Cerebral Oxygen Saturation Measured by Near-infrared Spectroscopy and Jugular Venous Bulb Oxygen Saturation during Arthroscopic Shoulder Surgery in Beach Chair Position under Sevoflurane-Nitrous Oxide or Propofol-Remifentanil Anesthesia

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ABSTRACT

Background: We examined the effects of different anesthetics on cerebral oxygenation and systemic hemodynamics in patients undergoing surgery in beach chair position (BCP). Jugular venous bulb oxygen saturation (SjvO2) and regional cerebral tissue oxygen saturation (SctO2) were determined while patients were placed from the supine to BCP. Whether SctO2 and SjvO2 are interchangeable in assessing the cerebral oxygenation was also examined.

Methods: Forty patients undergoing shoulder surgery in BCP were randomly assigned to receive sevoflurane-nitrous oxide (S/N) or propofol-remifentanil (P/R) anesthesia. Four patients taking angiotensin II receptor antagonists were excluded post hoc. Mean arterial pressure and heart rate, as well as SjvO2 and SctO2, were measured before (postinduction baseline in supine position) and after BCP.

Results: Mean arterial pressure decreased by BCP in both groups. It was, however, significantly higher in S/N (n = 19) than in P/R group (n = 17) at 7 to 8 min after the positioning. SjvO2 also significantly decreased after BCP in both groups, the magnitude of which was lower in S/N than in P/R group (11 ± 10% vs. 23 ± 9%, P = 0.0006). The incidences of SjvO2 <50% and mean arterial pressure less than 50 mmHg were lower in S/N group, but SctO2 and the incidence of cerebral desaturation (more than 20% decrease from baseline) did not significantly differ between the groups. SctO2 and SjvO2 were only weakly correlated (β = 0.218, r² = 0.133). Bland-Altman analysis showed a mean difference of −7.2% with 95% limit of agreement between −38.2% and 23.8%.

Conclusions: The margin of safety against impaired cerebral oxygenation is greater and SjvO2 is more preserved with S/N than with P/R anesthesia. SctO2 may not be reliable in detecting a low SjvO2 during the surgery in BCP.

What We Already Know about This Topic
- The beach chair position (BCP) carries a risk of cerebral ischemia.

What This Article Tells Us That Is New
- Using jugular venous bulb oxygen saturation (SjvO2) and regional cerebral tissue oxygen saturation (SctO2) monitoring in patients changed from the supine to BCP, the margin of safety against impaired cerebral oxygenation was greater and SjvO2 better preserved with sevoflurane-nitrous oxide than with propofol-remifentanil anesthesia. SctO2 may not be reliable in detecting a low SjvO2 during the surgery in BCP.

The beach chair position (BCP) is commonly used in arthroscopic and open shoulder procedures because of its numerous advantages, such as reduced direct neurovascular trauma, excellent intraarticular visualization, and ease of conversion to an open approach if needed compared with the lateral decubitus approach.1,2 However, it is associated with reductions in cardiac output, mean arterial pressure (MAP), and cerebral perfusion pressure because of the gravitational effect of positioning the head above the level of the heart.3,4 Sudden profound hypotensive and bradycardic events have been reported in more than 20% of patients undergoing shoulder arthroscopy in BCP.5 Systemic hypotension may compromise cerebral perfusion, resulting in a neurologic injury when the episode is prolonged. Brain and spinal cord ischemia,6,7 transient visual loss, ophthalmoplegia,8 and se-
vere cerebral desaturation events\textsuperscript{9,10} have been documented during a shoulder surgery in BCP.

Sevoflurane and propofol are widely used during orthopedic and neurosurgery. Although they similarly reduce the cerebral metabolic rate for oxygen (\CMRO\textsubscript{2}), their effects on cerebral blood flow (\CBF) may differ. During sevoflurane administration, because of its intrinsic cerebral vasodilator effect, \CBF is in excess relative to the cerebral oxygen demand.\textsuperscript{11} In contrast, propofol reduces \CBF greater than \CMRO\textsubscript{2}, resulting in a decrease of the \CBF/\CMRO\textsubscript{2} ratio.\textsuperscript{12–14} Indeed, cerebral oxygen balance is better maintained by sevoflurane-based than by propofol-based anesthesia when patients are positioned supine.\textsuperscript{15–19} In this context, it is also possible that the margin of safety against an impaired cerebral oxygenation will be greater with sevoflurane-based than with propofol-based anesthesia in BCP. The choice of anesthetic technique may also influence hemodynamic stability in the sitting position.\textsuperscript{19} However, cerebral oxygen balance and systemic hemodynamics have not been examined when surgery is performed in BCP under general anesthesia.

