Cerebral Oxygen Desaturation Events Assessed by Near-Infrared Spectroscopy During Shoulder Arthroscopy in the Beach Chair and Lateral Decubitus Positions

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BACKGROUND: Patients undergoing shoulder surgery in the beach chair position (BCP) may be at risk for adverse neurologic events due to cerebral ischemia. In this investigation, we sought to determine the incidence of cerebral desaturation events (CDEs) during shoulder arthroscopy in the BCP or lateral decubitus position (LDP).

METHODS: Data were collected on 124 patients undergoing elective shoulder arthroscopy in the BCP (61 subjects) or LDP (63 subjects). Anesthetic management was standardized in all patients. Regional cerebral tissue oxygen saturation ($Scto_2$) was quantified using near-infrared spectroscopy. Baseline heart rate, mean arterial blood pressure, arterial oxygen saturation, and $Scto_2$ were measured before patient positioning and then every 3 minutes for the duration of the surgical procedure. $Scto_2$ values below a critical threshold (≥20% decrease from baseline or absolute value ≤55% for >15 seconds) were defined as a CDE and treated using a predetermined protocol. The number of CDEs and types of intervention used to treat low $Scto_2$ values were recorded. The association between intraoperative CDEs and impaired postoperative recovery was also assessed.

RESULTS: Anesthetic management was similar in the BCP and LDP groups, with the exception of more interscalene blocks in the LDP group. Intraoperative hemodynamic variables did not differ between groups. $Scto_2$ values were lower in the BCP group throughout the intraoperative period ($P < 0.0001$). The incidence of CDEs was higher in the BCP group (80.3% vs 0% LDP group), as was the median number of CDEs per subject (4, range 0–38 vs 0, range 0–0 LDP group, all $P < 0.0001$). Among all study patients without interscalene blocks, a higher incidence of nausea (50.0% vs 6.7%, $P = 0.0001$) and vomiting (27.3% vs 3.3%, $P = 0.011$) was observed in subjects with intraoperative CDEs compared with subjects without CDEs.

CONCLUSIONS: Shoulder surgery in the BCP is associated with significant reductions in cerebral oxygenation compared with values obtained in the LDP. (Anesth Analg 2010;X:●●●–●●●)

The beach chair position (BCP) has been used for shoulder arthroscopic procedures since the early 1980s. The advantages of the conventional BCP (45°–90° above the horizontal plane) include lack of brachial plexus strain, a reduced risk of direct neurovascular trauma compared with the lateral decubitus approach, excellent intraarticular visualization, and ease of conversion to an open approach if needed.[1,2] In the United States, approximately two-thirds of arthroscopic and open shoulder procedures are performed with the patient in the sitting position.[3] Although the safety of orthopedic surgery in this position has been well established,[4] rare catastrophic neurologic events have been reported. Pohl and Cullen[5] reported 4 cases of ischemic brain and spinal cord injury occurring after surgery in the BCP. In an additional report, visual loss and ophthalmoplegia were described after shoulder surgery in a sitting position.[6] Eight intraoperative cerebrovascular events were reported in a survey of the American Shoulder and Elbow Surgeons Society; all events occurred during surgery in the BCP.[3]

The etiology of central nervous system injury after shoulder surgery in the BCP has not been established definitively. Several authors have hypothesized that cerebral ischemia may occur when anesthetized patients are placed in a 45° to 90° sitting position.[5,7] In awake volunteers, sympathetic nervous system activation occurs when assuming a sitting position; systemic vascular resistance and heart rate (HR) are increased to maintain mean arterial blood pressure (MAP) and cardiac output.[8–10] In anesthetized patients, however, the response of the autonomic nervous system is attenuated by the vasodilating effects of IV and volatile anesthetics. Significant decreases in cardiac output, MAP, and cerebral perfusion pressure (CPP) have been observed in neurosurgical patients when position was changed from supine to sitting.[11,12] Prolonged reductions in systemic pressures and CPP that exceed critical thresholds (severity and time) may result in permanent neurologic injury.

Near-infrared spectroscopy (NIRS) is a noninvasive technology that provides continuous monitoring of regional cerebral tissue oxygen saturation ($Scto_2$). NIRS technology allows for the immediate recognition and treatment of cerebral desaturation events (CDEs) that would otherwise be undetected with conventional intraoperative monitoring. NIRS has

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Accepted for publication March 29, 2010.

Supported by institutional and/or departmental sources (Department of Anesthesiology, NorthShore University HealthSystem). FORE-SIGHT cerebral oximetry probes were provided to the department at cost for the project by CAS Medical Systems, Inc., Branford, CT.

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DOI: 10.1213/ANE.0b013e3181e33bd9

been used to assess the incidence of CDEs in patients undergoing procedures at high risk for adverse neurologic outcomes (cardiac, vascular, liver transplant, and major abdominal surgery).[13] Although orthopedic patients in the BCP are at risk for cerebral hypoperfusion, no previous clinical trials have assessed changes in $Scto_2$ in this patient population. The aim of this prospective cohort study was to determine the incidence of CDEs in the BCP and compare this cohort to subjects undergoing shoulder surgery in the lateral decubitus position (LDP). In addition, the relationship between CDEs and early clinical recovery was examined.

METHODS

Patients and Anesthesia

This study was approved by the IRB of NorthShore University HealthSystem and written informed consent was obtained from all subjects. Seventy consecutive patients scheduled to undergo elective arthroscopic shoulder surgery under general anesthesia in the BCP were enrolled (BCP group). During this same time period, 70 additional consecutive patients presenting for the same surgical procedure in the right or left LDP were enrolled (LDP group). Exclusion criteria included preexisting cerebrovascular disease or orthostatic hypotension; age <18 years; ASA physical status IV or V; or anticipated procedure under interscalene blockade with monitored anesthesia care as the primary anesthetic. Allocation into the BCP and LDP groups was determined by surgical preference, and use of interscalene blocks in each group was also primarily influenced by surgeon preference. Anesthesia care was administered to the BCP and LDP groups by the same group of providers (15 anesthesiologists).

