Research Article

Cerebral oxygen saturation monitoring during hypotensive anesthesia in shoulder arthroscopy: A comparative study between dexmedetomidine and esmolol

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Abstract *Background:* Beach chair position (BCP) is used in arthroscopic shoulder operations for its advantages. The BCP together with deliberate hypotension used to decrease intraoperative blood loss during arthroscopic shoulder procedures, this may have risk to cause postoperative neurological insults. Dexmedetomidine and esmolol are used to induce deliberate hypotension. Near-infrared spectroscopy (NIRS) provides a non-invasive technique of continuous monitoring of regional cerebral tissue oxygen saturation ($rScO_2$). In this study we evaluate the prevalence of $rScO_2$ during hypotensive anesthesia induced by intra-operative infusion of either dexmedetomidine or esmolol in patients undergoing elective arthroscopic shoulder surgery in the BCP.

Patients and methods: Fifty patients scheduled for elective arthroscopic shoulder surgery under general anesthesia with hypotensive technique in BCP, randomly assigned into two equal groups, dexmedetomidine group (D Group) and esmolol group (E Group) according to the drug used for deliberate hypotension. MAP, HR, BIS and rS_cO_2 were recorded before induction of anesthesia T_0, post-induction of anesthesia T_1 as baseline, 5 min after BCP T_2, 5 min after starting the studied drug T_3, 30 min T_4, 60 min T_5, 90 min T_6, 5 min after stopping the studied drug T_7, 5 min after return to supine T_8 and after extubation T_9.

Results: In D group there was significant decrease in $Lt.rs_co_2$ and $Rt.rs_co_2$, at T2 to T7 compared to T1. In E group there was significant decrease in $Lt.rs_co_2$ and $Rt.rs_co_2$ at T2, to T7 compared to T1. In D group two patients had CDEs compared to five patients in E group.

Conclusion: In patients undergoing shoulder arthroscopic surgery under general anesthesia, the BCP significantly decreases rS_cO_2, with further slight decrease of rS_cO_2 with dexmedetomidine

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and esmolol induced hypotension with no affection of postoperative cognitive function with both drugs. Dexmedetomidine and esmolol are safe drugs with better safety of dexmedetomidine over esmolol.

1. Introduction

In the last decades open anterior stabilization and capsular shift were traditionally considered to be "mainstay and gold standard" for anterior shoulder instability, however, most surgeons lately found that arthroscopic anterior stabilization has become the procedure of choice [1].

The beach chair (BCP) and lateral decubitus (LDP) positions are both reliable techniques to perform effective arthroscopic shoulder operations [2]. The use of BCP for shoulder arthroscopic procedures started from early 1980s [3]. The BCP advantages include lack of brachial plexus strain, proper intra-articular visualization, and ease of conversion to an open approach if necessary [4], depending on joint positioning and surgeon training [5].

The BCP in association with deliberate hypotension has been used to decrease intraoperative blood loss and provide a relatively blood-free surgical environment to improve surgical field [6]. However, this combination has the potential to affect cerebral perfusion pressure and oxygenation during surgery and induce cerebral ischemia [7].

Deliberate hypotension can be achieved by using anesthetics, direct acting vasodilators, β adrenergic blockers and calcium channel blockers. However, it may have the risk to cause postoperative neurological insults due to cerebral hypoperfusion [8], so ideal hypotensive agent should be easy administrated in titration without toxic side effect, have short term effect, no change in cardiac performance and maintaining cerebrovascular autoregulation [9].

Dexmedetomidine (DEX) is an imidazole-derivative adrenoceptor agonist representing high selectivity for α_2 adrenergic agonist [10]. It has a sedative, analgesic and induces inhibition of central sympathetic outflow [11]. It specifically stimulates presynaptic α_2 receptors [12] with dose dependent decrease in arterial pressure, heart rate, cardiac output and norepinephrine release [13].

Esmolol is a cardio-selective β_1 blocker characterized by ultra-short half life of about 2 min. Its peak effect starts 6–10 min after IV injection. It is titrated easily with its short clearance time of average 9 min after stoppage of infusion. These special pharmacokinetic properties have led to the successful use of esmolol in deliberate hypotension [14].

