Haemodynamics and cerebral oxygenation during arthroscopic shoulder surgery in beach chair position under general anaesthesia

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Background: Patients undergoing surgery in beach chair position (BCP) are at risk of cerebral ischaemia. We determined the prevalence and risk factors of jugular venous bulb oxygen desaturation ($SjvO_2 < 50\%$) in BCP. It was also examined whether regional cerebral tissue oxygen saturation ($SctO_2$) measured by near-infrared spectroscopy and $SjvO_2$ are interchangeable for assessment of cerebral oxygenation.

Methods: Fifty-six consecutive patients undergoing arthroscopic shoulder surgery in BCP were studied. Anaesthesia was intravenous with propofol and remifentanil (P/R) or inhalational with sevoflurane and 50% nitrous oxide (S/N) depending on provider choice. Mean arterial pressure (MAP), heart rate (HR), $SjvO_2$, and $SctO_2$ were measured before (baseline; post-induction in supine position) and after the patients assumed BCP. Bland–Altman analysis was performed to measure the agreement between $SctO_2$ and $SjvO_2$.

Results: $SjvO_2$, $SctO_2$, MAP, and HR decreased significantly when patients were raised into BCP. Jugular desaturation occurred in 41% of patients (56% with P/R vs. 21% with S/N

anaesthesia, $P = 0.0077$). Risk factors for the desaturation included P/R anaesthesia [adjusted odds ratio (aOR) 4.76, 95% confidence interval (CI) 1.34–16.95, $P = 0.016$] and MAP < 50 mmHg (aOR 3.85, 95% CI 1.21–12.25, $P = 0.023$). Bland–Altman analysis showed a mean difference of –8.9% with 95% limit of agreement between –40.0% and 23.0%. The percentage error [1.96 standard deviation/mean of the reference method] was 48.5%.

Conclusions: The incidence of jugular desaturation in BCP was 41%, and P/R anaesthesia and hypotension were associated with its occurrence while undergoing surgery under general anaesthesia. $SctO_2$ may not replace $SjvO_2$ for the determination of cerebral oxygenation.

Tʜᴇ beach chair position (BCP) is commonly used in shoulder arthroscopy.[1,2] However, the sitting position is associated with significant reductions in cardiac output, mean arterial pressure (MAP), and cerebral perfusion pressure (CPP), which persists for up to 30 min after the positioning.[3,4] Severe cerebral desaturation events,[5,6] brain and spinal cord ischaemia,[7,8] hemiplegia,[9] transient visual loss, and opthalmoplegia[10] have been indeed documented in patients who had undergone shoulder surgery in the upright position. Although the aetiology of cerebral injury after BCP surgery has not been established, systemic hypotension developed immediately after the positioning has been assumed to compromise cerebral perfusion, resulting in neurological injury when the episodes are prolonged.

Jugular venous oximetry is used to continuously monitor jugular venous bulb oxygen saturation ($SjvO_2$) in a variety of clinical settings.[11] However, this modality is invasive and difficult to use. Moreover, it may miss focal ischaemia because it provides a more hemispheric assessment of oxygenation. As an alternative cerebral monitoring entity, near-infrared spectroscopy (NIRS) assessing regional cerebral tissue oxygen saturation ($SctO_2$) offers a noninvasive and less expensive technique, which is easy and quick to apply. It has been indeed successfully used to assess the adequacy of cerebral perfusion in patients undergoing procedures at high risk

of adverse neurological outcomes (cardiac, vascular, liver transplant, and major abdominal surgery).[12,13] Nevertheless, $SctO_2$ monitored by NIRS may not accurately reflect significant changes in $SjvO_2$ determined by jugular venous bulb oximetry.[14–16]

The present study was aimed to determine the prevalence and risk factors of jugular bulb desaturation ($SjvO_2 < 50\%$) when patients were raised into BCP. It was also examined whether $SctO_2$ reflects $SjvO_2$ for the assessment of the cerebral oxygenation in patients undergoing the surgery in BCP.