Cerebral oximetry values may be affected by depth of anesthesia, type of anesthetic administered, arterial carbon dioxide concentrations, inspired oxygen content, and systemic blood pressure management.[14–16] Therefore, anesthetic management was carefully standardized in subjects in both cohorts. Patients received midazolam 2 mg IV before being transported to the operating room. Intraoperative monitoring consisted of electrocardiography, automatic arterial blood pressure assessment using a cuff placed on the nonoperative upper extremity, pulse oximetry, capnography, bispectral index monitoring (BIS® system; Aspect Medical Systems, Newton, MA), and measurement of core temperature via an esophageal probe. Anesthesia was induced with propofol 2.0 to 2.5 mg/kg, fentanyl 100 μg, lidocaine 50 mg, and rocuronium 0.6 to 0.8 mg/kg. Maintenance of anesthesia consisted of sevoflurane 1% to 3% in an oxygen/air mixture (fraction of inspired oxygen [FIo_2] of 50%). Sevoflurane concentrations were adjusted to maintain MAP within 20% of baseline values and BIS values between 40 and 60. In addition, patients received 1 to 2 $\mu g \cdot kg^{-1} \cdot h^{-1}$ fentanyl throughout the surgical procedure. If required, rocuronium (10-mg boluses) was administered to maintain a train-of-four count of 2 to 3. Ventilation was controlled to maintain end-tidal carbon dioxide ($Etco_2$) between 30 and 34 mm Hg. Lower body forced-air warming devices (Bair Hugger®; Augustine Medical, Minneapolis, MN) were used to maintain core temperature above 35.0°C. Ondansetron 4 mg was given to all patients within 30 minutes of tracheal extubation. Neostigmine 50 μg/kg and glycopyrrolate 10 μg/kg were administered at a train-of-four count of at least 2 to reverse neuromuscular blockade at the conclusion of surgical closure.

Clinicians were instructed to maintain MAP within 20% of baseline values throughout the intraoperative period, as per standard practice involving surgery in the sitting position at our institution. Baseline MAP was determined in the sedated patient in the supine position. MAPs below this threshold were treated with phenylephrine (80 μg), ephedrine (5 mg), or a fluid bolus, as required clinically. Patients undergoing surgery in the LDP were treated with the same MAP protocol.

Cerebral Oxygenation Measurements and Perioperative Data Collection

Cerebral oxygen saturation was measured continuously using the FORE-SIGHT system (CAS Medical Systems, Inc., Branford, CT). The FORE-SIGHT device is a continuous wave, spatially resolved cerebral oximeter that uses 4 discrete wavelengths of laser light to calculate the absolute value of $Scto_2$. Four wavelengths of light allow for more accurate determination of oxyhemoglobin and deoxyhemoglobin levels by compensating for wavelength-dependent scattering losses and reducing interference from other background light absorbers.[17,18] Sensors were applied bilaterally to each frontotemporal area after cleansing the skin area with alcohol (the medial margin at the midline of the forehead [avoiding the temporalis muscle] and the lower margin 1–1.5 cm above the eyebrow). The cerebral oximetry and BIS probes were secured in the preoperative holding area and covered with an opaque wrapping to prevent light interference.

On arrival to the operating room, MAP and HR were recorded. Simultaneously, arterial oxygen saturation (Spo_2) and $Scto_2$ values were measured in patients before induction of anesthesia while breathing a 50% air/oxygen mixture. These variables ($Scto_2$, MAP, HR, and Spo_2) were then manually recorded by a research assistant every 3 minutes for the duration of the operative procedure. Intraoperative $Scto_2$ data were also collected continuously on a USB device. Baseline $Scto_2$ was the mean value observed over a 1-minute period after induction of anesthesia during a stable interval (MAP within 20% baseline values, BIS 40–60, $Etco_2$ 30–34 mm Hg, and FIo_2 50%) approximately 10 minutes after induction of anesthesia. Baseline measures for MAP, HR, Spo_2, and BIS used in the analysis were also recorded at this time. Five minutes after these initial data were collected, the patient was positioned for the surgical procedure. In the BCP group, the head was secured in a neutral position to ensure that cerebral venous drainage was not impaired. The back of the operating room table was then raised to 80° to 90° above the horizontal plane. In the LDP group, patients were placed in the LDP with the head supported with towels to maintained alignment with the thoracolumbar spine. The NIRS monitor was positioned so that $Scto_2$ data could be viewed by the research assistant but not by clinicians providing intraoperative care. If any $Scto_2$ values below a previously defined critical threshold (≥20% decrease from baseline or absolute value ≤55% for >15 seconds) were observed by the research assistant,

clinicians were instructed to treat the CDE. For this investigation, a prioritized intraoperative management protocol was used to increase $Scto_2$ values. Interventions to treat CDEs included the following: (1) increasing MAP with phenylephrine (80 μg), ephedrine (5 mg), or a fluid bolus, as clinically indicated; (2) increasing $Etco_2$ by decreasing ventilation; or (3) increasing Fio_2 concentrations. The number and type of interventions used to treat low $Scto_2$ values were recorded by the research assistant. In addition, interventions used by clinicians to treat MAP reductions unrelated to $Scto_2$ were noted. All data were collected until tracheal extubation.

After discontinuation of sevoflurane at the end of the surgical procedure, the time required to achieve the following end points were recorded: open eyes on verbal command; squeeze hand on verbal command; tracheal extubation; and admission to postanesthesia care unit (PACU). The Aldrete score was recorded on arrival to the PACU and then assessed every 15 minutes until discharge. Hydromorphone was used for postoperative analgesia and titrated to achieve pain scores of <2 on a scale of 0 to 10 (0 = no pain, 10 = worst pain imaginable). Any episodes of nausea and vomiting during the PACU admission were noted, and the severity of events graded on a 3-point scale (1 = mild, 1 episode; 2 = moderate, 2–3 episodes; and 3 = severe, >3 episodes). The need for rescue antiemetics was also assessed. The times needed to meet discharge criteria and achieve actual discharge were noted. All recovery data were collected by PACU nurses blinded to intraoperative cerebral oximetry information. However, PACU nurses and research assistants were not blinded to patient positioning.

Patient demographic data that were recorded included age, sex, height, weight, preoperative hemoglobin, ASA physical status, and preexisting medical conditions. Details of the intraoperative anesthetic management included duration of anesthesia, administration of crystalloids, doses of opioids and rocuronium provided intraoperatively, and core temperatures at the conclusion of the anesthetic.

Statistical Analysis

Sample size was determined based on the primary outcome variable, the incidence of CDEs. $Scto_2$ values below a predetermined critical threshold (≥20% decrease from baseline or absolute value ≤55% for >15 seconds) were used to define these events. In a pilot study of patients undergoing surgery in the BCP, CDEs were observed in 50% of the subjects. We hypothesized that we would observe 50% fewer CDEs in patients having surgery in the LDP. Group sample sizes of 66 in each group achieve 80% power to detect a difference of 0.25 between the null hypothesis that both group proportions are 0.50 and the alternative hypothesis that the proportion of the LDP group is 0.25 with a 2-tailed significance level (α) of 0.05 using χ^2 or Fisher exact test with continuity correction.