The central nervous system (CNS) is considered to be the main end-point of most general anesthetics, however, it remains the least monitored organ during anesthesia [14]. Near-infrared spectroscopy (NIRS) provides a non-invasive technique of continuous monitoring of regional cerebral tissue oxygen saturation (rScO$_2$) [15]. This method has been demonstrated repeatedly to monitor changes in parenchymal and microcirculatory oxygenation of the frontal cerebral cortex reflecting tissue oxygen use [16] as a marker of the balance between oxygen supply and demand [17], which allow for the immediate recognition and management of cerebral desaturation events (CDEs) that would otherwise be undetected with other conventional intraoperative monitoring during procedures with a high risk of adverse neurological outcomes [18].

The aim of this prospective, randomized, double blind study was to compare the prevalence of regional cerebral oxygen desaturation during hypotensive anesthesia induced by intra-operative infusion of either dexmedetomidine or esmolol in patients undergoing elective arthroscopic shoulder surgery in the beach chair position with further detection of the occurrence of postoperative cognitive dysfunction (POCD).

2. Materials and methods

This prospective, randomized, double blind study was conducted in Prince Salman Hospital in Kingdom of Saudi Arabia after obtaining the approval of the ethical committee board of the hospital from February 2013 till December 2013.

A written informed consent was taken from fifty patients American Society of Anesthesiologists physical status (ASA) I or II, 20–55 years old scheduled to undergo elective arthroscopic shoulder surgery under general anesthesia with hypotensive technique in beach chair position (BCP). Based on a computer-generated randomization list, patients were classified into two groups, dexmedetomidine group (D Group) and esmolol group (E Group) according to the drug used for deliberate hypotension during general anesthesia.

Exclusion criteria included, patients with preexisting cerebral pathology (such as previous episodes of cerebral ischemia or stroke), patients clinically apparent to have neurological or cognitive dysfunction, cervical stenosis, cervical disk herniation, documented carotid stenosis, history of serious psychiatric illness, Mini Mental State Examination (MMSE) score of 23 or less and history of orthostatic hypotension, myocardial infarction or uncontrolled hypertension.

Anesthetic management was carefully standardized in all patients in both groups. Patients were premedicated with midazolam IV 0.02 mg/kg after insertion of a 22 G peripheral intravenous cannula 1 h before induction of anesthesia. Upon arrival to the operating room, standard monitoring (Aisys; Datex-Ohmeda, Inc., a General Electric Company, doing business as GE Healthcare, Madison, Wisconsin, USA) was applied; heart rate (HR), non invasive blood pressure, and oxygen saturation (SPO2) were continuously monitored. 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).

Patients were actively warmed throughout the procedure using a forced-air warming system (Bair-Hugger, Augustine-Medical, Eden Prairie, MN) to maintain intraoperative normothermia as the esophageal temperature was monitored

continuously using (Mon-a-Therm; Mallinckrodt, St. Louis, MO) and maintained at 36–37°.

Regional cerebral oxygen saturation (rS_cO_2) was continuously monitored using near-infrared spectroscopy (NIRS) with an INVOS® 5100C cerebral oximeter (Somanetics, Troy, MI). Before induction of general anesthesia, after cleansing the skin area with alcohol, sensors for cerebral oximeter were placed bilaterally on the right and left sides of the forehead according to the manufacturer's instructions with the caudal border about 1 cm above the eyebrow with the medial edge at the midline as this position makes the light source and sensors away from the frontal sinus and temporalis muscle, rS_cO_2 values from the right and left frontal lobes were averaged to determine cerebral oxygenation. Another 20 G peripheral cannula was inserted; one for administration of the study drug and the other one used for induction of anesthesia.

After full preoxygenation, Granisetron 10 μg/kg IV was given then anesthesia was induced with propofol (1.5–2.0 mg/kg), and fentanyl (2 μg/kg). The trachea was intubated 2–3 min after administration of cis-atracurium 0.15 mg/kg IV, and the lungs were mechanically ventilated with 40% oxygen in nitrous oxide. Anesthesia was maintained by 1–2% end-tidal sevoflurane in 40% oxygen in nitrous oxide flow. Intraoperative fluid administration was 6–8 ml/kg of lactated ringer solution. Invasive blood pressure monitoring was done by using a 20 gauge catheter inserted into a radial artery in the contralateral side of operation and connected to a pressure transducer (Aisys; Datex-Ohmeda, Inc., a General Electric Company, doing business as GE Healthcare, Madison, Wisconsin, USA) for continuous monitoring of invasive blood pressure. The pressure transducer was referenced to the mid-axillary level when patient was supine and referenced to the external ear canal level when in BCP to reflect directly cerebral perfusion pressure.