Materials and methods

The study protocol was approved by the University Hospital Ethics Committee, and all patients gave informed consent. Fifty-six patients scheduled to undergo elective arthroscopic shoulder surgery under general anaesthesia in BCP were enrolled in the study. Patients were excluded if they had pre-existing cerebrovascular diseases or history of orthostatic hypotension, aged < 18 years, or were American Society of Anaesthesiologists physical status IV or V.

All patients were pre-medicated with midazolam (0.1 mg/kg, orally) 60 min before induction of anaesthesia. Support stockings were placed on the lower extremities. Upon arrival in the operating room, a 20-gauge catheter was inserted into a radial artery to continuously monitor blood pressure and to take blood samples. Ringer's lactate solution was administered at a rate of 10 ml/kg/h throughout the study. Pressure transducer was referenced to the mid-axillary level when patients were in supine and to the external ear canal level when in BCP. A standard bispectral index (BIS) electrode montage (BIS Sensor-Aspect Medical Systems, Natick, MA, USA) was applied to the forehead before induction of anaesthesia. BIS was continuously measured using an Aspect A-2000 BIS® monitor (BIS® XP, software version 3.31, Aspect Medical Systems). $SctO_2$ was determined by NIRS with an INVOS 5100B Cerebral Oximeter (Somanetics Corporation, Troy, MI, USA). The oximeter probes were placed on the right and left foreheads, with the caudal border ~1 cm above the eyebrow with the medial edge at the midline. $SctO_2$ values from the right and left frontal lobes were averaged to represent cerebral oxygenation.

After recording pre-induction values and full pre-oxygenation, anaesthesia was induced with propofol (2.0–2.5 mg/kg)/remifentanil (1.0–2.0 µg/kg) in patients receiving sevoflurane/nitrous oxide (S/N) anaesthesia, or with an effect-site target controlled infusion (TCI) of propofol (3.0 µg/ml)/remifentanil (3.0 ng/ml) in those receiving intravenous (i.v.) propofol/remifentanil (P/R) anaesthesia. Anaesthetic management was not randomized and depended entirely on provider choice. However, the providers for these cases fall neatly into two camps: those who always use inhalational agents (S/N) for maintenance and those who always use i.v. anaesthesia (P/R). After administration of rocuronium (0.8 mg/kg, i.v.), the trachea was intubated, and the lungs were mechanically ventilated with 50% nitrous oxide in oxygen in patients given S/N, and with air and oxygen mixture (50% oxygen) in those given P/R. Sevoflurane concentrations combined with 50% nitrous oxide in oxygen were then adjusted to maintain MAP within 20% of pre-induction values and BIS values between 40 and 50 throughout the surgery in patients given volatile anaesthetics. TCI effect-site concentrations of propofol were also adjusted to achieve BIS readings of 40–50, and those of remifentanil were adjusted to maintain MAP within 20% of the pre-induction value in patients given i.v. anaesthetics. For measurement of $SjvO_2$ and blood sampling, a PreSep™ catheter (Edwards Lifesciences, Irvine, CA, USA) 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.

Approximately 20 min after anaesthesia induction when haemodynamics 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. TCI effect-site concentrations for P/R were achieved using the Orchestra Base Primea® infusion pump (Fresenius, Brezins, France).

The surgery was started approximately 20 min after the positioning when vital signs became stable. Patients were mechanically ventilated to maintain the end-tidal carbon dioxide (CO_2) tension between 4.7 and 5.5 kPa. Routine monitoring included invasive measurement of systemic blood pressure, heart rate (HR) and rhythm by 5-lead electrocardiogram, and pulse oximetry. The end-tidal concentrations of CO_2 and sevoflurane were measured using a gas analyser (Capnomac Ultima, Datex-Ohmeda; Helsinki, Finland). Arterial blood gas analysis was determined to match the end-tidal CO_2 tension when a stable haemodynamic was achieved before the positioning and more if necessary.

