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 ($SjvO_2$) and regional cerebral tissue oxygen saturation ($SctO_2$) were determined while patients were placed from the supine to BCP. Whether $SctO_2$ and $SjvO_2$ 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 $SjvO_2$ and $SctO_2$, 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. $SjvO_2$ 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 $SjvO_2$ <50% and mean arterial pressure less than 50 mmHg were lower in S/N group, but $SctO_2$and the incidence of cerebral desaturation (more than 20% decrease from baseline) did not significantly differ between the groups. $SctO_2$ and $SjvO_2$ were only weakly correlated ($\beta = 0.218$, $r^2 = 0.133$). 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 $SjvO_2$ is more preserved with S/N than with P/R anesthesia. $SctO_2$ may not be reliable in detecting a low $SjvO_2$ 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 ($SjvO_2$) and regional cerebral tissue oxygen saturation ($SctO_2$) monitoring in patients changed from the supine to BCP, the margin of safety against impaired cerebral oxygenation was greater and $SjvO_2$ better preserved with sevoflurane-nitrous oxide than with propofol-remifentanil anesthesia. $SctO_2$ may not be reliable in detecting a low $SjvO_2$ 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, opthalmoplegia,[8] and se-

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Address correspondence to Dr. Yoo: Department of Anesthesiology and Pain Medicine, Chonnam National University Medical School, 8 Hak-dong, Gwangju 501-757, South Korea. kyyoo@jnu.ac.kr. Information on purchasing reprints may be found at www.anesthesiology.org or on the masthead page at the beginning of this issue. ANESTHESIOLOGY's articles are made freely accessible to all readers, for personal use only, 6 months from the cover date of the issue.

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vere cerebral desaturation events[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 ($CMRO_2$), 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.[11] In contrast, propofol reduces CBF greater than $CMRO_2$, resulting in a decrease of the $CBF/CMRO_2$ ratio.[12–14] Indeed, cerebral oxygen balance is better maintained by sevoflurane-based than by propofol-based anesthesia when patients are positioned supine.[15–18] In this context, it is also possible that the margin of safety against an impaired cerebral oxygenation would 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.[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 ($SjvO_2$) well reflects global $CBF/CMRO_2$ ratio and is used as an indirect marker of global cerebral oxygen use (*i.e.*, cerebral oxygenation) in a variety of clinical settings.[20] $SjvO_2$ 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 ($SctO_2$). 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).[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 $SctO_2$ may be an alternative to $SjvO_2$ 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).[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)® 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® 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. $SctO_2$ was monitored by NIRS with an INVOS® 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. $SctO_2$ 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 $SctO_2$) and full preoxygenation, anesthesia was induced with propofol (2.0–2.5 mg/kg) and remifentanil (1.0–2.0 μg/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 μg/kg bolus over 60 s) and propofol set at 3.0 μg/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% N_2O 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% N_2O 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 $SjvO_2$ and blood sampling, a central venous oximetry catheter (PreSep™ Oximetry Catheter; Edwards Lifesciences, Irvine, CA) connected to a Vigileo™ 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® Premier

3000; Instrumentation Laboratory, Lexington, KY) immediately after insertion of the catheter.

Approximately 20 min after anesthesia induction, when hemodynamics became stable, 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 65°–75° above the horizontal plane. The infusions of propofol and remifentanil were prepared using 2% propofol (Fresofol® 2%; Fresenius Kabi GmbH, Granz, Austria) and remifentanil hydrochloride (Ultiva®; GlaxoSmithKline Manufacturing; San Polo di Torrile, Italy), respectively. TCI effect-site concentrations for propofol and remifentanil were achieved using a TCI device (Orchestra Base Primea®; Fresenius, Brezins, France) using Marsh[24] and Minto[25] pharmacokinetic and pharmacodynamic models, respectively.