Approximately 10 min after anesthesia induction, when hemodynamics became stable, the head was secured in 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 to put the patient into the beach-chair position (BCP) using a beach-chair table (T-MAX beach chair; TENET Medical Engineering, Calgary, Alberta, Canada), after which the surgery was started.

After induction of anesthesia and the patient positioned in BCP, D group received loading dose of 1 μg/kg of dexmedetomidine {Precedex® 200 μg/2 ml (Hospira, USA)} diluted in 10 ml of 0.9% normal saline infused over 10 min then iv bolus of 10 ml of 0.9% normal saline, followed by infusion rate 0.2–0.6 μg/kg/h (200 μg dexmedetomidine diluted in 48 ml of 0.9% normal saline injection for a final concentration of 4 μg/ml). Patients who were allocated to E group, started to receive IV 10 ml of 0.9% normal saline over 10 min followed by iv bolus esmolol 0.5 mg/kg {Brevibloc® 100 mg/10 ml (Baxter Deutschland GmbH)} diluted in 10 ml of 0.9% normal saline followed by infusion rate of 0.05–0.3 mg/kg/min throughout the operation (400 mg esmolol added to 10 ml of 0.9% normal saline for a final volume 50 ml with concentration of 8 mg/ml).

Patients were mechanically ventilated to maintain the end-tidal carbon dioxide ($ETCO_2$) tension at 30–35 mmHg. Neuromuscular blockade was carefully controlled by train-of-four monitoring, and additional boluses of cis-atracurium were administered to maintain one twitch response in response to electrical stimulation of the ulnar nerve during the surgical procedure.

At the end of main surgical procedures, deliberate hypotensive drug infusion and incremental doses of cisatracurium were stopped. Paracetamol 1 g IVI over 15 min was given. The residual of neuromuscular blocker was antagonized with neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg. After extubation and full recovery, the patient received 1 mg/kg pethidine IM as postoperative analgesia then transferred to the post anesthesia care unit (PACU).

During the maintenance of anesthesia by the hypotensive technique, tachycardia was defined as a persistent increase in HR exceeding 90 beats/min for more than 5 min, which was treated by IV administration of 1 μg/kg of fentanyl. If bradycardia occurred (HR < 50 beats/min), it was treated with 0.01 mg/kg of atropine intravenously. MAP was maintained between 55 and 65 mmHg, hypo tension less than 50 mmHg of the MAP was treated by a gradual decrease of the studied drug to achieve the desired MAP, whereas an increase of MAP of more than 65 mmHg was treated by increasing the dose of hypotensive drug used.

Routine monitoring included invasive measurement of mean arterial blood pressure (MAP), heart rate (HR), and rhythm by 5-lead electrocardiography, pulse oximetry (O_2Sat), BIS, esophageal temperature and regional cerebral oxygen saturation (rS_cO_2) throughout the surgery. The end-tidal concentrations of carbon dioxide, sevoflurane, and nitrous oxide were measured using a gas analyzer.

These variables (MAP, heart rate, BIS and rS_cO_2) were recorded before induction of anesthesia T_0 (supine position), post-induction of anesthesia T_1 (supine position) as baseline values, 5 min after BCP T_2, 5 min after starting the studied drug T_3, 30 min after starting the studied drug T_4, 60 min after starting the studied drug T_5, 90 min after starting the studied drug T_6, 5 min after stopping the studied drug T_7, 5 min after change from BCP to supine position T_8 and after extubation of the patient T_9.

Baseline rS_cO_2 was defined as the average saturation value over a 1-min period after induction of general anesthesia. Cerebral desaturation was defined as a reduction of rS_cO_2 less than 75% of baseline for more than 15 s [19,14], which should be treated by the attending anesthesiologist by checking the ventilator, head position, and tubing system, increasing FiO_2, increasing end-tidal CO_2 partial pressure if the end-tidal CO_2 was less than 30 mmHg, and increasing arterial blood pressure with intravascular fluid administration and/or vasopressor drugs as ephedrine (5 mg IV) if MAP pressure was less than 50 mmHg [20].