Discrete data were compared using Fisher exact test (NCSS, Kaysville, UT). The 95% confidence intervals for the differences in percentages were calculated using the Farrington and Manning score. Ordinal data and continuous data that were not normally distributed are presented as median and range. These data were compared between groups using the Mann-Whitney *U* test and within groups using Wilcoxon signed rank test (StatsDirect, Cheshire, UK). The median differences and their 95% confidence intervals were calculated.

Normally distributed continuous data are presented as mean and SD. These data were compared using the unpaired *t* test (NCSS), except for the hemodynamic data. Mean differences and their 95% confidence intervals were calculated. Hemodynamic, Spo_2, BIS, and $Scto_2$ data were compared within and between groups using a 2-factor analysis of variance with repeated measures on 1 factor, with the Holm-Sidak method for pairwise multiple comparisons in post hoc analysis (SigmaPlot 11.0; Systat Software, Inc., San Jose, CA).

Multiple logistic regression analysis (StatsDirect) was performed to determine predictors of nausea. Variables included in the initial analysis were those identified in univariate analyses as having a value of $P < 0.10$. In the final analyses, variables with high *P* values were removed from the model 1 at a time and were excluded from the final model if their removal either did not diminish the fit of the model or actually improved it, as determined by the correct prediction of both positive and reference responses. The sensitivity and specificity of the logistic model were calculated from the model-predicted reference and model-predicted positive responses (using the default threshold probability for positive classification of 0.5) and the actual reference and actual positive responses. No external validation was attempted.

The criterion for rejection of the null hypothesis established a priori was a 2-tailed $P < 0.05$.

RESULTS

One hundred forty patients were enrolled in this clinical trial. Sixteen subjects were excluded from final analysis because of the following factors: changes in patient positioning (45° beach chair) (4 in BCP group); protocol violations (3 in BCP group and 3 in LDP group); incomplete data collection (2 in BCP group and 3 in LDP group); and procedure canceled before entering the operating room (1 in LDP group). As a result, data analysis was performed on 61 patients in the BCP group and 63 patients in the LDP group. The BCP and LDP groups were similar in terms of demographic characteristics. There were no differences between groups in age, weight, height, sex, preoperative hemoglobin values, preexisting medical conditions, or ASA physical status (Table 1). Intraoperative management data are presented in Table 2. The duration of anesthesia was longer in the LDP group. A higher percentage of patients in the LDP group received interscalene blocks (71.4%) compared with the BCP group (8.2%, $P < 0.0001$) and a lower dose of intraoperative fentanyl was used in the LDP group ($P < 0.0001$).

Hemodynamic data are presented in Figures 1 and 2. The ANOVA statistics revealed that whereas HR and MAP decreased in both the LDP and BCP groups after induction of anesthesia, intraoperative HR and MAP values did not differ between groups. No differences between groups were noted in Spo_2 (Fig. 3), end-tidal sevoflurane concentration (Fig. 4), or BIS (Fig. 5) data throughout the intraoperative period.

$Scto_2$ data are presented in Figure 6 and Table 3. $Scto_2$ values before (75.5 ± 4.0 vs 75.9 ± 3.9) and after (baseline:

Table 1. Patient Characteristics

	Beach chair group	Lateral group	Difference (95% CI)	*P* value
No. of patients	61	63	—	—
Sex (male/female)	38 (62.3%)/23 (37.7%)	40 (63.5%)/23 (36.5%)	−1.2% (−18.0% to 15.6%)	1.000
Age (y)	49.8 ± 13.9	47.8 ± 14.8	1.9 (−3.2 to 7.1)	0.453
Weight (kg)	81.5 ± 20.0	85.3 ± 17.9	−3.8 (−10.5 to 3.0)	0.268
Height (cm)	170.1 ± 9.8	173.3 ± 11.0	−3.1 (−6.8 to 0.6)	0.096
ASA physical status	II (I–III)	II (I–III)	0 (0–0)	0.533
Hemoglobin (g/dL)	14.0 ± 1.5[a]	13.9 ± 1.4[b]	0.1 (−0.5 to 0.6)	0.796
Previous MI	2 (3.3%)	1 (1.6%)	1.7% (−5.5% to 9.8%)	0.616
Arrhythmias	1 (1.6%)	3 (4.8%)	−3.1% (−11.7% to 4.5%)	0.619
Hypertension	26 (42.6%)	23 (36.5%)	6.1% (−11.0% to 22.9%)	0.582
COPD/emphysema	0 (0%)	1 (1.6%)	−1.6% (−8.5% to 4.4%)	1.000
Asthma	9 (14.8%)	6 (9.5%)	5.2% (−6.7% to 17.6%)	0.419
Sleep apnea	5 (8.2%)	4 (6.4%)	1.9% (−8.2% to 12.4%)	0.742
Thyroid disease	3 (4.9%)	5 (7.9%)	−3.0% (−13.1% to 6.6%)	0.718
Diabetes				
Insulin-dependent	0 (0%)	0 (0%)	—	—
Noninsulin-dependent	3 (4.9%)	6 (9.5%)	−4.6% (−15.1% to 5.3%)	0.492
CVA	0 (0%)	0 (0%)	—	—
TIA	0 (0%)	0 (0%)	—	—
Smoking history	7 (11.5%)	8 (12.7%)	−1.2% (−13.3% to 10.9%)	1.000
Drinking history	4 (6.6%)	8 (12.7%)	−6.1% (−17.5% to 4.8%)	0.364

CI = confidence interval; MI = myocardial infarction; COPD = chronic obstructive pulmonary disease; CVA = cerebrovascular accident; TIA = transient ischemic attack.
Data are mean ± SD, median (range), or number of patients (%).
[a] *n* = 60.
[b] *n* = 52.