The surgery began approximately 20 min after positioning when hemodynamics had become stable. Patients were mechanically ventilated to maintain the end-tidal carbon dioxide (ET_{CO_2}) tension at 35–40 mmHg. Neuromuscular blockade was carefully controlled by train-of-four monitoring, and additional boluses of rocuronium were administered to maintain one twitch response in response to electrical stimulation of the ulnar nerve during the surgical procedure. Routine monitoring included invasive measurement of systemic blood pressure, heart rate, and rhythm by 5-lead electrocardiography and pulse oximetry. Throughout the surgery, the end-tidal concentrations of carbon dioxide, sevoflurane, and nitrous oxide were measured using a gas analyzer (Capnomac Ultima; Datex-Ohmeda, Helsinki, Finland). Arterial blood gas analysis was determined when a stable hemodynamic was achieved before the positioning and more if necessary.

MAP and heart rate were recorded by an independent investigator before induction of anesthesia. Simultaneously, peripheral arterial oxygen saturation (Sp_{O_2}), $SctO_2$, and BIS values were measured in patients while breathing room air. These variables (MAP, heart rate, BIS, and $SctO_2$) and $SjvO_2$ were recorded before (postinduction baseline values in supine position) and every min after BCP for 15 min, and then every 5 min for 15 min and every 15 min thereafter until the end of the surgery. Baseline $SctO_2$ and $SjvO_2$ values were the mean over 1-min period during a stable interval (MAP within 20% preinduction values, BIS 40–50, ET_{CO_2} 35–40 mmHg, and inspired oxygen fraction of 0.5). The magnitudes of maximum changes in $SjvO_2$ after positioning were determined by calculating the differences between baseline $SjvO_2$ measured just before positioning and the lowest value observed within 10 min after positioning. Jugular bulb oxygen desaturation was defined as a $SjvO_2$ value of less than 50% lasting more than 5 min under conditions of normal catheter light intensity, and cerebral oxygen desaturation was defined as a decline in $SctO_2$ more than 20% from baseline for more than 15 s. The responsible anesthesiologist was blinded to the $SjvO_2$ and $SctO_2$ values. Hypotension was 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 $SjvO_2$ 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, $SctO_2$, $SjvO_2$, 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 $SctO_2$ and $SjvO_2$ were compared using linear regression analysis, and a Bland-Altman plot[26] was used to graphically compare the agreement of two measurements of $SctO_2$ and $SjvO_2$ using SPSS 17.0 for Windows (SPSS Inc., Chicago, IL). We decided that ±5% would be a clinically acceptable difference between the two methods ($SjvO_2$ and $SctO_2$) 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,

Table 1. Demographic and Intraoperative Variables

	S/N Group (n = 19)	P/R Group (n = 17)	*P* Value
Male/Female	5/14	6/11	0.559
Age, yr	65 ± 9	60 ± 7	0.111
Weight, kg	61 ± 11	64 ± 10	0.361
Hemoglobin, g/dl	13 ± 1	14 ± 1	0.273
Underlying diseases			
Hypertension	6	4	0.590
Diabetes	2	4	0.296
Preoperative medication			
β-blockers	5	2	0.271
Calcium-channel blockers	5	4	0.847
Smoking history	1	2	0.481
Ephedrine administered			
Number of patients	7	12	0.043
Total dose per patient, mg	14 ± 4	13 ± 5	0.869
Duration of anesthesia, min	193 ± 63	188 ± 58	0.806
Duration of sitting position, min	160 ± 63	148 ± 52	0.523
Duration of surgery, min	136 ± 59	127 ± 52	0.638
Fluid administered, ml	1,595 ± 565	1,574 ± 489	0.905
Blood loss, ml	158 ± 115	205 ± 197	0.220

Data are mean ± SD or numbers.

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

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

Table 2. Preoperative Hemodynamic and Intraoperative Blood Gas Data

	S/N Group (n = 19)	P/R Group (n = 17)	*P* Value
Mean arterial pressure, mmHg	99 ± 14	97 ± 11	0.653
Heart rate, beats/min	68 ± 6	68 ± 7	0.488
Spo_2, %	97 ± 2	97 ± 2	0.361
$SctO_2$, %	68 ± 6	68 ± 7	0.801
$Paco_2$, mmHg	40 ± 3	39 ± 2	0.301
Pao_2, mmHg	220 ± 38	202 ± 56	0.219

Data are mean ± SD or numbers.

