Impact of the Beach Chair Position on Cerebral Perfusion: What Do We Know So Far?

by Torin Shear, MD, and Glenn Murphy, MD

Arthroscopic repair of the shoulder is one of the most commonly performed surgical procedures in the United States. Although shoulder surgery can be conducted in the lateral decubitus position, the majority of surgeons in the United States use the sitting or beach chair position (BCP).[1] In 2005, Pohl and Cullen published a 4-patient case series describing catastrophic cerebral ischemia in patients undergoing shoulder surgery in the BCP.[2] This series has prompted investigators to study how intraoperative management factors (blood pressure, type of anesthesia) may potentially affect outcomes following BCP shoulder surgery. This review presents the current state of science regarding cerebral perfusion in the BCP.

A number of investigations have used near infrared spectroscopy (NIRS) or cerebral oximetry to examine the effect of the BCP on oxygen supply to the brain. Near-infrared spectroscopy is a non-invasive technology that provides continuous monitoring of regional cerebral oxygen saturation (rSO_2). Several investigations have demonstrated that significant decreases in rSO_2 (cerebral desaturation event (CDE), typically defined as a reduction in rSO_2 of ≥20% from baseline values) are not infrequent during BCP surgery. In 2009, Fischer reported the use of NIRS to measure rSO_2 in a 63-year-old female undergoing shoulder surgery in the BCP. After induction of general anesthesia (GA) and patient positioning, hypotension was observed along with a significant decrease in rSO_2.[3] In 2010, 2 similar cases were reported in which a reduction in mean arterial pressure (MAP) precipitated a decrease in rSO_2.[4] In a direct comparison of the effect of position on rSO_2, Murphy evaluated 124 patients under GA during shoulder arthroscopy. Sixty-one patients were in the BCP and 63 in the lateral decubitus position (LDP). The incidence of CDEs was significantly higher in the BCP (80.3% vs. 0% in LDP). An association between CDE and postoperative nausea and vomiting was also observed.[5] In an evaluation of 20 consecutive patients undergoing shoulder surgery in the BCP, Moerman again found an 80% incidence of CDE associated with the BCP.[6] Tange evaluated 30 patients undergoing shoulder surgery in the BCP under GA. All patients had a normal preoperative rSO_2 and MAP. In contrast to the previous studies, no change in rSO_2 was observed intraoperatively, even during periods of hypotension leading authors to conclude that the BCP did not alter rSO_2.[7] The absence of significant decreases in rSO_2 in this study may have been attributable to the degree of sitting position used (30-60°) or the short period of observation (5 min). Furthermore, while rSO_2 is a simple and easy to use surrogate for cerebral blood flow (CBF), it may underestimate malperfusion events. Jeong compared rSO_2 with jugular venous bulb saturations in 56 patients undergoing general anesthesia with either propofol/remifentanil or nitrous/sevoflurane. This study found that cerebral oximetry had only a 30%

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Publication	Study Design	Outcome Measure	Results
Fischer et al *Pain Pract* 2009	Case report 63 yo – ASA 3	$S(ct)O_2$	↓$S(ct)O_2$ correlated with ↓MAP, ↓$etCO_2$
Dippmann et al *Arthroscopy* 2010	Case report 1) 46 yo-ASA 1 2) 58 yo-ASA 2	$S(ct)O_2$	↓$S(ct)O_2$ correlated with ↓MAP
McCulloch et al *Anaesth Intensive Care* 2010	TCD supine v. 45° beachchair (n = 19)	MCA_v	SBP_{arm} 142 → 96 mm Hg SBP_{eam} 141 → 76 mmHg MAP_{eam} 95 → 50 mmHg (47% ↓) MCA_v 46 → 36 cm/sec (22% ↓)
Lee JH et al *Athroscopy* 2011	$S(ct)O_2$ supine v. beachchair (n = 27)	$S(ct)O_2$	MAP_{eam} 85 → 75 mm Hg $S(ct)O_2$ 74 → 67%
Jeong H et al *Acta Anaesthesiolo Scand* 2012	$S(ct)O_2$ and $SjvO_2$ supine v. 65-70° beachchair w/ propofol / remi (P/R) v. sevo / N_20 (S/N) (n = 56)	$S(ct)O_2$ and $SjvO_2$	MAP_{eam} goal w/in 20% baseline. MAP_{eam} < 20% baseline: P/R grp – 69%; S/N grp – 38% $SjvO_2$ < 50%: P/R grp –56% ; S/N grp – 21% P/R anesth (OR 4.76) MAP_{eam} < 50 mmHg (OR 3.85) (p=0.02) $S(ct)O_2$ > 20% ↓ from baseline: P/R grp – 28%; S/N grp – 25% ($S(ct)O_2$ sensitivity and specificity for detecting $SjvO_2$ < 50% was 30.4% and 75.8%)
Gillespie et al *J Bone Joint Surg Am* 2012	EEG supine v. 60° beachchair (n = 52)	EEG	MAP/ SBP goal < 20% below baseline and SBP > 90 mm Hg. All pts fell below BP goals. 3 pts w/ EEG ischemia

$S(ct)O_2$,cerebral oximetry saturation; MAP, mean arterial pressure; SBP, systolic blood pressure; TCD, transcranial Doppler; MCAv, middle cerebral artery velocity; eam, external auditory meatus; P/R, propofol/remi group; S/N, sevoflurance/N_20 group.