The number and duration of cerebral desaturation events (CDEs) together with the number and type of interventions used to treat CDEs were recorded. In addition, interventions used to treat MAP reductions which did not decrease $rScO_2$ were also noted. All these data were recorded from BCP until tracheal extubation.

At postoperative visit on the evening of surgery, the patient was assessed neurologically by an anesthetist who was not related to the study, for 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.

Cognitive function of the patients was assessed using the MMSE test the day before surgery and then reassessed 1 wk

after the operation. The **mini-mental state examination** (MMSE) or **Folstein test** is a brief 30-point questionnaire test that is used to screen for cognitive impairment. The MMSE test includes simple questions and problems in a number of areas: the time and place of the test, repeating lists of words, arithmetic, language use and comprehension, and basic motor skills [21] (Table 1).

Postoperative cognitive dysfunction (POCD) was considered when there was decrease in MMSE score 2 points or more from preoperative value [20,22].

3. Statistical analysis

Group sample sizes of 25 per group achieved 80% power to detect a clinically relevant decrease in cerebral oxygen saturation with mean difference of 10 and estimated SD of 5 and with a significance level (alpha) of 0.05 using a two-sided two-sample t-test.

Statistical analysis was done on a personal computer using the Statistical Package for Social Sciences version 16.0 (SPSS© v. 16.0, SPSS Inc., Chicago, IL, USA). Qualitative data were analyzed with Pearson's Chi-square test and were presented as number [%]. Quantitative data were analyzed using unpaired t-student test for between group comparison, and paired t-test for within group comparison, data were presented as mean ± standard deviation. A P value of <0.05 was considered statistically significant.

4. Results

A total of fifty-two adult patients were enrolled in this study, two cases were excluded throughout the course of the study, one case excluded due to incomplete data collection and other one due to unplanned conversion to open surgical procedure.

There was no statistical significant difference in the patients' demographic data as regards age, sex, weight and ASA classification (Table 2).

There was no statistical significant difference between both groups as regards patient characteristics (see Table 3).

As regards the change in MAP, within group D, there was significant decrease in MAP T2 (71.52 ± 8), T3 (64.5 ± 4), T4 (61.5 ± 2.9), T5 (59.5 ± 2.6), T6 (58.2 ± 2.7) when compared to T1 (83.5 ± 8.5), and insignificant decrease in MAP in T7 (77.8 ± 8.4) and T8 (79.6 ± 5.36) compared to T1 (83.5 ± 8.5). There was no significant change in MAP T9 (80.6 ± 7.3) when compared to T1 (83.5 ± 8.5) (Table 4).

As regards the change in MAP, within group E, there was significant decrease in MAP T2 (69.4 ± 6.7), T3 (64 ± 3.3), T4 (62.2 ± 5.7), T5 (59.4 ± 3.2), T6 (58.5 ± 3.7) when compared to T1 (83.8 ± 10), and insignificant decrease in MAP in T7 (78.2 ± 5.1) and T8 (81.3 ± 9.3) compared to T1 (83.8 ± 10). There was no significant change in MAP T9 (82.4 ± 5.9) when compared to T1 (83.8 ± 10) (Table 4).

As regards the change in MAP, in between groups, there was no significant difference between both groups in different time intervals (Table 4).

As regards the change in HR, within group comparison in D group, there was significant decrease in HR T2 (76.2 ± 8), T3 (76.9 ± 10), T4 (73.9 ± 6.7), T5 (69.6 ± 6.9), T6 (66.3 ± 7) when compared to T1 (88.4 ± 10.1), and insignificant decrease in HR in T7 (77.3 ± 8.7), T8 (76.8 ± 9.7) and T9 (77.6 ± 4) compared to T1 (88.4 ± 10.1) (Table 5).

While, the change in HR, within group comparison in E group, there was significant decrease in HR T2 (74.5 ± 9), T3 (77.8 ± 10), T4 (71.8 ± 6.2), T5 (67.4 ± 5.6), T6 (67.8 ± 6.4) when compared to T1 (87.3 ± 3.9), and insignificant decrease in HR in T7 (78.7 ± 7.6) and T8 (81.6 ± 6.4) compared to T1 (87.3 ± 3.9). There was no significant change in HR T9 (82 ± 7) when compared to T1 (87.3 ± 3.9) (Table 5).