Jugular venous bulb oxygen saturation (\SjO\textsubscript{2}) well reflects global \CBF/\CMRO\textsubscript{2} ratio and is used as an indirect marker of global cerebral oxygen use (i.e., cerebral oxygenation) in a variety of clinical settings.\textsuperscript{20} SjO\textsubscript{2} monitoring is invasive and difficult to use, and it may overlook regional differences in cerebral oxygenation. Near-infrared spectroscopy (NIRS) is a noninvasive technique that provides continuous monitoring of regional cerebral tissue oxygen saturation (\ScO\textsubscript{2}). It has been successfully used to assess the adequacy of cerebral oxygen delivery to demand in patients undergoing procedures at high risk of adverse neurologic outcomes (cardiac, vascular, liver transplant, and major abdominal surgery).\textsuperscript{21,22}

The current study was aimed at examining if sevoflurane-nitrous oxide (\S/N) anesthesia would better maintain cerebral oxygenation than propofol-remifentanil (\P/R) anesthesia. Whether \ScO\textsubscript{2} may be an alternative to \SjO\textsubscript{2} in assessing the cerebral oxygenation during surgery in BCP was also determined.

Materials and Methods

The study protocol was approved by the Chonnam National University Hospital Ethics Committee (Gwangju, Korea), and all patients gave informed consent. Forty-two patients scheduled to undergo elective arthroscopic shoulder surgery under general anesthesia in BCP were enrolled. They were assigned to either the \S/N or \P/R group, based on a computer-generated randomization list. Exclusion criteria included preexisting cerebrovascular diseases, history of orthostatic hypotension, age less than 18 yr, and the American Society of Anesthesiologists physical status IV or V. Patients taking angiotensin II receptor antagonists were in addition excluded post hoc.

All patients were premedicated with midazolam (0.1 mg/kg, orally) 60 min before induction of anesthesia. Upon arrival in the operating room, a 20-gauge catheter was placed into a radial artery and connected to a pressure transducer (Deltran; Utah Medical Products, Midvale, UT) to monitor blood pressure and to take blood samples. The pressure transducer was referenced to the mid-axillary level when patients were supine and referenced to the external ear canal level when in BCP. In sitting position, an arithmetic correction of MAP measured at other sites is required to determine the blood pressure at the level of the brain (1 mmHg for each 1.35 cm).\textsuperscript{6,23} Instead, in the current study, the pressure transducer was referenced to the external ear canal level in BCP. A standard Bispectral Index (BIS\textsuperscript{®}) electrode montage (Aspect Medical Systems, Natick, MA) was applied to the forehead before induction of anesthesia, and BIS was measured continuously using an Aspect BIS\textsuperscript{®} A-2000 monitor version 3.31 (Aspect Medical Systems). The esophageal temperature was monitored continuously (Mon-a-Therm; Mallinckrodt, St. Louis, MO) and maintained at 35.5°–36.5°C using a warming blanket. SctO\textsubscript{2} was monitored by NIRS with an INVOS\textsuperscript{®} 5100B cerebral oximeter (Somanetics, Troy, MI). The cerebral oximeter probes were placed on the right and left forehead, with the caudal border about 1 cm above the eyebrow with the medial edge at the midline. This position places the light source and sensors away from the frontal sinus. SctO\textsubscript{2} values from the right and left frontal lobes were averaged to determine cerebral oxygenation.