Current Studies Warrant Caution with Hemodyamic Management of Patients in the Beach Chair Position

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sensitivity for detecting a jugular venous saturation <50% which is typically considered a critical value. Although it raises questions regarding the sensitivity of rSO_2, this study clearly demonstrated cerebral malperfusion events in the BCP with 41% of patients suffering a jugular venous bulb desaturation.[8]

It is possible that cerebral perfusion may be better maintained during BCP surgery if regional anesthesia (RA) is used. In order to investigate this issue, Yadeau performed a study using NIRS in patients receiving RA for surgery in the BCP. Ninety-nine patients were monitored continuously using cerebral oximetry. Despite a relatively high incidence of hypotension (77%), cerebral desaturation occurred in only 0.77% of patients.[9] In another observational trial, Murphy evaluated 60 patients undergoing shoulder surgery in the BCP with either GA or RA. The GA group had significantly more CDEs (57.6%) than the RA (0%) group.[1]

In conclusion, investigations using cerebral oximetry have reported that significant decreases in rSO_2 are common in the BCP when GA is used, but are rare under RA. However, in the same studies, no cerebral vascular events (CVE) leading to gross neurologic injury were identified. Further data are needed to determine the clinical significance of these CDEs. It is possible that more subtle neurocognitive injury may occur when CDEs are prolonged.

At the present time, the incidence of stroke or significant cerebral injury remains poorly defined. Friedman attempted to answer this question in a large, retrospective survey study of 93 orthopedic surgeons specializing in shoulder surgery. The overall rate of CVE was reported as 8/274,225 or 0.00291%. All 8 cases were in the BCP. The type of anesthesia was not reported.[10] In a mixed prospective/retrospective case review of 4,169 patients undergoing shoulder surgery in the BCP under RA (95.7%), Yadeau found a similarly low incidence of CVEs, reporting no events despite significant and frequent hypotension.[11] More recently, a large retrospective review evaluated 15,014 patients over an 11-year period. All patients underwent shoulder surgery in the BCP under RA. Only one new neurologic deficit was reported occurring 24-hours after surgery.[12] These large database reviews suggest a very low incidence of CVE in this population, at least when surgery is performed under RA.

Despite the low incidence of catastrophic neurologic events in the BCP, the effect of low blood pressure on CBF in the sitting position remains a concern. Several studies evaluated the effect of controlled hypotension in the BCP on CBF. Caution should be used when interpreting these studies. To date, a standard definition of hypotension does not exist. In addition, the location of blood pressure measurement is important (external auditory meatus (EAM) versus arm; non-invasive blood pressure or arterial line) as blood pressure measured at the arm may not be an accurate reflection of cerebral pressure. Lee prospectively investigated 28 patients under GA and noted that when a MAP of 60-65 mmHg (radial arterial line measured at the EAM) was maintained during surgery, a significant reduction in rSO_2 as measured by NIRS occurred (a surrogate for CBF).[13] In another study of 40 patients randomized to RA or GA, CBF was estimated using Doppler ultrasound of the internal carotid artery. BCP significantly reduced MAP in the GA group as compared to RA. Despite this, no change in CBF occurred when MAP was maintained above 70 mmHg.[14] In a similar study, McCulloch measured middle cerebral artery blood velocity using transcranial Doppler in 19 patients under GA. When hypotension was induced (SBP 142 mm Hg to 96 ± 10 mmHg at the level of the arm), a 22% decrease in middle cerebral artery blood flow velocity was noted.[15] Gillespie used electroencephalography (EEG) to monitor for cerebral ischemia in 52 patients under GA with induced hypotension in the BCP. Ischemic changes on EEG were observed in 3 of 52 patients; cerebral ischemia resolved with an increase in blood pressure. No gross deficits were noted postoperatively as measured by the mini-mental status exam (MMSE). Interestingly, the authors concluded that controlled hypotension may be tolerated safely in this population.[16] This notion is concerning for several reasons. First, a 6% risk of cerebral ischemia should not be accepted as "safe." Second, one cannot presume the "safety" of a lower blood pressure threshold when detected ischemic events prompted a change in management. Third, the sensitivity of the MMSE for the detection of neurocognitive dysfunction is poor; therefore the MMSE may fail to identify subtle post-injury sequelae of cerebral malperfusion.[17]

While these studies do not provide conclusive evidence of the harm of hypotension in the BCP, concern is raised regarding an apparent decrease in cerebral perfusion as measured by multiple modalities (EEG, NIRS, Doppler). This concern is magnified by a high incidence of antihypertensive medication use in the American surgical population. Trentman, in a retrospective chart review of 384 patients in the BCP, identified an increased incidence of hypotensive episodes and vasopressor use in patients taking antihypertensive medications preoperatively.[18] Possible mitigating techniques include the use of RA and or sequential compression devices (SCDs). Kwak evaluated 66 patients undergoing shoulder surgery in the BCP. The incidence of hypotension was slightly higher in patients who were not wearing SCDs.[19]

There is clearly a growing body of literature addressing cerebral perfusion in patients undergoing shoulder surgery in the BCP (see Table 1). Some inferences can be made from the current level of data. First, cerebral malperfusion appears to occur frequently during shoulder surgery in the BCP. The incidence appears to be greatest in patients under GA with relative hypotension. RA may protect cerebral perfusion by better maintaining cerebral autoregulation. The incidence of catastrophic neurologic injury appears to be low, but to date there are no studies evaluating more subtle forms of neurologic injury. Clinicians should be aware that blood pressure measured at the level of the arm likely overestimates cerebral pressure in the BCP. Finally, one should carefully consider a patient's baseline blood pressure when determining an "adequate" pressure to maintain cerebral perfusion. Large clinical studies are underway and should offer more information regarding the risk of shoulder surgery in the BCP and possible clues toward a best practice in managing these patients.[20] However, a "best practice" will be difficult to define until we are able to better understand the definition of baseline blood pressure and to what degree a deviation from baseline is safe. In the interim, clinicians should remain aware of the potential danger of cerebral malperfusion in this patient population.

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