$Paco_2$ = arterial partial pressure of carbon dioxide; Pao_2 = arterial partial pressure of oxygen; P/R = propofol-remifentanil; $SctO_2$ = regional cerebral tissue oxygen saturation; S/N = sevoflurane-nitrous oxide; Spo_2 = peripheral arterial saturation of oxygen.

patients in either group developed gross neurologic or cognitive dysfunction postoperatively.

Hemodynamic data are presented in figure 1A and 1B. MAP before induction of anesthesia did not differ between

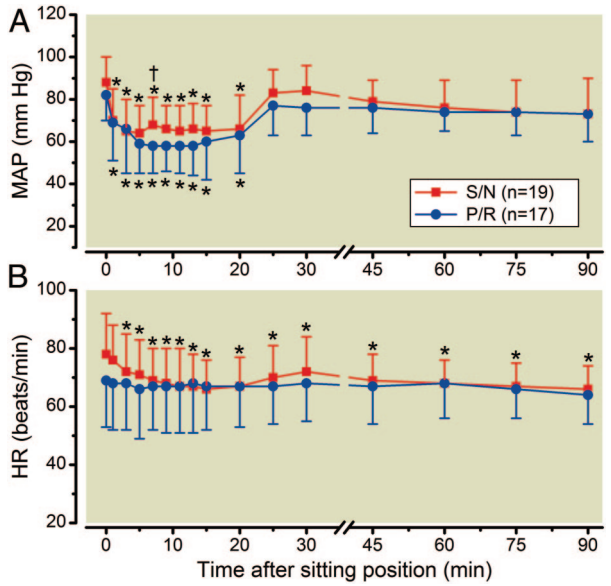

Fig. 1. Mean arterial pressure (*A*) and heart rate (*B*) after moving to the beach chair position in patients under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. Values are means ± SD. Postinduction baseline values in supine position are shown at time 0. *$P < 0.05$ *versus* baseline. †$P < 0.05$ *versus* propofol-remifentanil group (7 to 8 min). Heart rate did not differ between the groups at any time ($P = 0.291$); however, it was lower, although statistically insignificant, in the sevoflurane-nitrous oxide group from 13 min to 20 min into postural change and thus revealed time by group interaction ($P = 0.0017$). 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. HR = heart rate; MAP = mean arterial pressure; P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia.

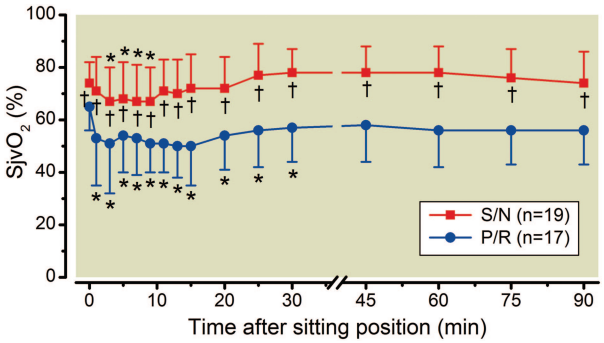

Fig. 2. Jugular venous oxygen saturation after moving to the beach chair position in patients under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. Values are means ± SD *$P < 0.05$ *versus* baseline. †$P < 0.05$ *versus* propofol-remifentanil anesthesia group (all the time). The number 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; $SjvO_2$ = jugular venous oxygen saturation.

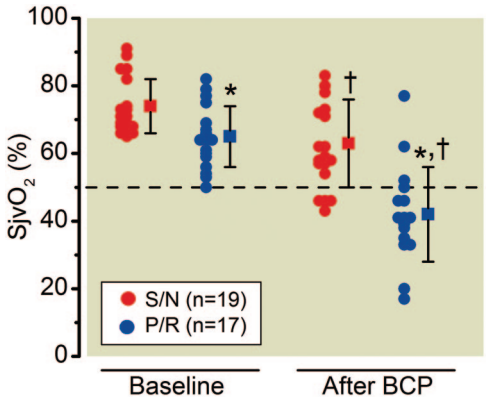

Fig. 3. Individual and mean (±SD) jugular venous oxygen saturation in patients under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. The baseline values obtained in supine position after induction of anesthesia and the lowest values reached within 10 min after the beach chair position are presented. *$P < 0.05$ *versus* propofol-remifentanil anesthesia group. †$P < 0.05$ *versus* baseline. The *horizontal dashed line* represents 50% of jugular venous oxygen saturation, an indirect threshold of cerebral hypoperfusion. BCP = beach chair position; P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia; $SjvO_2$ = jugular venous oxygen saturation.

the groups (table 2). 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 ($P = 0.0017$).