As regards the change in HR, in between groups, there was no significant difference between both groups in different time intervals (Table 5).

As regards the BIS readings representing the depth of anesthesia, there was no significant difference within group comparison in both group or in between groups in different time intervals except if we compared T9 with T1 within group comparison in both group as the patient is awake in T9 reading Fig. 1.

As regards the change in $Lt.rs_co_2$ within D group, there was significant decrease in $Lt.rs_co_2$ at T2 (76.8 ± 7.8), T3 (71 ± 5.8), T4 (70.4 ± 4.1), T5 (69.6 ± 3.5), T6 (72 ± 5.3) and T7 (71 ± 5.2) when compared to T1 (88.6 ± 4.3) and insignificant decrease in $Lt.rs_co_2$ in T8 (86.7 ± 7.7) compared to T1 (88.6 ± 4.3). There was no significant change in $Lt.rs_co_2$ at T9 (87.7 ± 4.1) when compared to T1 (88.6 ± 4.3) (Table 6).

As regards the change in $Lt.rs_co_2$ within in E group, there was significant decrease in $Lt.rs_co_2$ at T2 (73.5 ± 7.5), T3

Category	Possible points	Description
Orientation to time	5	From broadest to most narrow. Orientation to time has been correlated with future decline
Orientation to place	5	From broadest to most narrow. This is sometimes narrowed down to streets and sometimes to floor
Registration	3	Repeating named prompts
Attention and calculation	5	Serial sevens, or spelling "world" backwards It has been suggested that serial sevens may be more appropriate in a population where English is not the first language
Recall	3	Registration recall
Language	2	Naming a pencil and a watch
Repetition	1	Speaking back a phrase
Complex commands	6	Varies. Can involve drawing figure shown

Table 1 Mini-mental state examination (MMSE).

Table 2 Demographic data in both groups.

	D group (Dexmedetomidine) ($n = 25$)	E group (Esmolol) ($n = 25$)	P value
Age (yrs)	41.1 ± 10.3	36.9 ± 12	0.2
Sex (M/F)	12/13	14/11	0.6
Weight (kg)	81.6 ± 9.2	83.3 ± 11.4	0.57
ASA I No (%)	11 (44%)	12 (48%)	0.5
ASA II No (%)	14 (56%)	13 (52%)	

Data were expressed as mean $\pm$ SD or number (%).

(69.9 ± 6.3), T4 (71 ± 6.2), T5 (65.6 ± 5.7), T6 (70 ± 5.5) and T7 (68.4 ± 4.8) when compared to T1 (86.9 ± 3.7), and insignificant decrease in Lt.rs$_c$o$_2$ in T8 (85.5 ± 5.2) compared to T1 (86.9 ± 3.7). There was no significant change in Lt.rs$_c$o$_2$ at T9 (85.7 ± 4.4) when compared to T1 (86.9 ± 3.7) (Table 6).

As regards the change in Lt.rs$_c$o$_2$ in between groups, there was no significant difference between both groups in different time intervals except at T5 there was significant decrease in E group (65.6 ± 5.7) in comparison with D group (69.6 ± 3.5) at this time Table 6.

As regards the change in Rt.rs$_c$o$_2$, within D group, there was significant decrease in Lt.rs$_c$o$_2$ at T2 (76 ± 4.6), T3 (70.1 ± 5.6), T4 (71.2 ± 5.4), T5 (70 ± 3), T6 (68.4 ± 3.8) and T7 (70.9 ± 4) when compared to T1 (87.6 ± 5), and insignificant decrease in Lt.rs$_c$o$_2$ in T8 (85 ± 4.3) compared to T1 (87.6 ± 5). There was no significant change in Lt.rs$_c$o$_2$ at T9 (87.8 ± 4.4) when compared to T1 (87.6 ± 5) (Table 7).