MAP and HR were recorded by an independent investigator before induction of anaesthesia. Simul-

taneously, peripheral arterial oxygen saturation, $SctO_2$, and BIS values were measured in patients while breathing room air. These variables (MAP, HR, BIS, and $SctO_2$) and $SjvO_2$ were recorded before (baseline values; post-induction, supine position) and every minute after BCP for 15 min, and then every 5 min for 15 min. In addition, minimum values of $SctO_2$ and $SjvO_2$ were recorded within 15 min after BCP with MAP at their corresponding time points. Baseline $SctO_2$ and $SjvO_2$ values were the means over a 1-min period just before the positioning. Jugular venous bulb desaturation was defined as $SjvO_2$ value less than 50% lasting > 5 min,[11] and cerebral desaturation was defined as > 20% decrease of $SctO_2$ from the baseline value for > 15 s.[17,18] Hypotension was defined as MAP measured at the level of the external auditory canal of < 50 mmHg and was treated with a bolus of ephedrine (8 mg) or phenylephrine (100 μg), and fluid loading (100~200 ml crystalloid solution). Bradycardia, defined as HR < 50 beats/min, was treated with i.v. boluses of atropine 0.5 mg as required. Vasopressor treatment was repeated every 2 min if hypotension persisted or recurred. The incidences of cerebral and jugular venous desaturation and hypotension were recorded.

At the completion of surgery, the anaesthetic was discontinued. Residual neuromuscular block was antagonized with neostigmine and glycopyrrolate. Estimated blood loss and amount of fluid or blood administered during the surgery were recorded. The post-operative visit was made by the responsible surgeon at the evening of surgery, and the patient was assessed neurologically with a gross motor and sensory neurological evaluation and a gross cognitive evaluation (orientation in time and space, recall of name, date of birth, and address). Any side effects were recorded. Management of anaesthesia and haemodynamics were left completely to the discretion of the anaesthesiologist responsible, who was blinded to the $SjvO_2$ and $SctO_2$ values. Data were assessed by a person not involved in anaesthetic care.

Statistical analysis

Sample size calculation was based on the assumption that a relative decrease in $SjvO_2$ of 20% (the smallest effect to be clinically important[11]) would be detected. Based on the preliminary study, a mean and standard deviation (SD) of 69% and 15%, respectively, were chosen. For a power of 0.8 and an α of 0.01, a sample size of 41 patients was calculated to be appropriate to detect a clinically relevant decrease in $SjvO_2$.

Data are expressed as number or mean ± SD. They were analysed using StatView software version 4.0 (Abacus Concepts, Berkeley, CA, USA) on a Macintosh computer. Serial changes in cardiovascular, $SctO_2$, $SjvO_2$, and BIS data were analysed using one-way analysis of variance (ANOVA) with repeated measures. The Scheffé test was used for multiple pairwise comparisons when a significant difference was indicated with ANOVA. Relationship of $SjvO_2 < 50\%$ to anaesthetic methods (S/N vs. P/R), MAP < 50 mmHg, age > 60 years, diabetes mellitus, body mass index, haemoglobin, history of hypertension, or antihypertensive drugs were analysed using multiple logistic regression analysis. A Bland–Altman plot with multiple measurements per subject[19] was used to measure the agreement of two measurements of $SctO_2$ and $SjvO_2$, and percentage errors were calculated using MedCalc Version 12.2.1 (MedCalc Software, Mariakerke, Belgium). A mean percentage error not exceeding 30% was defined to indicate clinical useful reliability of the $SctO_2$. A *P*-value of < 0.05 was considered statistically significant.

Results

Demographical and surgical data are presented in Table 1. Of the 58 patients, one patient was excluded because of unsuccessful cannulation of internal jugular vein and another one because of malfunction of the monitoring system. They aged 60 years (ranging 18–77) and underwent shoulder arthroscopic surgery. Fifteen patients had the fibre-optic catheter placed in the right side and remaining 41 patients in the left side. BIS data remained constant (45.1 ± 3.0) throughout the intraoperative period. Average duration of surgery was 134 min (ranging 40–285). None of them developed gross neurological or cognitive dysfunction post-operatively.