$SjvO_2$ before (postinduction baseline in the supine position) and after BCP are presented in figure 2. Figure 3 shows individual values of $SjvO_2$. 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 $SjvO_2$ of less than 50%. Five patients with $SjvO_2$ of less than 40% were all from the P/R group.

$SctO_2$ 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 $SctO_2$ 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

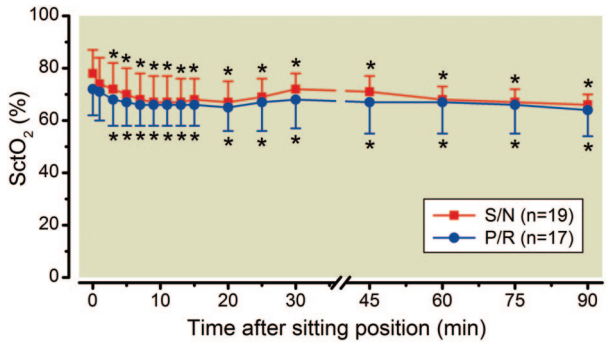

Fig. 4. Regional cerebral tissue oxygen saturation after moving to the beach chair position under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. Values are means ± SD. Postinduction baseline values in supine position are shown at time 0. *$P < 0.05$ *versus* baseline. There was no difference between the groups at any time ($P = 0.291$). The number of patients in the sevoflurane-nitrous oxide anesthesia group decreased from 19 at baseline to 17 at 90 min, whereas the number of patients in the propofol-remifentanil anesthesia group decreased from 17 at baseline to 15 at 90 min. P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia; $SctO_2$ = cerebral tissue oxygen saturation.

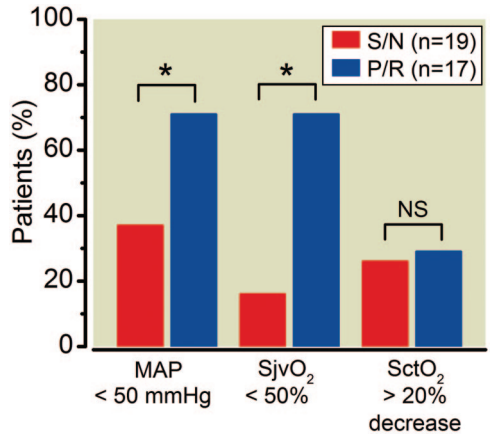

Fig. 5. The incidences of hypotension (mean arterial pressure of less than 50 mmHg), jugular venous oxygen desaturation (less than 50%), and cerebral oxygen desaturation (more than 20% decrease from baseline) after moving to the beach chair position in patients under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. *P < 0.05 between groups. MAP = mean arterial pressure; NS = nonsignificant; P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia; $SctO_2$ = cerebral tissue oxygen saturation; $SjvO_2$ = jugular venous oxygen saturation.

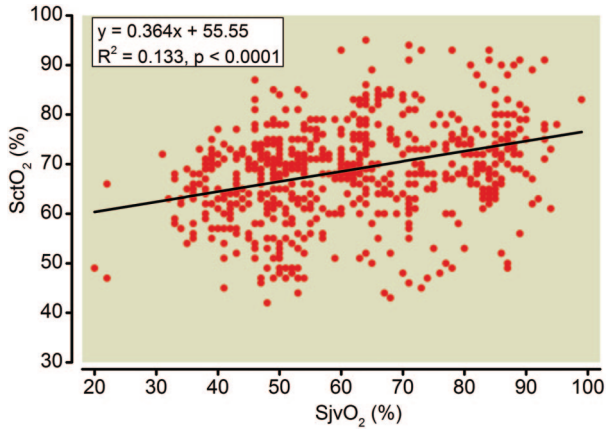

Fig. 6. Regional cerebral oxygenation using near-infrared spectroscopy plotted against jugular venous oxygen saturation from 726 pairs of 36 patients undergoing beach chair surgery under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. P/R = propofol-remifentanil anesthesia; S/N = sevoflurane-nitrous oxide anesthesia; $SctO_2$ = cerebral tissue oxygen saturation; $SjvO_2$ = jugular venous oxygen saturation.