Table 2. Perioperative Variables

	Beach chair group	Lateral group	Difference (95% CI)	*P* value
No. of patients	61	63	—	—
Interscalene block	5 (8.2%)	45 (71.4%)	−63.2% (−74.6% to −48.5%)	<0.0001
Intraoperative				
Total time (min)	117 ± 31	135 ± 46	−18.0 (−32 to 4)	0.011
Dose fentanyl (μg)	200 (50–500)	100 (0–325)	50 (25–100)	<0.0001
Dose rocuronium (mg)	50 (30–140)	50 (30–155)	0 (0–10)	0.054
Crystalloid (L)	1.11 ± 0.39	1.20 ± 0.38	−0.09 (−0.23 to 0.05)	0.201
Final OR temperature (°C)	35.9 ± 0.6	36.1 ± 0.6	−0.1 (−0.4 to 0.1)	0.224
Times to recovery landmarks (min)				
Open eyes	8 (1–25)	8 (2–16)	0 (−1 to 1)	0.896
Squeeze hands	8 (1–26)	8 (2–22)	1 (0–3)	0.088
Tracheal extubation	10 (2–36)	9 (0–24)	0 (−1 to 2)	0.576
Arrive PACU	17 (5–35)	13 (5–28)	3 (1–5)	<0.001
PACU				
Aldrete scores				
Arrival	8 (2–10)	8 (2–10)[a]	−1 (−1 to 0)	<0.001
30 min	9 (7–10)	9 (2–10)[a]	0 (−1 to 0)	0.050
60 min	9 (8–10)[b]	10 (8–10)[c]	0 (0–0)	0.475
90 min	10 (8–10)[d]	10 (8–10)[e]	0 (0–1)	0.494
Discharge	9 (8–10)[f]	10 (9–10)[g]	−1 (−1 to 0)	0.378
Nausea	24 (39.3%)	5 (8.1%)[a]	31.3% (17.1%–45.1%)	<0.0001
Nausea severity (1–3 scale)	1.5 (1–3)[h]	1 (1–2)[i]	0 (0–1)	0.970
Vomiting	13 (21.3%)	1 (1.6%)[a]	19.7% (9.8%–31.8%)	<0.001
Vomiting severity (1–3 scale)	1 (1–2)[j]	1[k]		—
Pain medication	52 (85.3%)	33 (52.4%)[a]	32.9% (17.0%–47.3%)	0.0001
Pain medication dose (mg hydromorphone)	1.5 (0.5–4)[l]	1 (0.5–4.0)[m]	0 (0–0.5)	0.374
PACU discharge (min)				
Criteria met	80 (35–193)	83.5 (39–145)[a]	−1 (−13 to 10)	0.830
Actual discharge	93 (45–298)	94 (55–181)[a]	2 (−10 to 12)	0.765

CI = confidence interval; OR = operating room; PACU = postanesthesia care unit.
Data are mean ± SD, median (range), or number of patients (%).
[a] *n* = 62, [b] *n* = 58, [c] *n* = 57, [d] *n* = 18, [e] *n* = 27, [f] *n* = 9, [g] *n* = 4, [h] *n* = 24, [i] *n* = 5, [j] *n* = 13, [k] *n* = 1, [l] *n* = 52, [m] *n* = 33.

80.4 ± 5.0 vs 81.1 ± 5.1) induction of anesthesia were similar between the LDP and BCP groups. The ANOVA statistics revealed that $Scto_2$ not only decreased over time but also was lower in the BCP group than the LDP group across time ($P < 0.0001$). The percentage of patients developing a CDE was higher in the BCP group (80.3%) compared with the LDP group (0%, $P < 0.0001$). In addition, the median number of CDEs was greater in the BCP group:

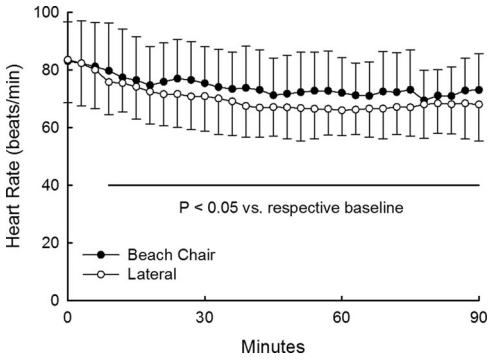

Figure 1. Heart rate (bpm) for the patients in the beach chair position group and in the lateral decubitus position group. The data are presented as mean ± SD. The horizontal line indicates the time during which the heart rates in the patients of the 2 groups differed from their baseline heart rates (9–90 minutes, overall $P < 0.05$). There were no differences between the groups at any time. The number of patients in the beach chair position group decreased from 61 at baseline to 60 at 45 minutes and then progressively over time to 50 at 1 hour and to 20 at 90 minutes, whereas the number of patients in the lateral decubitus position group decreased from 63 at baseline to 61 at 48 minutes and then progressively over time to 54 at 1 hour and to 40 at 90 minutes.

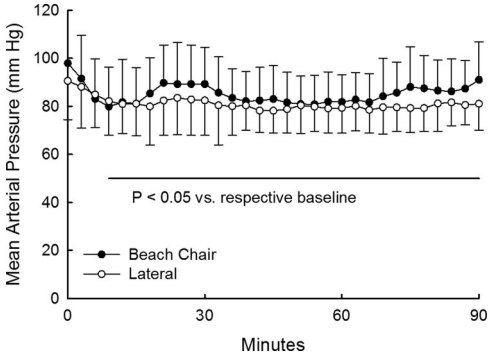

Figure 2. Mean arterial pressure (mm Hg) for the patients in the beach chair position group and in the lateral decubitus position group. The data are presented as mean ± SD. The horizontal line indicates the time during which the blood pressures in the patients of the 2 groups differed from their baseline blood pressures (6–90 minutes, overall $P < 0.05$). There were no differences between the groups at any time. The number of patients in the beach chair position group decreased from 61 at baseline to 60 at 45 minutes and then progressively over time to 51 at 1 hour and to 20 at 90 minutes, whereas the number of patients in the lateral decubitus position group decreased from 63 at baseline to 61 at 48 minutes and then progressively over time to 54 at 1 hour and to 40 at 90 minutes.

≥20% decreases in $Scto_2$ from baseline (4 [0–38] BCP; 0 [0–0] LDP; $P < 0.0001$) and $Scto_2$ ≤55% (0 [0–4] BCP; 0 [0–0] LDP; $P = 0.003$). Furthermore, the median number of interventions required to treat CDEs was also greater in the BCP group (2 [0–11] BCP; 0 [0–0] LDP; $P < 0.0001$). $Scto_2$ values increased after initial treatment interventions in 61% of patients, with most responses occurring within 30 to 45 seconds. Eight patients had 12 episodes of $Scto_2$ ≤55% (all in the BCP group). Nine of the episodes resolved within 1 minute of treatment. The duration of the episodes ranged from 30 seconds to 9 minutes.

