxygenation [111]. This could indicate that elevation of MAP causes dilatation of arterial vessels, increasing both oxygenated Hb and CBV. However these values are also reliant on relative saturation ratios and whether these remain unaffected by significant changes in cerebral blood volume with posture is unknown.

Vasopressor therapy, often used to maintain CPP, also influences ScO_2 measurement. Phenylephrine decreases ScO_2 by 7 % in conscious subjects [112] despite an increase in MAP and MCAv. This discordance between CPP and ScO_2 was abolished by increasing cerebral metabolic rate with exercise. Cerebral desaturation occurs

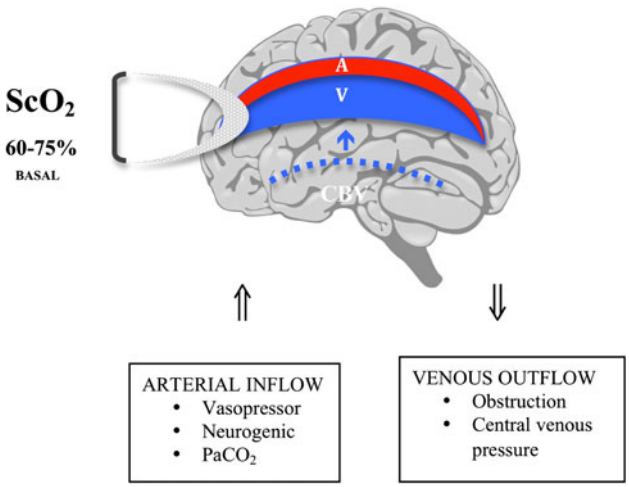

Fig. 2 The effect of head elevation on cerebral oxygen saturation (ScO_2) resulting from the dynamic change in intracerebral blood volume (CBV) and arterial: venous blood volume. *A* cerebral arterial blood volume, *V* cerebral venous blood volume

during anesthesia with phenylephrine [29] and noradrenaline [113•] but not ephedrine [114]. The degree of desaturation with phenylephrine is also modulated by $PaCO_2$ [115]. Since sympathomimetics are unlikely to cross the blood brain barrier, activation of cerebral sympathetic nerves has been postulated as a mechanism of action. In a separate study, where noradrenaline decreased ScO_2 by 6 %, and whole body heating increased ScO_2, a mechanism of noradrenaline-induced skin vasoconstriction was proposed [116]. The authors suggested that crosstalk between cutaneous and cerebral circulations is an inherent limitation of optode design, amplified by vasopressor therapy. Despite multiple studies showing that extracranial contamination is negligible, discussion remains concerning this phenomenon. This was evidenced in awake patients where desaturation occurred (6.8–18.8 %) from sequential forehead constriction using a pneumatic cuff [117]. Posture and vasopressor therapy together appear to be confounding influences on ScO_2 measurement.

The Orthopedic Sitting Position

First reported in 1988 by Skyhar [118], the beachchair position has been adopted as a standard approach for shoulder surgery by many orthopaedic surgeons [119]. The position is deemed to provide superior anatomical orientation, improved surgical access, a lower incidence of brachial plexus neuropraxia from traction, and improved outcome compared to the lateral decubitus position [120]. In its original description, the beachchair position placed the patient in 45° of head elevation, however many surgeons use a more upright position, termed the "steep beachchair" or "barberchair" position [121]. Complications with beachchair positioning were soon reported, including neurological injury [6], HBE [122], and rarer venous air embolism [121]. Concern that the beachchair was an independent risk factor for neurological injury initiated a survey of practice by the American Shoulder and Elbow Society [119]. The results indicated orthopaedic surgeons used the beachchair twice as frequently as the lateral decubitus position, and the reported incidence of neurological injury was 0.0038–0.0046 %. A second survey[4] demonstrated a beachchair incidence of 65 % and a diversity of practice including : induced hypotension 28 %, non-invasive blood pressure correction for height 48 %, and the use of an arterial line in 14 % of patients.