As regards the change in Rt.rs$_c$o$_2$, within E group, there was significant decrease in Lt.rs$_c$o$_2$ at T2 (74.3 ± 10), T3 (71.9 ± 4.2), T4 (70.6 ± 4.2), T5 (66.3 ± 4.4), T6 (68.7 ± 4.8) and T7 (71.6 ± 3.8) when compared to T1 (87.6 ± 6.8), and insignificant decrease in Lt.rs$_c$o$_2$ in T8 (84.5 ± 4.5) compared to T1 (87.6 ± 6.8). There was no significant change in Lt.rs$_c$o$_2$ at T9 (88 ± 4.5) when compared to T1 (87.6 ± 6.8) (Table 7).

As regards the change in Rt.rs$_c$o$_2$, in between groups, there was no significant difference between both groups in different time intervals except at T5 there is significant decrease in E (66.3 ± 4.4) group in comparison with D (70 ± 3) group at this time (Table 7).

Table 4 Changes of mean arterial blood pressure in both D group (Dexmedetomidine) and E group (Emolol).

	D group (Dexmedetomidine) ($n = 25$)	E group (Esmolol) ($n = 25$)	P value (between groups)
T1	83.5 ± 8.5	83.8 ± 10	0.9
T2	$71.52 \pm 8^*$	$69.4 \pm 6.7^*$	0.32
T3	$64.5 \pm 4^*$	$64 \pm 3.3^*$	0.3
T4	$61.5 \pm 2.9^*$	$62.2 \pm 5.7^*$	0.5
T5	$59.5 \pm 2.6^*$	$59.4 \pm 3.2^*$	0.9
T6	$58.2 \pm 2.7^*$	$58.5 \pm 3.7^*$	0.7
T7	77.8 ± 8.4	78.2 ± 5.1	0.8
T8	79.6 ± 5.36	81.3 ± 9.3	0.4
T9	80.6 ± 7.3	82.4 ± 5.9	0.7

Data were expressed as mean $\pm$ SD. P value > 0.05 was considered statistically not significant.
No statistically significant difference between the 2 groups.
* P value < 0.001 compared to T1 within the same group.

Table 5 Changes of heart rate in both D group (Dexmedetomidine) and E group (Emolol).

	D group (Dexmedetomidine) ($n = 25$)	E group (Esmolol) ($n = 25$)	P value (between groups)
T1	88.4 ± 10.1	87.3 ± 3.9	0.62
T2	$76.2 \pm 8^*$	$74.5 \pm 9^*$	0.3
T3	$76.9 \pm 10^*$	$77.8 \pm 10^*$	0.8
T4	$73.9 \pm 6.7^*$	$71.8 \pm 6.2^*$	0.25
T5	$69.6 \pm 6.9^*$	$67.4 \pm 5.6^*$	0.22
T6	$66.3 \pm 7^*$	$67.8 \pm 6.4^*$	0.44
T7	77.3 ± 8.7	78.7 ± 7.6	0.26
T8	76.8 ± 9.7	81.6 ± 6.4	0.37
T9	77.6 ± 4	82 ± 7	0.053

Data were expressed as mean $\pm$ SD. P value > 0.05 was considered statistically not significant.
No statistically significant difference between the 2 groups.
* P value < 0.001, compared to T1 within the same group.

As regards the occurrence of cerebral desaturation events (CDEs), only two patients had CDEs in D group compared to five patients had CDEs in E group. Each patient in D group had only one CDEs attack while in E group, three patients had

Table 3 Patients' characteristics in both groups.

	D group (Dexmedetomidine) ($n = 25$)	E group (Esmolol) ($n = 25$)	P value
Duration of anesthesia (min)	160.4 ± 11.2	155.4 ± 14.1	0.2
Duration of surgery (min)	143 ± 14.4	132 ± 14.2	0.09
Duration of BCP (min)	127 ± 14	116.6 ± 14	0.11
Duration of deliberate hypotension (min)	109.7 ± 10.7	105.8 ± 12	0.15
MAP T_0 (mmHg)	89.3 ± 11.9	85.3 ± 8	0.2
Heart rate T_0 (beat/min)	84.8 ± 6.3	80.7 ± 9.2	0.07
BIS (T_0)	87.6 ± 4.7	87.8 ± 3.5	0.8
Lt.rs$_c$o$_2$ (T_0)%	83 ± 5.3	81 ± 5	0.3
Rt.rs$_c$o$_2$ (T_0)%	82.7 ± 6.6	81 ± 6.2	0.2