After measurements of the preinduction values (MAP, heart rate, BIS, and SctO\textsubscript{2}) and full preoxygenation, anesthesia was induced with propofol (2.0–2.5 mg/kg) and remifentanil (1.0–2.0 \mu g/kg) in the \S/N group. In the \P/R group, anesthesia was induced with an effect-site target-controlled infusion (TCI) of remifentanil set at 3.0 ng/ml (starting with a 1 \mu g/kg bolus over 60 s) and propofol set at 3.0 \mu g/ml (starting with a 2.0 mg/kg bolus over 60 s). After administration of rocuronium 0.8 mg/kg IV, the trachea was intubated and the lungs were mechanically ventilated with 50% \textsubscript{N}\textsubscript{2}O in oxygen in the \S/N group, and with air and oxygen at 0.5 fraction of inspired oxygen in the \P/R group. Sevoflurane concentrations combined with 50% \textsubscript{N}\textsubscript{2}O in oxygen were then adjusted to maintain MAP within 20% of preinduction values and BIS values between 40 and 50 throughout surgery after a 5 min “washin” period whereby 3% sevoflurane (inspired) was administered in the \S/N group. TCI effect-site concentrations of propofol were also adjusted to achieve a BIS reading of 40–50, and those of remifentanil were adjusted to maintain MAP within 20% of the preinduction value in the \P/R group. For continuous monitoring of \SjO\textsubscript{2} and blood sampling, a central venous oximetry catheter (Pre-Sep\textsuperscript{TM} Oximetry Catheter; Edwards Lifesciences, Irvine, CA) connected to a Vigileo\textsuperscript{TM} monitor (Edwards Lifesciences) was placed retrogradely in the jugular bulb contralateral to the side of surgery. Proper positioning of the catheter was verified radiographically. The system was calibrated with a blood gas/electrolyte analyzer (GEM\textsuperscript{®} Premier
and remifentanil hydrochloride (Ultiva®; GlaxoSmithKline) were defined as MAP less than 50 mmHg, measured at the level of the external auditory canal. When hypotension occurred, it was treated with a bolus of ephedrine (8 mg) and rapid fluid infusion. Vasopressor treatment was repeated every 2 min if hypotension persisted or recurred. The incidences of cerebral and jugular bulb oxygen desaturation and hypotension were recorded. Upon completion of the surgery, the anesthetic was discontinued, and residual neuromuscular block was antagonized with neostigmine and glycopyrrolate. Estimated blood loss and amounts of fluid or blood administered during the surgery were recorded. At a postoperative visit on the evening of surgery by a surgeon who was not informed about the purpose of the study, the patient was assessed neurologically by gross motor and sensory neurologic evaluation and gross cognitive evaluation (orientation in time and space, recall of name, date of birth, and address). Any side effects were recorded. All anesthetic procedures were conducted by an anesthesiologist, and data were assessed by a person not involved in anesthetic care.

Statistical Analysis
Sample size was calculated to detect 10% difference of the lowest SjvO2 value observed within 10 min after the BCP between the groups. Based on our pilot study (n = 5 each), 17 patients per group were required to detect an effect size of 0.89 from an analysis that used an independent Student t test with an α of 0.05 and a power of 0.80. Taking into account possible dropouts, we aimed to recruit about 20 patients in each group. Sample size was determined by “G power.”

Data are expressed as number or mean ± SD. They were analyzed using StatView software version 4.0 (Abacus Concepts Inc., Berkeley, CA). The patient characteristics and complication rates were compared using the Student unpaired t tests or the chi-square test. Serial changes in cardiovascular, SctO2, SjvO2, and BIS data were analyzed using a two-way ANOVA with repeated measures, with time as a within-group factor, group (SN/PR) as a between-group factor, and the interaction between time and group was also compared. Scheffé F test was used for multiple pair-wise comparisons when a significant difference was indicated with analysis of variance. All statistical tests were two-tailed. Paired measurements of SctO2 and SjvO2 were compared using linear regression analysis, and a Bland-Altman plot was used to graphically compare the agreement of two measurements of SctO2 and SjvO2 using SPSS 17.0 for Windows (SPSS Inc., Chicago, IL). We decided that ±5% would be a clinically acceptable difference between the two methods (SjvO2 and SctO2) while still supporting the conclusion that the two methods are interchangeable. A P value < 0.05 was considered statistically significant.