Haemodynamic data are presented in Table 2 and Fig. 1. MAP decreased after induction of anaesthesia and decreased further from 1 min to 20 min after the positioning (one-way ANOVA, $P < 0.0001$). Thirty-one patients (55%) developed hypotension (69% vs. 38% in patients given P/R and S/N, respectively, $P = 0.0199$), and received ephedrine ($n = 22$, 14 ± 7 mg) or phenylephrine ($n = 9$, 160 ± 100 μg). The duration of the hypotensive episode ranged 1–15 min. HR decreased from 3 min to 20 min into BCP ($P < 0.0001$).

$SjvO_2$ and $SctO_2$ values are presented in Fig. 2. $SjvO_2$ values significantly decreased below baseline (70 ± 12%) from 1 min to 20 min into BCP

Table 1

Demographical and intraoperative variables in patients undergoing surgery in the beach chair position under general anaesthesia.

	All patients (n = 56)
Male/female	21/35
Age (years)	60 ± 10
Weight (kg)	63 ± 11
Haemoglobin (g/dl)	14 ± 1
Underlying diseases, n (%)	
Hypertension	20 (36%)
Diabetes mellitus	11 (20%)
Preoperative medication, n (%)	
β-blockers	5 (9%)
Calcium-channel blockers	11 (20%)
ACEI or angiotensin II antagonist	12 (21%)
Smoking history, n (%)	2 (4%)
Methods of anaesthesia, n (%)	
Sevoflurane/nitrous oxide	24 (43%)
Propofol/remifentanil	32 (57%)
Vasopressor administered, n (%)	
Ephedrine	22 (39%)
Total ephedrine dose per patient (mg)	14 ± 7
Phenylephrine	9 (16%)
Total phenylephrine dose per patient (μg)	160 ± 100
Duration of anesthesia (min)	191 ± 59
Duration of surgery (min)	134 ± 56
Fluid administered (ml)	1577 ± 514
Blood loss (ml)	170 ± 162

Data are mean ± standard deviation or numbers (%). ACEI, angiotensin converting enzyme inhibitor.

Table 2

Preoperative haemodynamic and intraoperative blood gas data in patients undergoing surgery in the beach chair position under general anaesthesia.

	All patients (n = 56)
Mean arterial pressure (mmHg)	102 ± 15
Heart rate (beats/min)	69 ± 16
SpO_2 (%)	99 ± 0
$SctO_2$ (%)	69 ± 7
$PaCO_2$ (mmHg)	39 ± 3
PaO_2 (mmHg)	212 ± 50

Data are mean ± standard deviation or numbers. SpO_2, peripheral arterial saturation of oxygen; $SctO_2$, regional cerebral tissue oxygen saturation; $PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen.
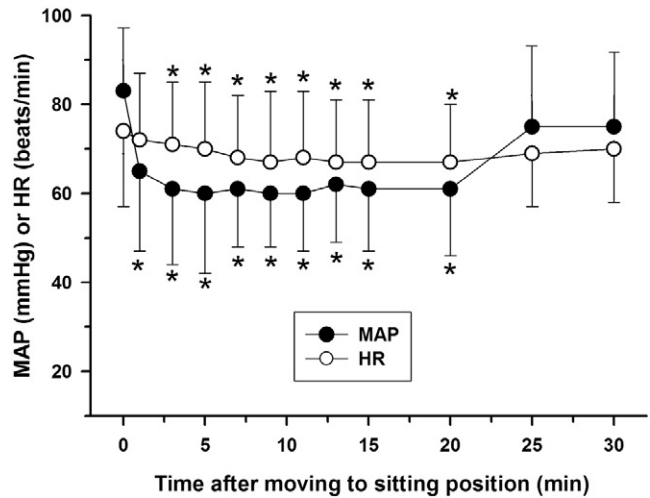

