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Systemic and cerebral hemodynamic changes in patients undergoing shoulder arthroscopy in the beach chair position

K. I. Lyzohub, M. V. Lyzohub

Sytenko Institute of Spine and Joint Pathology National Academy of Medical Sciences of Ukraine, Kharkiv

The objective of study was verifying the impact of changing position and general anesthesia on internal carotid artery peak systolic velocities in patients undergoing shoulder arthroscopic surgery. Methods. A total of 85 American Society of Anesthesiologist (ASA) physical status I–II patients of median age (33.0 ± 11.3) y. o. underwent shoulder arthroscopy under general anesthesia. The primary measures were systolic blood pressure, diastolic blood pressure), MAP, and ICA PSV. The main measurements were taken in the next time points. The first one (initial data — 1) in the supine position on a table before induction, the second one — 3 min after induction to anesthesia in supine position and the third one in the beach chair position (BCP). BCP was declared as a trunk elevation to 80° . An induction and maintenance of general anesthesia were performed by propofol and fentanyl. Results. The initial level of the SBP before induction was (143.7 ± 22.1) mm Hg, after induction to general anesthesia SBP ($p = 0.001$) decreased to (125.1 ± 26.7) mm Hg, after positioning changed in the same manner. The initial level of DBP was (87.6 ± 11.2) mm Hg, then it dropped ($p = 0.013$) to (77.6 ± 20.2) mm Hg with statistically significant ($p = 0.005$) decreasing to (66.1 ± 17.7) mm Hg. MAP showed statistically changes from initial level of (113.3 ± 20.0) mm Hg to (101.3 ± 23.1) mm Hg ($p = 0.015$) after induction to anesthesia and to (86.5 ± 21.6) mm Hg after positioning ($p = 0.015$). After positioning to BCP ICAPSV reduced significantly from (81.5 ± 15.1) cm/sec to (69.5 ± 16.8) cm/sec ($p = 0.001$). The overall drop in ICAPSV was (16.9 ± 19.0) cm/sec. Conclusion. SBP, DBP and MAP were decreased after induction to general anesthesia and after subsequent patient positioning to the BCP, however post-induction hypotension does not provoke changes in ICA PSV. Though, further positioning of patients to the BCP leads to decreasing of ICA PSV.

Поєднання таких факторів як загальна анестезія та зміна положення тіла після індукції мають вплив на системну та церебральну гемодинаміку. Мета. Виявити вплив зміни положення тіла та загальної анестезії на пікову систолічну швидкість внутрішньої сонної артерії (ПСШВСА) у пацієнтів під час проведення артроскопії. Методи. 85 пацієнтів із фізичним статусом за ASA I–II, середнього віку ($33,0 \pm 11,3$) років, проведено артроскопію плечового суглоба під загальним наркозом. Основними вимірюваннями були систолічний артеріальний тиск (САТ), діастолічний артеріальний тиск (ДАТ), середній артеріальний тиск та ПСШВСА. Основні вимірювання: в положенні лежачи на столі перед індукцією, через 3 хв після введення в наркоз в положенні лежачи та в положенні пляжного крісла (ППК). Індукцію та забезпечення загальною анестезією проводили сибазоном, пропофолом і фентанілом. Дані проаналізовано статистично. Методи описової статистики використовували для розрахунку середнього, стандартного відхилення, мінімального і максимального значень вибірки. Порівняння проводили за допомогою Т-тесту для повторних порівнянь з розрахунком різниці середніх зі стандартним відхиленням ($M \pm SD$), критичного значення критерію (t) та його статистичної значущості (p). Для порівняння динаміки зміни параметрів було проведено загальний лінійний аналіз з визначенням значення критерію Піллаї та його значення. Аналіз проводився в пакеті IBM SPSS Statistics 20.0. Результати. Початкові дані САТ, ДАТ та середнього артеріального тиску достовірно знижувались після індукції та після позиціонування, але індукція не вплинула на показник ПСШВСА. Після позиціонування до ППК було констатовано значне зниження показників ПСШВСА. Висновки. Констатовано достовірне зниження САТ, ДАТ, середнього артеріального тиску після індукції та подальшого позиціонування хворих у ППК. Постіндукційна гіпотензія не впливає на динаміку ПСШВСА, але виявлено достовірне її зниження після позиціонування. Ключові слова. положення пляжного крісла, артеріальний тиск, пікова систолічна швидкість внутрішньої сонної артерії, артроскопія.