Recovery data are presented in Table 2. Lower doses of opioids were used in the LDP group in the operating room

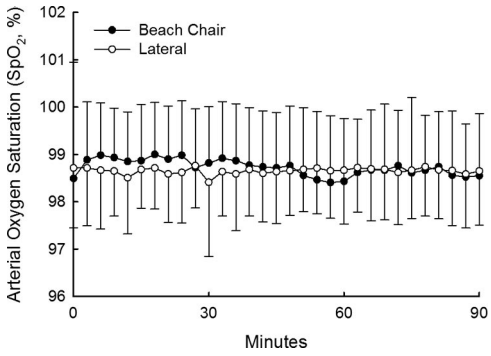

Figure 3. Arterial oxygen saturation (Spo_2, %) for the patients in the beach chair position group and in the lateral decubitus position group. The data are presented as mean ± SD. There were no differences between the groups at any time. The number of patients in the beach chair position group decreased from 61 at baseline to 60 at 45 minutes and then progressively over time to 51 at 1 hour and to 20 at 90 minutes, whereas the number of patients in the lateral decubitus position group decreased from 63 at baseline to 61 at 48 minutes and then progressively over time to 54 at 1 hour and to 40 at 90 minutes.

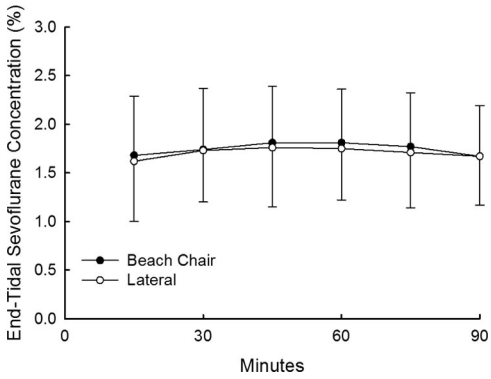

Figure 4. End-tidal sevoflurane concentrations for the patients in the beach chair position group and in the lateral decubitus position group. The data are presented as mean ± SD. There were no differences between the groups at any time. The number of patients in the beach chair position group decreased from 61 at 15 minutes to 60 at 45 minutes, to 58 at 60 minutes, to 45 at 75 minutes, and to 25 at 90 minutes, whereas the number of patients in the lateral decubitus position group decreased from 63 at 15 minutes to 58 at 45 minutes, to 55 at 60 minutes, to 49 at 75 minutes, and to 43 at 90 minutes.

and PACU, which was likely attributable to the higher use of interscalene blocks in this group. The incidence of nausea and vomiting was also lower in this group. The time from the end of the procedure to PACU admission was shorter, and early Aldrete scores were higher, in the LDP group. All other immediate and early recovery variables were similar between groups.

Further analysis was performed to determine the impact of intraoperative CDEs on postoperative recovery (Tables 4 and 5). Data were analyzed only on subjects who did not receive interscalene blocks because use of this technique was not equally distributed between the BCP and LDP groups and interscalene blocks can beneficially influence recovery from anesthesia. Patient characteristics did not differ between subjects with and without CDEs. Perioperative management variables, including opioid dosing in the

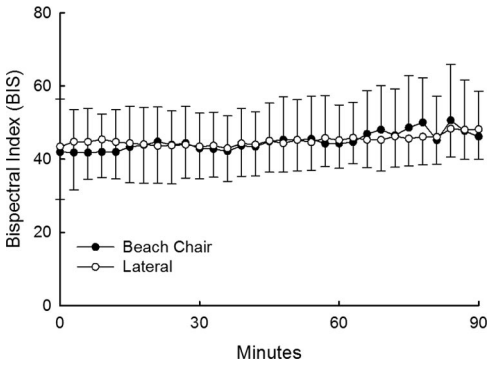

Figure 5. Bispectral index (BIS) for the patients in the beach chair position group and in the lateral decubitus position group. The data are presented as mean ± SD. There were no differences between the groups at any time. The number of patients in the beach chair position group increased from 57 at baseline to 59 at 6 minutes, then decreased to 58 at 45 minutes after which it decreased progressively over time to 49 at 1 hour and to 18 at 90 minutes, whereas the number of patients in the lateral decubitus position group decreased from 63 at baseline to 61 at 48 minutes and then progressively over time to 54 at 1 hour and to 40 at 90 minutes.

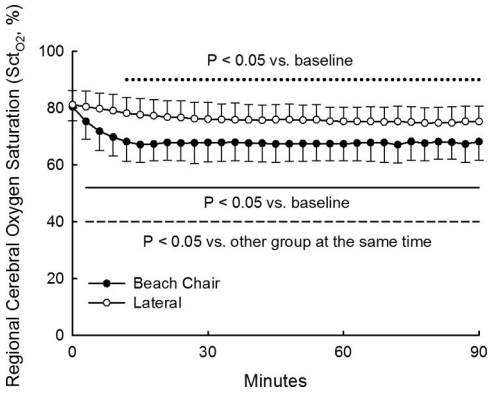

Figure 6. Regional cerebral tissue oxygen saturation ($Scto_2$) for the patients in the beach chair position group and in the lateral decubitus position group. The data are presented as mean ± SD. The solid horizontal line indicates the time during which the $Scto_2$ values in the patients of the beach chair position group differed from their baseline $Scto_2$ values (3– 0 minutes, overall $P < 0.05$) whereas the dotted horizontal line indicates the time during which the $Scto_2$ values in the patients of the lateral decubitus position group differed from their baseline $Scto_2$ values (12– 0 minutes, overall $P < 0.05$). The dashed horizontal line indicates the times during which the $Scto_2$ values in the patients of the beach chair position group differed from those in the lateral decubitus position group (3–90 minutes, overall $P < 0.05$). The number of patients in the beach chair position group decreased from 61 at baseline to 60 at 45 minutes and then progressively over time to 51 at 1 hour and to 20 at 90 minutes, whereas the number of patients in the lateral decubitus position group decreased from 63 at baseline to 61 at 48 minutes and then progressively over time to 54 at 1 hour and to 40 at 90 minutes.

operating room and PACU, also did not differ between groups. A significantly higher incidence of nausea (50.0% vs 6.7%, $P = 0.0001$) and vomiting (27.3% vs 3.3%, $P = 0.011$) was observed in subjects with intraoperative CDEs compared with subjects with no CDE. All other recovery landmarks (with the exception of Aldrete scores on PACU admission) did not differ between groups.