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 $SjvO_2$ 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 $SctO_2$ 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 $SjvO_2$ 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 $SctO_2$ reflects $SjvO_2$, a total of 726 paired measurements of both values from 36 patients were compared using linear regression and Bland-Altman analyses. $SjvO_2$ and $SctO_2$ 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 $SjvO_2$ and $SctO_2$ (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%, 23.8%). The width of the interval was 62%, suggesting an unacceptable agreement. The bias between $SjvO_2$ and $SctO_2$ seemed to vary with the level of their means, with $SjvO_2$ lower than $SctO_2$ 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 $SjvO_2$ 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

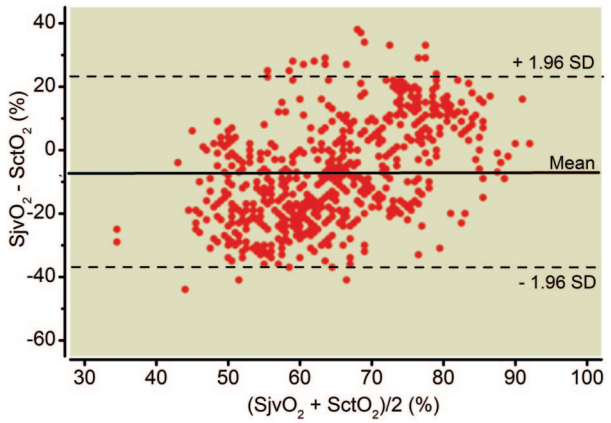

Fig. 7. Bland-Altman plot of the means of the measured jugular venous oxygen saturation and the cerebral tissue oxygen saturation against the difference between the means for all patients. Each *circle* represents one patient. Mean bias −7.7% (*solid line*) with 95% limits of agreement from −38.2% to +23.8% (*dotted lines*) are shown. $SctO_2$ = cerebral tissue oxygen saturation; $SjvO_2$ = jugular venous oxygen saturation.

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

$SjvO_2$ is an indirect assessment of global cerebral oxygen use.[20] Normal $SjvO_2$ averages 62% (range, 50–75%) in awake healthy humans.[27] $SjvO_2$ of less than 50% is an indicative of cerebral hypoperfusion, and readings of less than 40% are supposed to be associated with cerebral ischemia.[28,29] 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 $SjvO_2$. In the current study, the postinduction baseline $SjvO_2$ was higher in S/N than in P/R group, being in agreement with previous observations.[15–18] In addition, the magnitude of decreases in $SjvO_2$ in BCP was less pronounced ($11 \pm 10\%$ *vs.* $23 \pm 9\%$, $P = 0.0006$) and a reduced $SjvO_2$ value (less than 50%) was less frequently observed in S/N than in P/R group. Furthermore, the incidence of $SjvO_2$ 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.[3,4] In conscious subjects, sitting position activates the sympathetic nervous system, resulting in an increase of systemic blood pressure (10–15%) along with increased systemic vascular resistance (30–40%) and reduced cardiac output (15–20%).[30] Under general anesthesia, on the contrary, the baroreceptor response is blunted and MAP decreased in association with a less pronounced increase of systemic vascular resistance and a greater reduction of cardiac output.[3] In the current study, MAP decreased abruptly from the baseline values when patients were switched to BCP in both groups. However, it was higher at 7 to 8 min after the positioning ($P = 0.0212$) and the incidence of hypotension (MAP less than 50 mmHg) was lower in S/N than in P/R group, along with a less frequent use of vasopressors ($P = 0.0429$). MAP is a primary determinant of cerebral perfusion pressure and cerebral oxygenation, and a reduction below 50 mmHg may decrease CBF.[31] It has been known that sevoflurane-based anesthesia inhibits the neuroendocrine stress response less markedly than propofol-based anesthesia.[32] In this context, the higher $SjvO_2$ in S/N group may be at least in part attributed to a greater cerebral oxygen delivery because of a smaller reduction of MAP and hence cerebral perfusion pressure, in which the compensatory sympathoadrenal activity is more preserved.