Keywords. Beach chair position, blood pressure, internal carotid artery peak systolic velocities, arthroscopic surgery

Introduction

The beach chair position (BCP) was developed in the 1980s for shoulder arthroscopy procedures. Approximately two-third of shoulder surgeries are conducted in the beach chair position, as opposed to the lateral decubitus position [1]. The advantages of the beach chair position include the ease of set-up, limited brachial plexus stress, increased glenohumeral and subacromial visualization, anesthesia flexibility, and the ability to easily convert to an open procedure [2]. However, there are risks associated with moving an anesthetized patient from the supine to the sitting position, which include reductions in blood pressure, cardiac output (CO), and cerebral perfusion, which if prolonged, may result in serious adverse events such as stroke, coma, spinal cord ischemia, and visual loss [3]. Salazar et al. reported cerebral desaturation in 18 % of patients during shoulder surgery in the BCP [4]. Hemiparesis was reported in a patient with a congenital asymmetry of the circle of Willis, which may have resulted in limited collateral flow to the left anterior and middle cerebral arteries (incomplete circle of Willis may be present in up to 40 % of patients) [5]. Another report noted a case of a patient who did not regain consciousness after an orthopedic BCP procedure despite the maintenance of appropriate blood pressure [6]. When patients are positioned in the BCP under anesthesia, significant hemodynamic changes, such as decreases in mean arterial pressure (MAP), heart rate, and CO may develop. The circulatory challenge of being positioned in a sitting position during induction of anesthesia may jeopardize maintenance of MAP leading to hypotensive events. We questioned whether hemodynamic challenge of anesthesia in the sitting beach-chair position compromises cerebral perfusion in patients, undergoing shoulder arthroscopic surgery.

The *purpose* of the study was to evaluate the impact of changing position and general anesthesia on internal carotid artery peak systolic velocities (ICA PSV) in patients undergoing shoulder arthroscopic surgery.

Material and methods

A case series was accomplished in SI “Sytenko Institute of Spine and Joint Pathology National Academy of Medical Sciences of Ukraine”. A total of 85 American Society of Anesthesiologist (ASA) physical status I–II patients of median age

(33.0 ± 11.3) y. o. underwent shoulder arthroscopy under general anesthesia. Inclusion criteria included patients with a torn or damaged cartilage ring or ligaments, shoulder instability, in which the shoulder joint is loose and slides around or becomes dislocated. Exclusion criteria included patients who had previously documented stroke, brain injury, heart failure or arterial hypertension. The primary measures were systolic blood pressure (SBP), diastolic blood pressure (DBP), MAP, and ICA PSV. Hemodynamic parameters were measured by patient monitor Mediana YM 6000. Ultrasound examinations of ICA PSV was performed using ultrasound device Toshiba Aplio-500 with a linear transducer with a frequency of 7,5 MHz by standard method by one examiner. The main measurements were taken in the next time points. The first one (initial data — I) in the supine position on a table before induction, the second one — 3 min after induction to anesthesia in supine position and the third one in the beach chair position (after positioning). BCP was declared as a trunk elevation to 80°. The anesthesia was induced by fentanyl, diazepam, and propofol in standard doses by manual bolus. Maintenance of general anesthesia was performed by propofol and fentanyl infusion to adjust BIS parameters at level 40–60. If the hemodynamic conditions were judged as stable the patients were tilted up, and remained in that position throughout the surgical procedure.

The data were analyzed statistically. Descriptive statistic methods were used to calculate the mean (M), standard deviation (SD), minimum (min) and maximum (max) sample values. Comparisons were made using the T-test for repeated comparisons with the calculation of the difference in means with standard deviation (M ± SD), the critical value of the criterion (t) and its statistical significance (p). The calculated power of the test with the received calculation data is 0.99 (R package). To compare the dynamics of parameter changes, a general linear analysis was conducted, with the determination of the value of the Pillai criterion and its value. The analysis was carried out in the IBM SPSS Statistics 20.0 package.

The trial was conducted in accordance with the ethical principles as well as all patients provided written informed consent before study initiation.