Results
Of the 42 patients who were enrolled in the study, two were excluded because of unsuccessful cannulation of the internal jugular vein. Four patients (one in S/N group and three in P/R group) were taking angiotensin II receptor antagonists,
and were in addition excluded from the final analysis, because the three in the P/R group showed very low postinduction MAP (62, 65, and 62 mmHg) on *post hoc* analysis. There were no differences in demographic or surgical data between the groups (table 1). Most surgeries were for full-thickness rotator cuff tears, shoulder instability, or shoulder impingement syndrome. Seven patients had the fiberoptic catheter placed in the right jugular bulb and the remaining 12 patients had it placed in the left side in S/N group, whereas the catheter was placed in the right side in five patients and in the left side in the remaining 12 patients in P/R group. Total anesthesia and operative times, intraoperative fluid requirements, and blood loss were not different between the groups.

Table 2 shows preoperative hemodynamic and peripheral oxygen saturation, and intraoperative blood gas data. There were no statistically significant differences in these variables between the groups. BIS (mean ± SD) did not differ between the groups throughout surgery (45.8 ± 6.2 in S/N and 44.5 ± 6.4 in P/R group). The end-tidal sevoflurane concentrations ranged between 1.3% and 1.9% in S/N group during surgery. Effect-site TCI concentrations of propofol ranged between 2.1 µg/ml and 3.0 µg/ml, and those of remifentanil between 1.9 ng/ml and 3.2 ng/ml. None of the variables, and blood loss were not different between the groups.

| Table 2. Preoperative Hemodynamic and Intraoperative Blood Gas Data |
|----------------------|----------------------|--------------|
|                      | S/N Group (n = 19)   | P/R Group (n = 17) |
| Mean arterial pressure, mmHg | 99 ± 14             | 97 ± 11 | 0.653 |
| Heart rate, beats/min | 68 ± 6              | 68 ± 7 | 0.488 |
| SpO₂, %              | 97 ± 2              | 97 ± 2 | 0.361 |
| SctO₂, %             | 68 ± 6              | 68 ± 7 | 0.801 |
| PaCO₂, mmHg          | 40 ± 3              | 39 ± 2 | 0.301 |
| PaO₂, mmHg           | 220 ± 38            | 202 ± 56 | 0.219 |

Data are mean ± SD or numbers.

P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia.

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The number of patients in the sevoflurane-nitrous oxide group decreased from 19 at baseline to 17 at 90 min, whereas the number of patients in the propofol-remifentanil group decreased from 17 at baseline to 15 at 90 min. P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia; SjvO₂ = jugular venous oxygen saturation.

MAP was decreased after induction of anesthesia in both groups; however, post hoc analysis showed that it was higher in S/N than in P/R group (89 ± 12 vs. 80 ± 13 mmHg, P = 0.0364). Four patients taking angiotensin II receptor antagonists were thus excluded, resulting in similar postinduction baseline values between the two groups (88 ± 12 vs. 82 ± 12 mmHg, P = 0.124). MAP further decreased by BCP for 20 min in both groups (P < 0.0001). However, MAP was higher in S/N than in P/R group at 7 to 8 min after BCP (P = 0.0212) despite the less frequent use of vasopressors in the former. Heart rate did not differ between the groups before induction of anesthesia (68 ± 11 vs. 66 ± 11 beats/min). It was increased by induction of anesthesia and then decreased from 3 min after the positioning in S/N group (P = 0.0001), whereas it remained unaltered in P/R group. However, heart rate did not differ between the groups throughout the study, although it tended to be lower in S/N group at 13–20 min into postural change, thus revealing time by group interaction (P = 0.0017).

SjvO₂ before (postinduction baseline in the supine position) and after BCP are presented in figure 2. Figure 3 shows individual values of SjvO₂. Baseline values were higher in the S/N than in the P/R group (74 ± 8% vs. 65 ± 9%, P = 0.0048). They decreased significantly after the positioning in both groups, but the magnitude of which was smaller in the S/N than in the P/R group (11 ± 10% vs. 23 ± 9%, P = 0.0006), with the lowest values within 10 min being 63 ± 13% and 42 ± 14%, respectively. Of the 38 patients studied, 15 (three in S/N group and 12 in P/R group) showed SjvO₂ of less than 50%. Five patients with SjvO₂ of less than 40% were all from the P/R group.

SctO₂ values are presented in figure 4. They were comparable between the groups before induction of anesthesia. Postinduction baseline values before BCP also did not differ between the groups (78 ± 9% vs. 72 ± 10% in the S/N and P/R groups, respectively). The two-way ANOVA revealed that SctO₂ decreased over time after BCP in both groups (P < 0.0001), with no intergroup differences across time (P = 0.959).