Fig. 1. *Mean arterial pressure (MAP) and heart rate (HR) before (time 0; baseline, post-induction in supine position) and after the beach chair position in patients (n = 56) undergoing surgery under general anaesthesia. Values are means ± standard deviation. *P < 0.05 vs. baseline.*
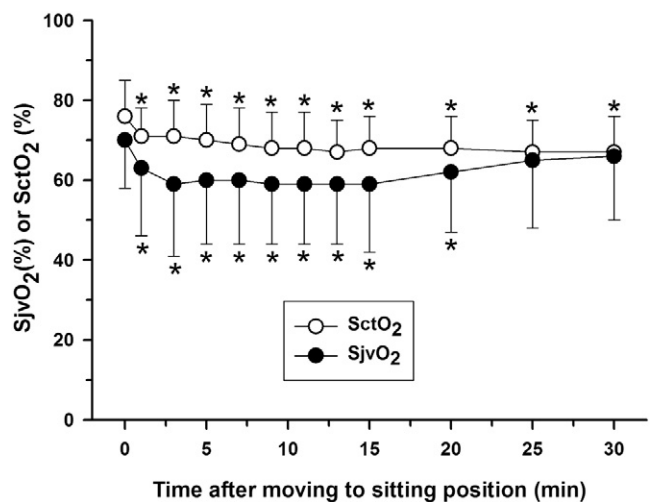

Fig. 2. *Jugular venous bulb oxygen saturation (SjvO₂) and regional cerebral tissue oxygen saturation (SctO₂) before (time 0; baseline, post-induction in supine position) and after the beach chair position in patients (n = 56) undergoing surgery under general anaesthesia. Values are means ± standard deviation. *P < 0.05 vs. baseline.*

($P < 0.0001$). However, the magnitude of decreases of $SjvO_2$ ($22 ± 12\%$ vs. $14 ± 12\%$, $P = 0.0103$) was more pronounced in patients given P/R along with lower pre-sitting baseline values ($65 ± 8\%$ vs. $76 ± 10\%$, $P = 0.0001$) than in those given S/N. On the other hand, $SctO_2$ values increased after induction of anaesthesia from $69 ± 7\%$ (pre-induction) to $76 ± 9\%$ (post-induction baseline, $P < 0.0001$). $SctO_2$ decreased significantly from 2 min after BCP throughout the study ($P < 0.0001$). Of 56 patients, 23 (41%) developed episodes of $SjvO_2 < 50\%$ (56% vs. 21% in patients given P/R and S/N respectively, $P = 0.0077$) and nine patients (16%) had a $SjvO_2 < 40\%$ (25% vs. 4%, $P = 0.0357$).

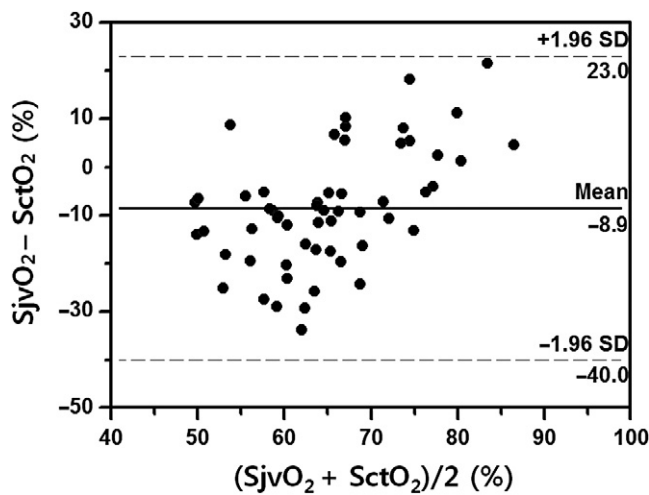Risk factors for jugular desaturation included P/R anaesthesia [adjusted odds ratio (aOR) 4.76, 95%

Fig. 3. Bland–Altman plot with multiple measurements per subject of the means of the measurements of the jugular venous oxygen saturation (SjvO2) and the cerebral tissue oxygen saturation (SctO2) against the difference between the means of all patients. Mean bias –8.9% (solid line) with the 95% limits of agreement from –40.0 to 23.0% (dotted line) is shown. SD, standard deviation.