To determine whether confounding variables may be contributing to the nausea, age, sex, smoking status, hemoglobin concentration, operative position, fentanyl dose, end-tidal sevoflurane concentration, and the occurrence of cerebral desaturation were compared between patients who had not had regional anesthesia and did not become nauseated and patients who had not had regional anesthesia but did become nauseated. Variables identified in the univariate analyses as having a value of $P < 0.10$ and included in the initial multiple logistic regression analysis as predictors of nausea were hemoglobin concentration, operative position, and the occurrence of cerebral desaturation. The only variable included in the final multiple logistic regression model as a predictor of nausea was the occurrence of cerebral desaturation (logit $P = -2.639 + 2.639 \times$ [0 = no desaturation, 1 = desaturation]; likelihood ratio test statistic = 17.56, $df = 1$, $P < 0.0001$). The sensitivity of the logistic model was 91.7%, whereas its specificity was 56.0%.

DISCUSSION

Patients undergoing shoulder surgery in the BCP may be at risk for cerebral hypoxia because of decreases in CPP. NIRS technology, which provides information on the balance between oxygen supply and demand in the frontal region of the brain, has not been used previously to determine changes in $Scto_2$ in this patient population. Our results indicate that CDEs, defined as a ≥20% decrease in $Scto_2$ values from baseline measurements or an $Scto_2$ value of ≤55%, occurred frequently in patients having arthroscopic surgery in the BCP. Despite the use of a protocol designed to optimize CPP, CDEs were observed in 80.3% of subjects. In contrast, no CDEs were noted in a similar cohort of patients undergoing shoulder arthroscopy in the LDP. An association between intraoperative CDEs and postoperative nausea and vomiting was also observed.

Alterations in systemic hemodynamics occur when postural conditions are changed from supine to sitting. Reductions in cardiac output, MAP, and CPP may subsequently compromise cerebral oxygen delivery. In awake volunteers, assumption of a head-up posture results in a 10% to 15% increase in HR, a 5- to 10-second decrease in MAP and

Table 3. Primary Outcome Variables

	Beach chair group	Lateral group	Difference or median difference (95% CI)	*P* value
No. of patients	61	63	—	—
Patients with cerebral desaturation events	49 (80.3%)	0 (0%)	80.3% (68.7%–88.4%)	<0.0001
Interventions for $Scto_2$ decreases	2 (0–11)	0 (0–0)	2 (2–3)	<0.0001
Interventions for MAP decreases	1 (0–6)	0 (0–9)	0 (0–1)	0.008
Episodes $Scto_2$ ≤55	0 (0–4)	0 (0–0)	0 (0–0)	0.003
Episodes ≥20% decrease $Scto_2$	4 (0–38)	0 (0–0)	4 (2–5)	<0.0001

CI = confidence interval; $Scto_2$ = regional cerebral tissue oxygen saturation; MAP = mean arterial blood pressure.
Data are number of patients (%) or median (range).

Table 4. Patient Characteristics

	No interscalene block, no cerebral desaturation events group	No interscalene block, cerebral desaturation events group	Difference (95% CI)	*P* value
No. of patients	30	44	—	—
Sex (male/female)	18 (60.0%)/12 (40.0%)	28 (63.6%)/16 (36.4%)	−3.6% (−25.9% to 18.2%)	0.810
Age (y)	50.5 ± 12.4	48.8 ± 14.7	1.7 (−4.8 to 8.2)	0.601
Weight (kg)	85.4 ± 20.8	81.5 ± 20.4	3.9 (−5.8 to 13.6)	0.426
Height (cm)	172.9 ± 9.7	170.5 ± 10.5	2.4 (−2.4 to 7.2)	0.320
ASA physical status	2 (1–3)	2 (1–3)	0 (0–1)	0.066
Hemoglobin (g/dL)	13.8 ± 1.4[a]	13.8 ± 1.6[b]	−0.1 (−0.8 to 0.7)	0.847
Previous MI	2 (6.7%)	0 (0%)	6.7% (−1.7% to 21.3%)	0.161
Arrhythmias	1 (3.3%)	1 (2.3%)	1.1% (−8.9% to 14.6%)	1.000
Hypertension	11 (36.7%)	18 (40.9%)	−4.2% (−25.7% to 18.4%)	0.810
COPD/emphysema	1 (3.3%)	0 (0%)	3.3% (−4.9% to 16.7%)	0.405
Asthma	5 (16.7%)	6 (13.6%)	3.0% (−13.3% to 21.8%)	0.749
Sleep apnea	4 (13.3%)	4 (9.1%)	4.2% (−10.4% to 21.8%)	0.707
Thyroid disease	0 (0%)	3 (6.8%)	−6.8% (−18.2% to 4.9%)	0.267
Diabetes				
Insulin-dependent	0 (0%)	0 (0%)	—	—
Noninsulin-dependent	2 (6.7%)	3 (6.8%)	−0.2% (−12.8% to 15.3%)	1.000
CVA	0 (0%)	0 (0%)	—	—
TIA	0 (0%)	0 (0%)	—	—
Smoking history	4 (13.3%)	2 (4.6%)	8.8% (−4.3% to 25.8%)	0.215
Drinking history	5 (16.7%)	2 (4.6%)	12.1% (−1.6% to 29.7%)	0.112

CI = confidence interval; MI = myocardial infarction; COPD = chronic obstructive pulmonary disease; CVA = cerebrovascular accident; TIA = transient ischemic attack.

Data are mean ± SD, median (range), or number of patients (%).

[a] *n* = 29.

[b] *n* = 43.