The adverse effects are presented in figure 5. The incidence of hypotension (MAP less than 50 mmHg, measured at the level of the external auditory canal) was lower in the
S/N than in the P/R group, along with a less frequent use of ephedrine (26% vs. 71%, respectively, \(P < 0.043\)). One patient in the P/R group received ephedrine three times. However, total dosage of ephedrine did not differ between the groups. The duration of the hypotensive episodes ranged from 30 s to 15 min. The incidence of SjvO₂ of less than 50% was also significantly lower in the S/N than in the P/R group (16% vs. 71%, \(P = 0.0009\)). There were no differences in the incidence of cerebral oxygen desaturation (more than 20% decrease of SctO₂ from baseline) between the groups (\(P = 0.836\)). The duration of the desaturation episode ranged from 2 min to 13 min in the S/N group, and from 1 min to 1 h or longer in the P/R group. Of 15 patients with SjvO₂ of less than 50% in all, four (27%) had both hypotension and cerebral desaturation, four (27%) had hypotension alone, and one (7%) had cerebral desaturation alone, whereas the remaining six (40%) had neither hypotension nor cerebral desaturation.

To determine whether SctO₂ reflects SjvO₂, a total of 726 paired measurements of both values from 36 patients were compared using linear regression and Bland-Altman analyses. SjvO₂ and SctO₂ showed a significant but weak correlation (\(\beta = 0.218, r^2 = 0.133, P = 0.0001\)) (fig. 6). Figure 7 shows Bland-Altman plot depicting the difference between SjvO₂ and SctO₂ (Y-axis) against their means (X-axis) for all patients. The mean difference (bias) between the two measurements was \(-7.2\%\) with 95% limit of agreement (\(-38.2\%\) to \(23.8\%\)). The width of the interval was 62%, suggesting an unacceptable agreement. The bias between SjvO₂ and SctO₂ seemed to vary with the level of their means, with SjvO₂ lower than SctO₂ at low values of their means, and higher at high values of their means. A mean (2 SD) bias (\(-9.0[30.0]\%)\) was also lower during the period of decreased MAP after postural change (1–20 min into BCP in 526 measurement pairs), as compared with that (\(-1.3[32.6]\%\) in 164 pairs) after resuming the normotension (25–90 min into BCP)\((P < 0.0001)\).

**Discussion**

The present study demonstrated that S/N group had a higher SjvO₂ than P/R group in BCP. The lowest values were 63 ± 13% versus 42 ± 14% within 10 min, respectively \((P = 0.0006)\). In addition, MAP was higher at 7 to 8 min in BCP in S/N group, despite a less frequent use of vasopressors. S/N

![Fig. 5](http://pubs.asahq.org/anesthesiology/article-pdf/116/5/1047/256940/0000542-201205000-00014.pdf)  
**Fig. 5.** The incidences of hypotension (mean arterial pressure of less than 50 mmHg), jugular venous oxygen desaturation (less than 50%), and cerebral oxygen desaturation (more than 20% decrease from baseline) after moving to the beach chair position in patients under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. *P < 0.05* between groups. MAP = mean arterial pressure; NS = nonsignificant; P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia; SctO₂ = cerebral tissue oxygen saturation; SjvO₂ = jugular venous oxygen saturation.

![Fig. 6](http://pubs.asahq.org/anesthesiology/article-pdf/116/5/1047/256940/0000542-201205000-00014.pdf)  
**Fig. 6.** Regional cerebral oxygenation using near-infrared spectroscopy plotted against jugular venous oxygen saturation from 726 pairs of 36 patients undergoing beach chair surgery under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia; SctO₂ = cerebral tissue oxygen saturation; SjvO₂ = jugular venous oxygen saturation.

![Fig. 7](http://pubs.asahq.org/anesthesiology/article-pdf/116/5/1047/256940/0000542-201205000-00014.pdf)  
**Fig. 7.** Bland-Altman plot of the means of the measured jugular venous oxygen saturation and the cerebral tissue oxygen saturation against the difference between the means for all patients. Each circle represents one patient. Mean bias \(-7.7\%\) (solid line) with 95% limits of agreement from \(-38.2\%\) to +23.8% (dotted lines) are shown. SctO₂ = cerebral tissue oxygen saturation; SjvO₂ = jugular venous oxygen saturation.