Data are expressed as mean $\pm$ SD.
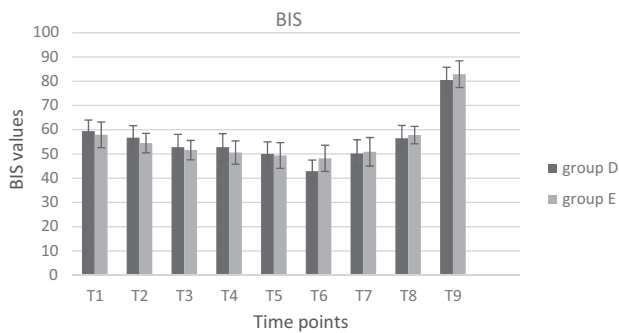P-value > 0.05 is considered statistically non-significant.

Figure 1 BIS at different time intervals. Columns are mean and error bars are SD.

Table 6 Changes of $Lt.rs_co_2$ in both D group (Dexmedetomidine) and E group (Emolol).

	D group (Dexmedetomidine) ($n = 25$)	E group (Esmolol) ($n = 25$)	P value (between groups)
T1	88.6 ± 4.3	86.9 ± 3.7	0.15
T2	76.8 ± 7.8*	73.5 ± 7.5*	0.26
T3	71 ± 5.8*	69.9 ± 6.3*	0.54
T4	70.4 ± 4.1*	71 ± 6.2*	0.79
T5	69.6 ± 3.5*	65.6 ± 5.7*†	0.004
T6	72 ± 5.3*	70 ± 5.5*	0.3
T7	71 ± 5.2*	68.4 ± 4.8*	0.06
T8	86.7 ± 7.7	85.5 ± 5.2	0.5
T9	87.7 ± 4.1	85.7 ± 4.4	0.11

Data were expressed as mean ± SD. P value > 0.05 was considered statistically not significant.
* P value < 0.05 was considered statistically significant compared to T1.
† Statistically significant decrease compared to D group.

Table 7 Changes of $Rt.rs_co_2$ in both D group (Dexmedetomidine) and E group (Emolol).

	D group (Dexmedetomidine) ($n = 25$)	E group (Esmolol) ($n = 25$)	P value (between groups)
T1	87.6 ± 5	87.6 ± 6.8	0.9
T2	76 ± 4.6*	74.3 ± 10*	0.26
T3	70.1 ± 5.6*	71.9 ± 4.2*	0.2
T4	71.2 ± 5.4*	70.6 ± 4.2*	0.66
T5	70 ± 3*	66.3 ± 4.4*,†	0.002
T6	68.4 ± 3.8*	68.7 ± 4.8*	0.28
T7	70.9 ± 4*	71.6 ± 3.8*	0.56
T8	85 ± 4.3	84.5 ± 4.5	0.69
T9	87.8 ± 4.4	88 ± 4.5	0.8

Data were expressed as mean ± SD. P value > 0.05 was considered statistically not significant.
* P value < 0.05 was considered statistically significant compared to T1.
† Statistically significant decrease compared to D group.

one CDEs attack and the other two patients each one had two CDEs attack. Patients in D group who had CDEs attack require no interventional management and disappeared spontaneously, while in E group two patients of the five who had CDEs required management by the attending anesthetist to treat the CDEs attacks as they last more than 30 s (Table 8).

As regards the MMSE score there was no significant difference in both groups within group comparison or in-between group comparison Fig. 2.

P value > 0.05 was considered statistically not significant.

5. Discussion

This prospective, randomized, double blind study was conducted to compare the prevalence of rS_cO_2 during hypotensive anesthesia induced by intra-operative intra-venous infusion of either dexmedetomidine or esmolol in patients undergoing elective arthroscopic shoulder surgery in the BCP with further detection of the occurrence of POCD, as the association of deliberate hypotension with BCP under general anesthesia has the potential to induce changes in cerebral perfusion pressure which may be the major etiology of poor neurological outcomes [23].