Table 5. Perioperative Variables

	No interscalene block, no cerebral desaturation events group	No interscalene block, cerebral desaturation events group	Difference (95% CI)	*P* value
No. of patients	30	44	—	—
Lateral position	18 (60.0%)	0 (0%)	60.0% (74.1%–90.6%)	<0.0001
Intraoperative				
Total time (min)	130 ± 44	120 ± 32	10 (−7.8 to 27.4)	0.300
Dose fentanyl (μg)	200 (200–325)	200 (100–500)	0 (−50 to 25)	0.717
Dose rocuronium (mg)	50 (30–100)	50 (30–140)	0 (−10 to 0)	0.544
Crystalloid (L)	1.25 ± 0.46	1.13 ± 0.41	0.13 (−0.07 to 0.33)	0.212
Final OR temperature (°C)	35.8 ± 0.7	36.0 ± 0.6	−0.2 (−0.5 to 0.1)	0.139
Times to recovery landmarks (min)				
Open eyes	7 (2–16)	8 (1–25)	0 (−2 to 1)	0.275
Squeeze hands	7 (2–22)	9 (1–26)	−1 (−2 to 1)	0.201
Tracheal extubation	9 (4–24)	10 (2–36)	−1 (−3 to 1)	0.114
Arrive PACU	16 (8–35)	18 (5–35)	−2 (−4 to 1)	0.09
PACU				
Aldrete scores				
Arrival	8 (7–10)	8 (2–10)	0 (0–1)	0.041
30 min	9 (8–10)	9 (7–10)	0 (0–1)	0.054
60 min	10 (8–10)[a]	9 (8–10)[b]	0 (0–1)	0.242
90 min	9 (9–10)[c]	10 (8–10)[d]	0 (−1 to 0)	0.609
Discharge	9.5 (9–10)[e]	9 (8–10)[f]	0.5 (0–1)	0.495
Nausea	2 (6.7%)	22 (50.0%)	−43.3% (−59.1% to −24.0%)	0.0001
Nausea severity (1–3 scale)	1[g]	1.5 (1–3)[h]	—	—
Vomiting	1 (3.3%)	12 (27.3%)	−23.9% (−39.3% to −7.8%)	0.011
Vomiting severity (1–3 scale)	1[i]	1 (1–2)[j]	—	—
Pain medication	28 (93.3%)	39 (88.6%)	4.7% (−11.3% to 18.6%)	0.694
Pain medication dose (mg hydromorphone)	1.5 (0.5–4.0)[k]	1.5 (0.5–4.0)[l]	0 (0–0.5)	0.299
PACU discharge (min)				
Criteria met	88 (45–145)	86.5 (35–193)	2.5 (−12 to 17)	0.704
Actual discharge	114 (65–180)	99.5 (50–298)	2.5 (−12 to 22)	0.700

CI = confidence interval; OR = operating room; PACU = postanesthesia care unit.

Data are mean ± SD, median (range), or number of patients (%).

[a] *n* = 29, [b] *n* = 43, [c] *n* = 16, [d] *n* = 12, [e] *n* = 4, [f] *n* = 6, [g] *n* = 2, [h] *n*= 22, [i] *n*= 1, [j] *n* = 12, [k] *n* = 28, [l] *n* = 39.

systemic vascular resistance followed by a rapid rebound and overshoot (15%–40%), and a sustained reduction in thoracic blood volume and cardiac output (15%–30%).[8–10] Compensatory increases in sympathetic nervous system activation and systemic vascular resistance to maintain systemic perfusion pressures are attenuated under general anesthesia. Raising anesthetized neurosurgical patients from the supine to the sitting position resulted in significant reductions in cardiac output, MAP, and CPP, which persisted for up to 30 minutes after positioning.[11,12]

The influence of alterations in positioning on cerebral oxygenation in anesthetized surgical patients has been examined in only 1 previous trial. In these 12 subjects, no changes in $Scto_2$ values were observed in the LDP, but a small (6%), statistically significant decrease in $Scto_2$ occurred after assuming the sitting position.[19] However, cerebral oximetry data were only collected for 5 minutes after each change in position. In the present investigation, clinically significant reductions in $Scto_2$ values were absent when position was altered from supine to right or left lateral decubitus; $Scto_2$ remained near baseline measures throughout the operative procedure. No episodes of CDEs were recorded in any of the 63 LDP subjects, and no interventions to treat low $Scto_2$ were required. In contrast, pronounced reductions in $Scto_2$ were observed in the BCP group. $Scto_2$ decreased from a baseline mean of 80% to mean values of <70% by 9 minutes, and the mean remained between 66% and 70% throughout the operative procedure. The median number of CDEs was significantly higher in the BCP group, despite the use of a similar protocol to maintain MAP. In addition, more interventions to treat reductions in $Scto_2$ were required in the BCP group. These findings suggest that CDEs (as defined in this investigation) occur frequently during sitting position surgery and that cerebral oxygenation in the frontal cortices may potentially be compromised in these patients undergoing general anesthesia.

Hemodynamic and systemic oxygenation variables (HR, MAP, Spo_2) were not different between the BCP and LDP groups throughout the intraoperative period. HR and MAP decreased from initial values in both groups over time, but were within 20% of baseline measures. MAP is a primary determinant of CPP and oxygenation, and reductions in MAP are associated with comparable decreases in $Scto_2$.[15,20] MAP values, measured at the brachial artery, did not differ between the 2 groups of patients. It is possible, however, that MAP measured at the brachial artery may overestimate the actual pressure at the level of the brain when the sitting position is assumed. According to the "open model" or "waterfall" theory, as blood flows vertically from the heart, there is a reduction in arterial pressure directly related to the weight of the column of blood.[5,21] When the sitting position is used, an arithmetic correction of MAPs obtained from other sites is required to determine blood pressure at the level of the brain (1 mm Hg for each 1.35 cm).[5,21] If the average vertical distance between the brachial artery measurement site and cerebral oximeter probe was 38 cm (measured in 10 sitting patients), and the average MAP intraoperatively was 80 mm Hg, a "corrected" MAP value of 51.8 mm Hg at the level of the frontal cortex is derived. This "corrected" pressure might represent an inadequate CPP in some subjects. Although the lower limit of cerebral autoregulation is generally accepted to be a MAP of approximately 50 mm Hg, some studies have demonstrated that this lower threshold may be as high as 70 to 80 mm Hg in awake, normotensive subjects.[22] Consequently, if the "waterfall" theory is correct, maintenance of a MAP of 80 mm Hg using a manual blood pressure cuff might have represented suboptimal blood pressure management and accounted for the lower $Scto_2$ values in the BCP group.

Despite the frequent occurrence of CDEs in patients undergoing surgery in the sitting position, no obvious neurologic deficits were observed in this study cohort. This finding is not unexpected because major adverse cerebrovascular events after orthopedic procedures in the BCP have been rarely described in the literature. Only 13 cases of stroke, coma, or blindness have been reported in this patient population.[3,5,6] At the present time, the incidence of permanent neurologic events after BCP surgery has not been assessed in a prospective or retrospective investigation. However, a survey of the membership of the American Shoulder and Elbow Surgeons yielded an estimated rate of stroke of 0.00382% to 0.00461% during shoulder surgery, with all events occurring in the BCP.[3] The low incidence of adverse neurologic outcomes is likely related to the relatively limited duration of the surgical procedure. Severity and duration of ischemia are critical determinants of tissue damage, and viability-time thresholds must be exceeded to produce stroke. In a pig model, low $Scto_2$ values that persisted for <2 hours did not result in neurologic injury.[23] An analysis of NIRS data from 265 coronary artery bypass graft patients revealed a desaturation-time threshold of 50 minutes that was associated with cognitive decline and longer hospital length of stay.[24] In the present investigation, the duration of CDEs was limited, with no CDEs exceeding previously defined viability-time thresholds. However, the degree and duration of cerebral ischemia required to produce overt neurologic symptoms in a relatively healthy patient population is unknown at the present time. The use of a protocol designed to detect, treat, and reduce the duration of CDEs (as used in this study) would likely minimize the risk of obvious neurologic deficits.