It is apparent that beachchair positioning is practiced widely and safely in many centres [8, 123]. A recent review of 15,014 cases of shoulder arthroscopy over an 11-year period reported the incidence of perioperative neurologic complications to be rare [8]. In this review, patients underwent beachchair surgery in conjunction with regional anesthesia, propofol sedation, and spontaneous respiration via oxygen mask. The MAP was measured non-invasively and maintained at not <80 % of the preoperative value or >60 mmHg. The MAP was not corrected for height. These authors demonstrate the benefit of sedation in minimizing the hypotensive effect of the upright posture and a low incidence of morbidity. The technique of interscalene regional anesthesia with sedation is associated with less hypotension during upright positioning compared to volatile inhalational anaesthesia [124]. However, it may not be appropriate to extrapolate these results to patients receiving general inhalational anaesthesia.

General inhalational anesthesia, with spontaneous or IPPV, is associated with more hypotension in the sitting position compared to sedation, with the response dependant on drug choice and dosage. The increased intrathoracic pressure associated with IPPV is an important contributor to impaired CBF in the seated position [125]. In neurosurgical sitting patients [32] the effect of different anesthetic techniques (nitrous oxide with either enflurane, halothane, fentanyl-droperidol or morphine) on haemodynamic variables were compared [32]. Volatile anesthesia resulted in significant hypotension, and was associated with a reduction in cardiac index and systemic vascular resistance index but likely depends on the volatile agent used in each case. With general anesthesia, hyperventilation can decrease CBF, and volatile agents act to vasodilate cerebral vessels to increase CBF. Compared to isoflurane, sevoflurane preserves the dynamic cerebral pressure autoregulation in humans [126]. Further measures, such as fluid loading and the use of lower limb compression devices, can reduce the incidence of hypotension [127].

Monitoring ScO_2 in Beachchair

A number of studies, including case reports, observational studies and a randomized trial have used ScO_2 measurement to monitor cerebral oxygenation during beachchair surgery (Table 1) [29, 125, 128–135]. These studies confirm that beachchair positioning is associated with hypotension and ScO_2 desaturation, ranging from 0 % to over 80 %. Importantly, anesthetic technique and definition of ScO_2 baseline account for the variable incidence. The magnitude of desaturation is very dependent on how a relative change from baseline is measured. Desaturation will vary depending on whether the ScO_2 baseline is measured as ScO_2 inspiring room air, pre-induction ScO_2 with oxygen supplementation or post induction ScO_2 with assisted ventilation. Ventilation can significantly affect ScO_2 values with 100 % inspiratory oxygen increasing ScO_2 by 8 % and further with hypercapnia [136]. The ScO_2 levels are elevated post-induction conferring a margin of

safety before upright positioning. In the study reporting a desaturation incidence of 80 %, patients received positive pressure ventilation and the relative change was measured from a post-induction oxygenated ScO_2 baseline [133]. In contrast, with a sevoflurane-interscalene technique, ScO_2 desaturation was present but with ScO_2 values returning to physiological levels on air, indicating adequate oxygenation during surgery [29]. Spontaneous ventilation will likely affect CPP differently than PPV, with augmentation of venous return and the effect of permissive hypercapnia on CBF both playing a role. The ScO_2 levels are also influenced by choice of induction agent, with propofol and thiopentone increasing ScO_2 and etomidate decreasing ScO_2 [137]. Interscalene anaesthesia with sedation has significantly less hypotension and episodes of cerebral desaturation. In addition, there is speculation that interscalene anesthesia may influence ScO_2 via sympathetic neural blockade.

Ultimately the relevance of desaturation depends on whether there is a significant change from basal levels. No studies were able to detect post-operative neurocognitive impairment despite reported episodes of desaturation. Neurological and neurocognitive outcome relating to ScO_2 desaturation is a difficult area to study because the incidence of stroke in non-cardiac surgery is very low [138] and secondly, the reported cases with severe neurological injury related to beachchair surgery is extremely small [6, 119, 139]. However, post operative neurocognitive dysfunction (POCD) is prevalent in surgical patients, and appears to be related to episodes of cerebral desaturation [79, 140, 141] rather than to type of surgery [142]. Many of these studies in Table 1 focused on POCD as a primary endpoint in order to define a critical threshold for ScO_2 desaturation.