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anesthesia may be a better choice in patients undergoing surgery in BCP, where hemodynamics and cerebral perfusion may be rapidly deteriorated after the positioning.

SjvO2 is an indirect assessment of global cerebral oxygen use. Normal SjvO2 averages 62% (range, 50–75%) in awake healthy humans. SjvO2 of less than 50% is an indicative of cerebral hypoperfusion, and readings of less than 40% are supposed to be associated with cerebral ischemia. When the metabolic demand exceeds cerebral oxygen supply, such as with low CBF because of systemic hypotension, the brain extracts a great amount of oxygen with resultant low SjvO2. In the current study, the postinduction baseline SjvO2 was higher in S/N than in P/R group, being in agreement with previous observations. In addition, the magnitude of decreases in SjvO2 in BCP was less pronounced (11 ± 10% vs 23 ± 9%, P = 0.0006) and a reduced SjvO2 value (less than 50%) was less frequently observed in S/N than in P/R group. Furthermore, the incidence of SjvO2 less than 40% was null in S/N group as compared with 29% in P/R group (P = 0.0109). These findings suggest that S/N anesthesia may provide a wider margin of safety against impaired cerebral oxygenation under conditions of impaired cerebral perfusion such as in BCP.

The major adverse hemodynamic consequence of BCP is a decrease of venous return, leading to reductions of cardiac output, MAP, and cerebral perfusion pressure, which persists for up to 30 min after the positioning. In conscious suboutput, MAP, and cerebral perfusion pressure, which persists a decrease of venous return, leading to reductions of cardiac such as in BCP.

Paired cerebral oxygenation under conditions of impaired anesthesia may provide a wider margin of safety against ischemia. When the metabolic demand exceeds cerebral oxygen supply, such as with low CBF because of systemic hypotension, the brain extracts a great amount of oxygen with resultant low SjvO2. In the current study, the postinduction baseline SjvO2 was higher in S/N than in P/R group, being in agreement with previous observations. In addition, the magnitude of decreases in SjvO2 in BCP was less pronounced (11 ± 10% vs 23 ± 9%, P = 0.0006) and a reduced SjvO2 value (less than 50%) was less frequently observed in S/N than in P/R group. Furthermore, the incidence of SjvO2 less than 40% was null in S/N group as compared with 29% in P/R group (P = 0.0109). These findings suggest that S/N anesthesia may provide a wider margin of safety against impaired cerebral oxygenation under conditions of impaired cerebral perfusion such as in BCP.

The major adverse hemodynamic consequence of BCP is a decrease of venous return, leading to reductions of cardiac output, MAP, and cerebral perfusion pressure, which persists for up to 30 min after the positioning. In conscious suboutput, MAP, and cerebral perfusion pressure, which persists a decrease of venous return, leading to reductions of cardiac such as in BCP.

Despite the high prevalence (29%) of SjvO2 <40% in P/R group, which may be related to global ischemia, no new major neurologic deficits were observed in the early postoperative period. One may thus argue that there were some methodological problems or the established criteria for “global ischemia” were simply wrong. The severity and du
ration of ischemia are critical determinants of tissue damage, and viability-time thresholds must be exceeded to produce stroke. Jugular desaturation (SjvO₂ less than 50%) has been reported in 20–60% of patients undergoing neurosurgery with propofol-based anesthesia,13–17 suggesting that propofol anesthesia provides marginally adequate cerebral oxygenation without incurring neurologic dysfunction. In addition, the episodes of desaturation (SjvO₂ < 40%) lasted no longer than 30 min in BCP and all were free of cerebral pathology in the current study. Moreover, an association between desaturation (SjvO₂ below 40%) and global ischemia has been noted in patients with acute brain injury,28,29 whereas a threshold value of SjvO₂ indicating impending cerebral ischemia to date has not been defined during clinical anesthesia. It is possible that the short duration of hypotension in BCP may have resulted in a subtle neurocognitive dysfunction and cerebral injury, which cannot be detected easily on routine clinical examinations. In fact, the prevalence of cerebrovascular events was exceedingly rare (0.00382–0.00461%) during shoulder surgery in BCP in a survey of the membership of the American Shoulder and Elbow Surgeons.46 Nevertheless, clinical outcomes and implications for cognitive function of cerebral oxygen imbalance observed in BCP remain to be further determined.