A number of recovery variables were assessed in the PACU to determine whether CDEs in the operating room were associated with impaired early recovery from anesthesia and surgery. A larger number of patients in the LDP group received interscalene blocks, per surgeon preference. The use of regional anesthesia may facilitate recovery from anesthesia and surgery; therefore, we performed an analysis only on subjects not administered regional anesthesia. The times required to achieve immediate recovery landmarks (time to open eyes, squeeze hand, tracheal extubation, and arrive in the PACU) and meet PACU recovery criteria did not differ between patients with and without CDEs. However, an approximately 7-fold higher incidence of both nausea and vomiting was observed in subjects with CDEs. Some authors have suggested that an important perioperative cause of nausea and vomiting is anesthetic-induced systemic hypotension, which produces a reduction

in cerebral perfusion and oxygenation.[25] In patients undergoing prostate resection surgery, spinal anesthesia resulted in decreases in CPP and oxygenation, and an association between intraoperative CDEs and nausea at the end of surgery was observed.[26] Our findings provide further support for an association between CDEs in the operating room and nausea and vomiting during early recovery.

A CDE was defined in this investigation as a ≥20% decrease in $Scto_2$ values from baseline measures or an $Scto_2$ of ≤55%. At the present time, there is not a universally accepted threshold used to identify pathological cerebral saturation. The threshold for identifying cerebral ischemia may be influenced by a number of patient-specific (presence of cerebrovascular disease, incomplete circle of Willis) or technology-dependent variables. Because of wide patient-to-patient variability in baseline $Scto_2$ values, some authors recommend monitoring changes from baseline measurements; a reduction of 15% to 20% from baseline has been used as a critical threshold in many investigations. In awake patients undergoing carotid endarterectomy, a 20% decrease in $Scto_2$ was associated with symptoms of cerebral ischemia.[27] In another group of carotid endarterectomy patients, a 15% to 20% decrease in $Scto_2$ was associated with a 20-fold increase in the odds for developing cerebral ischemia on electroencephalography.[28] In addition, 15% to 25% decreases in $Scto_2$ have been significantly correlated with cognitive dysfunction after cardiac surgery,[29] longer PACU and hospital admissions after abdominal surgery,[30] and greater release of biochemical markers of brain injury after liver transplantation.[31] Preclinical studies suggest that when quantitative NIRS technology is used, absolute $Scto_2$ values of ≤55% represent cerebral ischemia.[18,32] Recent studies have demonstrated that FORE-SIGHT–derived values below this threshold were associated with adverse outcomes after cardiac and aortic surgery.[29,33]

There are several limitations to the present investigation. First, NIRS devices measure saturation in an uncertain mix of arterial, venous, and capillary compartments. In the supine position, the venous contribution to cerebral oximetry predominates, with 70% to 84% of $Scto_2$ values determined by venous blood.[34,35] Changes in body position may alter venous and arterial blood pressure and affect the ratio of the compartments in the cerebral circulation.[36] Therefore, reductions in $Scto_2$ may not only reflect decreases in oxygen supply but also changes in cerebral blood volumes/compartments. Second, baseline $Scto_2$ values used to define a clinically significant reduction in cerebral oxygenation were measured after induction of anesthesia, because we were interested in assessing position-related changes in $Scto_2$ under identical anesthetic conditions (similar Fio_2, MAP, $Etco_2$, and BIS values). At the present time, there is no consensus on the setting under which baseline measures should be obtained; previous investigators have collected these data before[24,30] and after[37,38] induction of anesthesia. We believe that assessing baseline and subsequent $Scto_2$ measures under very different physiologic conditions (awake versus anesthetized) would make the determination of changes in $Scto_2$ due to alterations in patient positioning extremely difficult. Third, neurocognitive testing to assess the presence or absence of subtle neurologic dysfunction potentially related to CDEs was not performed. In addition, transcranial Doppler, which can be used to indirectly measure cerebral blood flow changes related to alterations in position, was not used intraoperatively. Fourth, arterial carbon dioxide levels were not measured (ventilation was determined on the basis of $Etco_2$ values). Large inter- and intraindividual variations in arterial to $Etco_2$ gradients have been reported, and the degree of this variability may be influenced by patient positioning.[39] Finally, cerebral oxygenation data were recorded using the FORE-SIGHT cerebral oximeter. Another Food and Drug Administration–approved NIRS device (INVOS; Somanetics Corp., Troy, MI) has been used in the majority of previous perioperative studies, and there are fewer data supporting a beneficial effect of the FORE-SIGHT cerebral oximeter on clinical outcomes. However, a recent volunteer study has demonstrated that the FORE-SIGHT monitor has greater precision with respect to measuring absolute $Scto_2$ than the INVOS monitor.[32]

In conclusion, our findings demonstrate that significant reductions in $Scto_2$ occur when position is changed from supine to sitting in patients undergoing general anesthesia. These changes occurred despite the use of a protocol designed to maintain systemic MAP within 20% of baseline values. Furthermore, intraoperative CDEs were associated with a higher incidence of nausea and vomiting in the PACU. Future larger-scale investigations are required to define the degree and duration of reduction in $Scto_2$ associated with permanent neurologic injury.

AUTHOR CONTRIBUTIONS

GSM helped design and conduct the study, analyze the data, and write the manuscript. This author has seen the original study data, reviewed the analysis of the data, approved the final manuscript, and is the author responsible for archiving the study files. JWS helped design and conduct the study and write the manuscript. This author approved the final manuscript. JHM helped design and conduct the study. This author approved the final manuscript. SBG helped conduct the study. This author approved the final manuscript. MJA helped design the study, analyze the data, and write the manuscript. This author has seen the original study data, reviewed the analysis of the data, and approved the final manuscript. JSV helped design the study. This author approved the final manuscript. JV helped conduct the study. This author approved the final manuscript. MN helped conduct the study. This author has seen the original study data and approved the final manuscript.

DISCLOSURE

GSM received honoraria from CASMED. All other authors report no conflicts of interest.

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