confidence interval (CI) 1.34–16.95, $P = 0.016$] and MAP < 50 mmHg (aOR 3.85, 95% CI 1.21–12.25; $P = 0.023$). In four patients (7%), jugular desaturation episode happened concomitantly with both hypotension (MAP < 50 mmHg) and > 20% decrease of $SctO_2$ from the baseline. In another 13 patients (23%), the episode happened concomitantly with a hypotensive episode (MAP of < 50 mmHg). The incidence of cerebral desaturation (> 20% decrease of $SctO_2$ from the baseline) was 27% (28% vs. 25%, patients given P/R and S/N, respectively; $P = 0.7938$). The duration of the episodes ranged from 1 min to longer than 30 min. The sensitivity and specificity of MAP < 50 mmHg to detect $SjvO_2$ < 50% were 73.9% and 57.6% (area under the curve = 0.657, $P = 0.047$), whereas those of cerebral desaturation to detect $SjvO_2$ < 50% were 30.4% and 75.8% (area under the curve = 0.531, $P = 0.696$), respectively.

The Bland–Altman analysis showed a mean difference (bias) between the two measurements of –8.9% and 95% limit of agreement (1.96 SD) of –40.0% to 23.0% (Fig. 3). The percentage error (1.96 SD/mean of reference method) was 48.5%.

Discussion

The present study demonstrated that the jugular venous desaturation ($SjvO_2$ < 50%), indicative of cerebral hypoperfusion,[11] occurred in 41% of patients undergoing shoulder surgery in BCP under general anaesthesia. The desaturation was related to hypotension and P/R anaesthesia. The agreement between $SctO_2$ monitored by NIRS and $SjvO_2$ detected by jugular venous oximetry was unacceptable.

The major adverse haemodynamic consequence after raising patients from the supine to sitting position is a decrease in venous return, leading to significant reductions of cardiac output, MAP, and CPP.[3,4] A sitting positioning activates the sympathetic nervous system and hence baroreceptor reflexes,[20] but during anaesthesia, the response may be attenuated, resulting in a greater decrease in MAP.[21] It has been reported that propofol-based anaesthesia results in lower levels of epinephrine, norepinephrine, and cortisol in response to surgical stimuli than does sevoflurane-based anaesthesia,[22] implying that compensatory sympathoadrenal activity is less preserved with the former. Indeed, 55% of patients developed hypotension (MAP < 50 mmHg) in BCP with higher frequency in patients given P/R in the present study. Moreover, the hypotensive episodes was causally related to jugular desaturation ($P = 0.023$). This finding is in line with that in a recent study that showed a high prevalence of significant cerebral oxygen desaturation as determined by NIRS related to decreases of MAP in patients undergoing surgery in BCP.[23] Cerebral blood flow (CBF) is tightly controlled via the cerebral autoregulation within a range of MAP from 50 to 150 mmHg, and a reduction of MAP below the lower limit of autoregulation is associated with comparable decreases in CBF.[24] It is likely that a sitting positioning, especially under P/R anaesthesia, reduces MAP and thus CPP and CBF, leading to jugular desaturation. In fact, a reduction in $SjvO_2$ represents a reduction in CBF and global cerebral oxygenation in the absence of a change in arterial O_2 content, haemoglobin concentration, and cerebral metabolic rate of oxygen ($CMRO_2$).

P/R anaesthesia was associated with jugular desaturation in BCP ($P = 0.016$). Sevoflurane increases CBF in excess relative to the cerebral oxygen demand.[25,26] In contrast, propofol decreases CBF greater than $CMRO_2$,[25,26] implying that the margin of safety against impaired cerebral oxygenation is small. Indeed, patients given P/R had significantly lower pre-sitting baseline values of $SjvO_2$, being consistent with previous observations.[25,26] They also showed a more pronounced fall of $SjvO_2$ in BCP than in those given S/N. It is likely that the P/R anaesthesia reduces jugular saturation because

of a decreased cerebral oxygen delivery in the wake of an unfavourable cerebral oxygen balance and a greater reduction of MAP and CPP. S/N rather than P/R anaesthesia may be a better choice in BCP, where haemodynamics and cerebral perfusion may be rapidly deteriorated.