Results

We analyzed the hemodynamic effects of induction to anesthesia in the supine position with

subsequent stepwise rise to the sitting BCP (Table). The initial level of the SBP before induction was (143.7 ± 22.1) mm Hg, after induction to general anesthesia SBP significantly ($p = 0.001$) decreased to (125.1 ± 26.7) mm Hg, after positioning SBP further declined significantly to (106.8 ± 27.0) mm Hg ($p = 0.028$). DBP changed in the same manner. The initial level of DBP was (87.6 ± 11.2) mm Hg, then it dropped significantly ($p = 0.013$) to (77.6 ± 20.2) mm Hg with subsequent statistically significant ($p = 0.005$) decreasing to (66.1 ± 17.7) mm Hg. MAP as the main index of tissue perfusion also showed statistically significant changes from initial level of (113.3 ± 20.0) mm Hg to (101.3 ± 23.1) mm Hg ($p = 0.015$) after induction to anesthesia and to (86.5 ± 21.6) mm Hg after positioning ($p = 0.015$). None of the patients required vasopressor support in perioperative period.

Interestingly, dynamics of ICA PSV was different (Table). Despite significant changes in systemic hemodynamics after induction to anesthesia (decreasing of SBP, DBP and MAP) ICA PSV did not show statistically significant changes. After positioning to BCP ICAPSV reduced significantly from 81.5 ± 15.1 cm/sec

to 69.5 ± 16.8 cm/sec ($p = 0.001$). The overall drop in ICAPSV was (16.9 ± 19.0) cm/sec.

Discussion

Cerebral blood flow is dependent on systemic circulation that can be altered by both general anesthesia and position changing. The brain accounts only for 2 % of body weight, yet it receives about 15–20 % of cardiac output. Most of the arterial blood supply (85 %) is derived from internal carotid arteries [7]. Cerebral autoregulation is the essential local regulatory mechanism that keeps CBF relatively constant despite large changes in systemic arterial pressure. The present clinical study aims to examine whether the combination of factors such as a general anesthesia and changing position may have an impact on the changing of flow velocity in carotid artery.

Internal carotid artery blood flow may reflect both systemic and cerebral circulation. The mean ICA PSV depends on the age of the patient ranging from 82 (50–112) cm/sec at age 20–39, 76 (60–101) cm/sec at age 40–49, 80 (59–96) cm/sec at age 50–59, 79 (62–109) cm/sec at age 60–69 to 71 (40–97) cm/sec at age 70–79. The most important fact is that systolic and diastolic middle cerebral artery flow velocities

Table

Systemic and cerebral hemodynamic changes in patients at different time points

Parameters		Initial data (I)	After induction to anaesthesia (II)	After positioning (III)	
Systolic blood pressure, mm Hg	M \pm SD min \div max	143.7 \pm 22.1 106.0 \div 202.0	125.1 \pm 26.7 67.0 \div 179.0	106.8 \pm 27.0 70.0 \div 195.0	
	(M \pm SD), t, p	I–II II–III	—	19.6 \pm 26.6 t = 3.678; p = 0.001	15.3 \pm 28.0 t = 2.386; p = 0.028
		I–III	37.3 \pm 31.7 t = 5.516; p = 0.001		
Diastolic blood pressure, mm Hg	M \pm SD min \div max	87.6 \pm 11.2 63.0 \div 120.0	77.6 \pm 20.2 30.0 \div 130.0	66.1 \pm 17.7 40.0 \div 93.0	
	(M \pm SD), t, p	I–II II–III	—	11.2 \pm 21.4 t = 2.662; p = 0.013	9.9 \pm 13.5 t = 3.193; p = 0.005
		I–III	21.4 \pm 19.4 t = 5.191; p = 0.001		
Mean arterial pressure, mm Hg	M \pm SD min \div max	113.3 \pm 20.0 50.0 \div 161.0	101.3 \pm 23.1 48.5 \div 150.0	86.5 \pm 21.6 55.0 \div 144.0	
	(M \pm SD), t, p	I–II II–III	—	12.9 \pm 24.7 t = 2.614; p = 0.015	12.5 \pm 19.5 t = 2.717; p = 0.015
		I–III	29.4 \pm 24.5 t = 5.490; p = 0.001		
Internal carotid artery peak systolic velocities, cm/sec	M \pm SD min \div max	84.9 \pm 16.7 61.0 \div 120.0	81.5 \pm 15.1 60.0 \div 107.0	69.5 \pm 16.8 45.0 \div 117.0	
	(M \pm SD), t, p	I–II II–III	—	3.2 \pm 15.4 t = 0.962; p = 0.348	15.4 \pm 12.8 t = 5.231; p = 0.001
		I–III	16.9 \pm 19.0 t = 4.093; p = 0.001		

are significantly correlated with internal carotid artery flow velocities [8]. Thus, ICA PSV with some limitations may represent intracranial hemodynamics. Arkadiusz Siennicki-Lantz et al. have shown that ICA PSV correlates with cerebral blood flow measured by SPECT in patients without hypertension [9]. That is why patients with arterial hypertension were excluded from investigation.