There is no universally accepted critical cut-off value of SctO₂ below which cerebral ischemia may develop. The current study defined a cerebral oxygen desaturation as a decrease in SctO₂ more than 20% from the baseline value. The threshold to identify cerebral ischemia may be influenced by a number of patient-specific (i.e., presence of cerebrovascular disease) or technology-dependent variables. Moreover, the large degree of intersubject and intrasubject (the average difference of SctO₂ readings between the right and the left hemisphere 4.9 ± 4.5 units [median 4.0, range 0–15]) at postinduction baseline in the current study) variability makes the clinical usefulness of their absolute values uncertain. Therefore, it has been recommended to monitor changes from baseline measurements, i.e., trend monitoring. Until recently, INVOS Cerebral/Somatic Oximeter (Somanetics) has been the only clinical device approved by the U.S. Food and Drug Administration. Clinical studies using the INVOS Oximeter have shown that absolute SctO₂ values below 40 and decline of more than 25% from the baseline are associated with neurologic dysfunction and adverse outcomes,47 and a decline more than 25% below the preoperative baseline is associated with a significant increase in the Major Organ Morbidity and Mortality Index.48 A reduction of 15–20% from the baseline or a reduction below 50 has been used as a critical threshold for concern and initiation of interventions.49,50 In contrast, with a more recently approved NIRS monitoring system (i.e., Fore-Sight®, CAS Medical Systems, Branford, CT), which adopts a relatively quantitative NIRS technology, absolute SctO₂ values of less than 55% represent cerebral ischemia.10,22

Phenylephrine and epidural are commonly used to maintain MAP and thus CPP in neurosurgical patients who develop hypotension.51 However, a recent study showed that a bolus administration of phenylephrine reduces cardiac output and brain oxygenation measured by NIRS device under P/R anesthesia, whereas ephedrine maintains both parameters.52 In agreement with these findings, ephedrine, the only vasopressor used in the current study, increased MAP within 2 min after the administration. SjvO₂ and SctO₂ were not affected in either group, with no intergroup differences. The ET_co2 tension that matched the concomitant arterial value before the study (table 2) was kept between 35 and 40 mmHg throughout the study. It has been indeed known that the arterial carbon dioxide tensions closely related to CBF and thus SjvO₂ during anesthesia.14,35

The current study used a TCI device to achieve and maintain stable effect-site concentrations of propofol and remifentanil.24,25 However, it requires complex and expensive infusion devices, including a computer to control the infusion pump. For practical purposes, when using conventional weight-adjusted administration, effect-site TCI concentration of propofol at 2.1–3.0 μg/ml used in the current study can be achieved with infusing a 2.0 mg/kg bolus over 60 s followed by a continuous infusion of 0.09–0.14 mg·kg⁻¹·min⁻¹, and TCI concentration of remifentanil at 1.9–3.2 ng/ml can be achieved by administering a 1 μg/kg bolus over 60 s followed by a continuous infusion of 0.06–0.11 μg·kg⁻¹·min⁻¹.

Our study has several limitations. First, all patients were free of cerebral pathology. It remains unclear how SjvO₂ and SctO₂ would have responded after shifting to BCP in patients with a cerebral pathology. Second, we did not measure CBF or CMRO₂, so that a differentiation between the changes of flow and oxygen consumption was impossible. Third, the SjvO₂ catheter was inserted into the contralateral side of surgery for better handling. However, most patients have dominant right-sided drainage for the jugular vein, although we did not examine the drainage system by angiography in each patient. The lack of catheterization in the dominant drainage system in every patient may have affected the results.

In conclusion, our study suggests that S/N anesthesia should provide a wider margin of safety against impaired cerebral oxygenation and better maintain systemic hemodynamics than P/R anesthesia. It was also shown that cerebral oxygen saturation determined by NIRS may not reflect virtual changes in cerebral oxygenation detected by jugular venous oximetry in patients undergoing shoulder surgery in BCP.

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