Bland–Altman analysis showed a poor agreement between the $SjvO_2$ and $SctO_2$ in measuring cerebral oxygenation (Fig. 3). In addition, the sensitivity of cerebral desaturation to detect a $SjvO_2 < 50\%$ was low (30.4%, $P = 0.696$). The lack of agreement may be explained by several factors. First, because NIRS technology does not distinguish between arterial and venous haemoglobin saturations, changes in the proportion of cerebral arterial and venous blood volume may confound measurements.[27] Sevoflurane has intrinsic cerebral vasodilator effect,[28,29] leading to an increased arterial blood proportion and oxygen state.[30] On the contrary, propofol has a cerebral vasoconstrictor effect *in vivo*,[31] thus decreasing the cerebral blood volume. Moreover, changes in body position may also affect the ratio of arterial to venous compartments through alterations of venous and arterial blood pressures in the cerebral circulation.[27] If the ratio changes, the output of the device may be altered without real changes in oxygen availability, resulting in a discordance between the two modalities. Second, cerebral oximetry evaluates only a part of the region of the anterior cerebral artery distribution (cortical tissue of the frontal lobes), whereas $SjvO_2$ reflects a more global oxygen balance as determined by venous blood from the grey as well as the white matter. Thus, any inhomogenous distribution of blood or metabolic activity will reduce the agreement. Third, the brain may have the ability to extract more oxygen from the blood despite a decrease in CBF resulting in a decreased $SjvO_2$, but not necessarily a reduced $SctO_2$. Finally, NIRS values are contaminated by extracerebral blood flow, haemoglobin concentration, and the layer of cerebrospinal fluid.[32] Moreover, cerebral oximetry values may be affected by arterial CO_2 concentrations, inspired oxygen content, and systemic blood pressure management.[33,34]

Despite the frequent occurrence of $SjvO_2$ of less than 40% (16%), which may be associated with global ischaemia,[35,36] no new major neurological deficits were observed in the early post-operative period in the present study. An association between desaturation ($SjvO_2 < 40\%$) and global ischaemia has been noted in patients with acute brain injury.[35,36] In addition, the duration of low MAP was relatively brief (i.e. 1–15 min) in the present study. It is likely that the short duration of hypotension may have resulted in subtle neurocognitive dysfunction and cerebral injury, which cannot be detected easily on routine clinical examination. In fact, the prevalence of cerebrovascular events was exceedingly rare (0.00382–00461%) during shoulder surgery in BCP in a survey of the membership of the American Shoulder and Elbow Surgeons.[37] Nevertheless, clinical outcomes and implications for cognitive function of cerebral oxygen imbalance observed in BCP still need to be determined.

When the sitting position is used, 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) because of the hydrostatic gradient within a vertical column of blood.[38] Instead of arithmetic correction of MAP, in the present study, the blood pressure was monitored at the level of the external ear canal during surgery in BCP, and MAP of 50 mmHg was chosen as a threshold for intervention on the assumption of a normal range of autoregulation in all patients. However, the lower limits of autoregulation may be much higher, particularly in elderly patients with chronic hypertension, atherosclerosis, diabetes mellitus, or a cerebral pathology.[38] Moreover, 41% of patients developed jugular desaturation with our blood pressure management protocol in the present study. Therefore, we would like to emphasize that when BCP is adopted for surgery, it is highly recommended to raise a threshold of MAP for intervention to above 50 mmHg with active fluid management. On the other hand, arterial CO_2 is closely related to CBF and thus $SjvO_2$ values during anaesthesia.[15] Therefore, the end-tidal CO_2 tension that matched the concomitant arterial value before the study was kept between 4.7 and 5.5 kPa throughout the study.

One limitation of our study is that 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 demonstrates that the incidence of jugular desaturation is 41%. Hypotension and P/R anaesthesia increase the risk of its development in patients undergoing shoulder surgery in BCP. It is also shown that NIRS cerebral oximetry does not reflect significant changes in

cerebral oxygenation measured by jugular venous oximetry. NIRS cannot be possibly used as a standard monitoring technique to prevent perioperative cerebral ischaemia from BCP.

Acknowledgements

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