After induction to anesthesia, patients are at particular risk of hypotension mainly due to preexisting hypovolemia, the cardiovascular depressant and vasodilatory effects of induction agents, and a lack of surgical stimulation. Blood pressure during anesthesia induction with propofol can be decreased by venous dilatation, arterial dilatation, or a reduction in cardiac contractility. The recent investigation emphasized domination of arterial dilatation effect of propofol on the occurrence of post-induction hypotension [10]. Certain risk factors, such as age, sex, weight, ASA score, and comorbidities associated with post-induction hypotension, have been described in various studies. Moreover, the post-induction hypotension risk increases 2-fold with a history of hypertension [11]. Our data revealed decreasing of MAP by (12.9 ± 24.7) mm Hg after induction ($p = 0.015$). Only 12.5 % of patients had drop of MAP ≥ 30 % from initial level. All patients showed quick restoration of hemodynamics after fluid boluses.

ICA PSV dynamics were not altered by systemic hemodynamic changes caused by induction to anesthesia. The same results were obtained by Eun-Hee Kim et al. in pediatric patients [12]. The limitation of our study was that we did not use continuous monitoring of blood pressure and flow velocities. Only parameters in 3 min after induction were measured. In such investigation peak parameters may be missed. No guidelines currently exist for invasive perioperative blood pressure monitoring in patients undergoing shoulder arthroscopy in the beach chair position. According to U.S. National Anesthesia Clinical Outcomes Registry (NACOR) only 1.92 % of such surgeries require arterial line placement [13].

After positioning we revealed further decreasing of SBP, DBP and MAP. Our data are similar to those obtained by M. Ko et al. [14]. The total incidence of hypotension after placement into the BCP in their investigation came up 93.65 % in all patients. It was calculated that in BCP the MAP at the level of the brain would decrease by 0.77 mm Hg for each 1 cm in head elevation from a heart-level MAP [15]. Another method of estimate calculation of MAP on the level of brain was proposed by J. Triplet [16]. The mean ratio of cerebral MAP to brachial NIBP is

0.939 in supine position, 0.738 at 30 degrees of incline, and 0.629 at 70 degrees of incline. In patients undergoing shoulder surgery in the BCP, crystalloid fluid loading before positioning appears to be an effective alternative for protecting patients from hemodynamic instability [17].

We hypothesized that decreasing of MAP in BCP would reduce the level of ICA flow velocities. We revealed that after positioning ICA PSV decreased by (15.4 ± 12.8) cm/sec ($p = 0.001$). The same dynamics were found in the recent study by J. Antony et al. [18]. It was revealed a strong correlation between MAP and middle cerebral artery flow velocity, examined by ultrasound transcranial doppler. Further investigations are needed to determine how these changes impact cerebral blood flow, cerebral saturation and cognitive restoration after surgery.

Conclusions

We revealed statistically significant reduction of SBP, DBP and MAP after induction to general anesthesia and after subsequent patient positioning to the BCP.

Post-induction hypotension does not provoke changes in ICA PSV. Subsequent positioning of patients to the BCP leads to statistically significant decreasing of ICA PSV.

Conflict of interest. The authors declare no conflict of interest.

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СИСТЕМНІ ТА ЦЕРЕБРАЛЬНІ ГЕМОДИНАМІЧНІ ЗМІНИ ПРИ АРТРОСКОПІЧНИХ ВТРУЧАННЯХ У ПОЛОЖЕННІ ПЛЯЖНОГО КРИСЛА

К. І. Лизогуб, М. В. Лизогуб

ДУ «Інститут патології хребта та суглобів ім. проф. М. І. Ситенка НАМН України», Харків

✉ Kseniia Lyzohub, MD: kslizogub@gmail.com

✉ Mykola Lyzohub, MD, PhD: nlizogub@gmail.com