The preoperative use of renin-angiotensin system blockers has been associated with a greater incidence of hypotension particularly during the first 30 min after anesthetic induction.[33] Moreover, propofol increases hypotensive episodes in a dose-dependent manner during induction of anesthesia in patients taking angiotensin-converting enzyme inhibitors.[34] Indeed, the three patients taking angiotensin II receptor antagonists in P/R group developed moderate to severe hypotension before moving to BCP and were excluded from the data analysis in the current study.

In the current study, Bland-Altman analysis showed a wide limit of agreement between $SjvO_2$ and $SctO_2$ (-38.2% to $+23.8\%$). This finding suggests that $SctO_2$ determined by NIRS does not accurately reflect $SjvO_2$, being consistent with previous observations.[16,35–37] The lack of agreement between the two measurement modalities may be explained by several factors. First, NIRS cerebral monitoring measures a regional area of cortical and subcortical cerebral oxygenation noninvasively, whereas $SjvO_2$ reflects a more global measurement. Any inhomogeneous distribution of blood or metabolic activity would reduce the concordance between the two methods. Second, NIRS assumes a fixed arterial/venous ratio, whereas cerebral oximeter interrogated brain tissue microvasculature is about 70% venous and 30% arterial under most physiologic conditions in humans.[38,39] Sevoflurane has an intrinsic cerebral vasodilator effect that significantly increases regional CBF relative to regional $CMRO_2$,[18,40] leading to increased cerebral arterial volume fraction and ultimately increased arterial/venous ratio.[41] On the contrary, propofol reduces regional CBF and regional $CMRO_2$ in parallel, thus decreasing the cerebral blood volume in the human brain.[18] Changes in body position may also affect the ratio of the compartments through alterations of venous and arterial blood pressures in the cerebral circulation.[42] If the ratio changes, the output of the device may be altered in the absence of real changes in oxygen availability to neuronal mitochondria. Third, NIRS values are contaminated by extracerebral blood flow, hemoglobin concentration, and the layer of cerebrospinal fluid.[43] Moreover, cerebral oximetry values may be affected by depth of anesthesia, the type of anesthetic administered, arterial carbon dioxide-concentrations, inspired oxygen content, and systemic blood pressure management.[44,45] In the current study, the mean (2 SD) bias (-9.0[30.0]%) was lower during the period of decreased MAP after postural change (1–20 min into BCP) as compared with that (-1.3[32.6]%) after resuming the normotension (25–90 min into BCP). This finding is consistent with the lower bias at the low range of their means (fig. 7). Factors that may have led to the lower bias need to be determined. Overall, NIRS, as measured by the machine used in the current study, cannot be routinely recommended in assessing cerebral oxygenation in patients undergoing surgery in BCP.

Despite the high prevalence (29%) of $SjvO_2$ <40% in P/R group, which may be related to global ischemia,[28,29] 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 ($SjvO_2$ 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 ($SjvO_2$ <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 ($SjvO_2$ less than 40%) and global ischemia has been noted in patients with acute brain injury,[28,29] whereas a threshold value of $SjvO_2$ 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 $SctO_2$ below which cerebral ischemia may develop. The current study defined a cerebral oxygen desaturation as a decrease in $SctO_2$ 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 $SctO_2$ 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 $SctO_2$ 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 $SctO_2$ values of less than 55% represent cerebral ischemia.[10,22]

Phenylephrine and ephedrine 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. $SjvO_2$ and $SctO_2$ were not affected in either group, with no intergroup differences. The $ETCO_2$ 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 $SjvO_2$ 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 $mg \cdot kg^{-1} \cdot min^{-1}$, 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 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

Our study has several limitations. First, all patients were free of cerebral pathology. It remains unclear how $SjvO_2$ and $SctO_2$ would have responded after shifting to BCP in patients with a cerebral pathology. Second, we did not measure CBF or $CMRO_2$, so that a differentiation between the changes of flow and oxygen consumption was impossible. Third, the $SjvO_2$ 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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