



Cerebral desaturation events in the beach chair position: correlation of noninvasive blood pressure and estimated temporal mean arterial pressure

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Background: Cerebral oximetry (rSO₂) has emerged as an important tool for monitoring of cerebral perfusion during surgery. High rates of cerebral desaturation events (CDEs) have been reported during surgery in the beach chair position. However, correlations have not been made with blood pressure measured at the cerebral level. The purpose of this study was to examine the correlations between brachial noninvasive blood pressure (NIBP) and estimated temporal mean arterial pressure (eTMAP) during CDEs in the beach chair position.

Methods: Fifty-seven patients underwent elective shoulder surgery in the beach chair position. Values for eTMAP, NIBP, and rSO₂ were recorded supine (0°) after induction and when a CDE occurred in the 70° beach chair position. Twenty-six patients experienced 45 CDEs, defined as a 20% drop in rSO₂ from baseline.

Results: Median reduction in NIBP, eTMAP, and rSO₂ from baseline to the CDE were 48.2%, 75.5%, and 33.3%, respectively. At baseline, there was a significant weak negative correlation between rSO₂ and NIBP ($r_s = -0.300$; $P = .045$) and no significant association between rSO₂ and eTMAP ($r_s = -0.202$; $P = .183$). During CDEs, there were no significant correlations between rSO₂ and NIBP ($r_s = -0.240$; $P = .112$) or between rSO₂ and eTMAP ($r_s = -0.190$; $P = .212$). No significant correlation between the decrease in rSO₂ and NIBP ($r_s = 0.064$; $P = .675$) or between rSO₂ and eTMAP ($r_s = 0.121$; $P = .430$) from baseline to CDE was found.

Conclusion: NIBP and eTMAP are unreliable methods for identifying a CDE in the beach chair position. Cerebral oximetry provides additional information to the values obtained from NIBP and eTMAP, and all should be considered independently and collectively.

Level of evidence: Basic Science, Physiology.

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Hypoxic neurologic injuries after inadequate cerebral perfusion during surgery performed in the beach chair position have been reported even in healthy patients.^{1,8,10,20} Such reported injuries include blindness,² neurapraxia of the cranial nerves,⁷ pituitary apoplexy,¹² cardiovascular collapse,²³ and death.⁸ Consequently, reports have cited the beach chair position as an independent risk factor for cerebral ischemia during surgery.^{15,16,24} Position-related hypoxia is concerning as the majority of arthroscopic and open shoulder surgeries are performed in the beach chair position.¹⁰ Accurate estimation of cerebral perfusion is essential in preventing hypoxic cerebral injury. Brachial noninvasive mean arterial blood pressure (NIBP) is often observed to ensure adequate cerebral perfusion as it is used to estimate cerebral perfusion pressure and cerebral autoregulation. Several authors have reported an intraoperative decrease in systolic and mean arterial pressures in the beach chair position,^{8,13,20,25,29} believed to result in ischemic brain injury.²⁰

Cerebral oximetry has recently gained popularity as a noninvasive means of estimating cerebral perfusion during surgery through the measurement of cerebral oxygenation. Much less invasive than a jugular venous catheter,²⁸ this device estimates regional tissue oxygenation by way of transcutaneous measurements with light penetration of the skull, giving real-time estimates of cerebral perfusion.^{5,28} Cerebral oximetry potentially allows early detection of desaturation events and appropriate intervention of presumed decreased cerebral perfusion.²¹ Numerous randomized controlled trials have demonstrated that detection and treatment of cerebral desaturation events (CDEs) result in better clinical outcomes.^{4,11,18,22} Whereas the incidence of intraoperative cerebrovascular events in the beach chair position is <0.1%,¹⁰ CDEs, defined as a 20% or greater drop in cerebral oxygenation,⁹ are much more common, seen in up to 80% of beach chair procedures.^{6,17,19} It is presumed that during a CDE, cerebral perfusion is diminished. However, the correlation between desaturation events and cerebral blood pressure measurements has not been described.

The purpose of this study was to determine if there is a correlation between the changes in brachial NIBP and estimated cerebral mean arterial pressure (estimated temporal mean arterial pressure [eTMAP]) with respect to cerebral oxygen saturation (rSO₂) during a CDE. We hypothesize that no correlation exists between NIBP and eTMAP with respect to rSO₂ during a CDE.

Materials and methods

From July 2012 to July 2013, a cohort of 57 patients who underwent elective shoulder surgery (56 shoulder arthroplasties and 1 humeral fracture fixation) were enrolled in a prospective study using cerebral oximetry and invasive arterial monitoring of blood pressure in the beach chair position. Of the 57 patients, only those who experienced a CDE, defined as a 20% drop in rSO₂ from baseline (after induction, supine), during their operation were included in this analysis; 26 of the 57 patients met this criterion.

Each CDE was reviewed independently; a total of 45 CDEs were recorded. Of these CDEs, 34 were bilateral and 11 were unilateral. In the case of bilateral involvement, the side with the greater drop in rSO₂ from baseline was used for analysis purposes. Patients were included if they were undergoing elective open shoulder surgery and were at least 18 years of age. All minors, pregnant patients, prisoners, and patients with impaired decision-making abilities were excluded. Patients were included in the study after they consented to the use of invasive arterial monitoring during surgery.

The average age of the patients was 71 years (range, 57-85 years). There were 16 men and 10 women. American Society of Anesthesiologists scores of 2 and 3 were assigned to 17 and 9 patients, respectively. All patients underwent a preoperative interscalene block and peripheral arterial catheterization. All patients were induced with propofol and maintained with sevoflurane inhalation agent. Throughout the duration of anesthesia, attempts were made to maintain end-tidal carbon dioxide between 35 and 40 mm Hg.

The INVOS cerebral oximeter (Covidien, Mansfield, MA, USA) was placed on the right and left side of the patient's forehead (frontal region) to measure cerebral oxygen saturation (rSO₂). An arterial transducer was leveled at the temporal artery to represent the arterial pressure at the level of the brain. Estimated mean arterial pressure at the temporal level (eTMAP) was recorded from the reading of the transducer at the level of the temporal artery. Brachial NIBP was obtained from the measurements of an automated sphygmomanometer placed on the upper arm. Baseline values for eTMAP, NIBP, and rSO₂ were recorded in the supine position after the induction of general anesthesia. Values were again recorded in the beach chair position as well as anytime a CDE was determined by cerebral oximetry, defined as a 20% drop in rSO₂ from baseline.⁹

When a CDE occurred, no single variable was used to justify an intervention. Episodes of hypotension were treated with ephedrine or phenylephrine on the basis of heart rate measurements and a fluid bolus if vital signs or blood loss warranted. If end-tidal carbon dioxide was below 35 mm Hg and the patient was normotensive, ventilator settings were adjusted to decrease ventilation and to increase carbon dioxide. If the CDE was unilateral, blood pressure, end-tidal carbon dioxide, head position, and sensor were checked. The sensor or patient position was changed if needed.

Any postoperative neurologic sequelae were reported after the procedure.

Statistical analysis

Data were analyzed with PASW Statistics version 18.0 (SPSS Inc, Chicago, IL, USA). Because the data were not normally distributed, descriptive statistics including medians with interquartile ranges were computed for each of the examined variables. Spearman correlation coefficients were calculated to assess rSO₂ and its relation to NIBP and eTMAP at baseline and during a CDE. For all statistical tests, a *P* value of <.05 was used to determine significance.

Results

Descriptive analysis of the 45 CDEs showed that median reduction in NIBP from baseline to CDE was 48.2%, median reduction in eTMAP from baseline to CDE was 75.5%, and

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