Clinical Recommendations for Upright Positioning

Preoperative assessment aims to identify patients who may be susceptible to postural hypotension and increased risk of cerebral ischemia. Significant cardiovascular and cerebrovascular disease including carotid artery stenosis, may make such patients unsuitable for upright positioning. In these cases alternative surgical positioning should be sought or a regional technique with minimal sedation considered. Obesity, identified as a risk factor for cerebral desaturation [128] and patients with autonomic failure are more susceptible to postural hypotension [91]. During awake tilt-testing patients with autonomic dysfunction exhibit a critical reduction in cerebral oxygen delivery and ScO_2 [143]. Similarly, patients with symptoms suggestive of orthostatic hypotension may be at risk of developing HBE, whilst patients with symptoms of vertebrobasilar insufficiency, termed "salon parlour syncope" require careful assessment. Preoperative ScO_2 measurement with tilt-testing may be a valuable screen in such patients [91]. Baseline measurement of MAP, and ScO_2 on room air will define individual endpoints for blood pressure management. Pre-induction hydration, leg stockings and intermittent calf compression can minimise venous pooling [33]. Attention to drug dosage, limitation of airway pressure with positive pressure ventilation, avoidance of hyperventilation, and correct positioning with graduated elevation, are recommended.

Blood Pressure Management

Arterial blood pressure management during upright surgery has been a focus of debate since the time of Enderby. In the case series described by Pohl and Cullen, neurological injury was attributed to a decrease in arterial pressure of 28–42 %. Vasopressor therapy is clinically appropriate to treat hypotension when arterial pressure is considered below the LLA. However, determination of this arterial threshold remains difficult since autoregulation exhibits wide individual variation, and the correlation between hypotension and neurological injury is poorly defined. A 30 % decrease in MAP from baseline has been associated with an increased risk of perioperative stroke [144], and during controlled hypotension with electroencephalographic monitoring, a target systolic blood pressure 90–100 mmHg was reported as "safe" [145]. Monitoring requires vigilance, particularly during the process of positioning. Some patients may be more susceptible to developing HBE with positioning and, in these cases, significant hypotension requires immediate intervention. A cautious approach is to target MAP close to baseline levels [7]. Prophylactic administration of vasopresssor by bolus or infusion, before positioning may attenuate the hemodynamic disturbance of the upright posture [29]. Measurement of arterial pressure via the calf is best avoided, and non-invasive arm measurement requires correction for head elevation. Intra-arterial measurement transduced at the tragus, has the advantage of being continuous and indicative of cerebral arterial pressure.

Significant ScO_2 desaturation indicates reduced cerebral perfusion, with desaturation >20 % from baseline or an absolute value <50 %, being triggers for intervention. Desaturation can be managed systematically [18] by increasing CPP (with vasopressor or atropine), increasing venous return (leg elevation/compression, fluids), inducing cerebral vasodilatation with hypercapnia, facilitating cerebral venous drainage (adjusting head position, lowering airway pressure) or increasing blood oxygen content (FiO_2 and haemoglobin). Asymmetrical desaturation between frontal cortices may indicate atypical cerebrovascular anatomy or vertebrobasilar compression.

Conclusion

Clinical experience has demonstrated cerebral oximetry to be a valid neurological monitor in supine patients. However upright positioning induces a dynamic change in intracranial blood volume and venous content, which artificially affects ScO_2 measurement. The evidence indicates this artifact is small, but, nevertheless, requires awareness when interpreting ScO_2 measurements during postural change, and with concomitant vasopressor use. Once the upright posture is attained, ScO_2 monitoring remains valid to trend changes in cerebral perfusion, provided posture and $PaCO_2$ remain constant. The ScO_2 can similarly provide an endpoint for intervention when individual desaturation thresholds are reached. The question remains whether future developments in NIRS technology may account for these dynamic changes related to the sitting posture. Whether cerebral oximetry can identify and improve neurological outcome in noncardiac patients is currently unclear, particularly when the incidence of severe neurological injury is rare. Future studies of ScO_2 monitoring may define more subtle injury, such as POCD, arising from episodes of silent ischemia during surgery.

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