Alterations in systemic hemodynamics occur when postural conditions are changed from supine to BCP, including diminished cardiac index, stroke volume, and arterial pressure [24] caused by marked decrease of venous return which persists for up to 30 min after the positioning in an anesthetized patient [25]. In conscious subjects, sitting position activates the sympathetic nervous system, resulting in an increase of systemic blood pressure associated with increased systemic vascular resistance and sustained reduction in cardiac output. Compensatory increases in sympathetic nervous system activation and systemic vascular resistance are attenuated under general anesthesia causing more pronounced hypotension [26].

In the current study, MAP decreased abruptly from the baseline values (T1) when patients were switched to BCP in both groups, with a statistically significant value. MAP was not statistically significant between the 2 groups during the study. Our findings corroborate previous studies of Fuchs and his colleagues [27] and Tange and his colleagues [28] who have confirmed considerable hemodynamic shift when positioning from the supine to the BCP.

In D group, this hemodynamic profile after starting the hypotensive agents can be attributed to the known sympatholytic effect of α_2 agonists which are involved in regulating the autonomic and cardiovascular systems [29]. Dexmedetomidine sympatholytic action is manifested by dose-dependent decrease in arterial blood pressure, heart rate, cardiac output and norepinephrine release [30]. This was coinciding with Basar and his colleagues [31] who investigated the effect of single dose of dexmedetomidine and reported significant reduction in MAP and HR.

Esmolol is an ultra-short acting selective B_1 adrenergic antagonist and possesses no significant membrane stabilizing activity, given as continuous infusion to induce stable and controllable beta-adrenergic block [32], providing a stable course of controlled hypotension and produces beneficial effects in the surgical field and in blood conservation [33]. Negative chronotropic and ionotropic effects of esmolol decrease the

Table 8 Cerebral desaturation events (CDES).

	D group (Dexmedetomidine) ($n = 25$)	E group (Esmolol) ($n = 25$)	P value
No. of patient with CDEs 0:1:2	23:2:0	20:3:2	0.7
Management of CDEs (number of patients)	0	2	0.35

0:1:2 = represent number of CDE events. Data are presented as ratio.
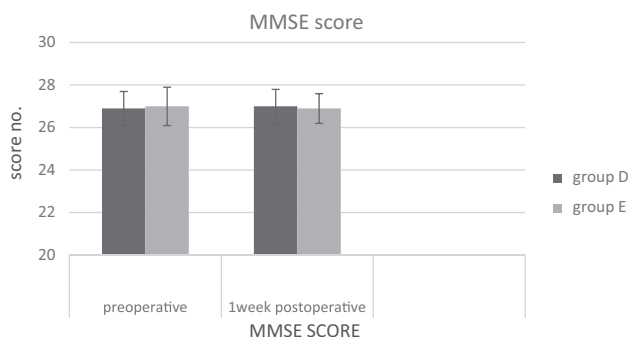P value > 0.05 was considered statistically not significant.

Figure 2 MMSE score at different time interval. Columns are mean and error bars are SD.

cardiac output which lowers arterial blood pressure. When heart rate and strength of contractions are decreased this will reduce arterial pressure and carotid sinus baroreceptors response [34].

Esmolol had extremely short half-life (about 9 min). This explained that there was no significant change in HR in T9 when compared to T1, in contrast to dexmedetomidine which had insignificant decrease in HR in T9 when compared to T1, as it had more prolonged action compared to esmolol.

In the present study, MAP was maintained between 55 and 65 mmHg as outcome studies have demonstrated that a reduction of the mean arterial pressure (MAP) to 55–65 mmHg or a decrease of 30% from baseline is safe with regards to endorgan perfusion and oxygen delivery [35].

During this research work, invasive blood pressure was continuously measured at the level of the external auditory meatus, which is representing the level of the circle of Willis, to assess accurately cerebral perfusion pressure. This was explained by the "open model" or "waterfall" theory, as blood flows vertically from the heart, there is a reduction in arterial pressure directly related to the weight of the column of blood [36,37]. This was coinciding with ko and his colleagues [38] who stated that "Monitoring cerebral rS_cO_2 and MAP at the level of brain can be helpful to detect the possibility of cerebral desaturation earlier". However, Pranevicius and his colleagues [39] stated that no need for modifying the level of measurement as long as the CVP can be maintained above 18 mmHg.

Factors which may modulate cerebral oximetry values include depth of anesthesia, anesthetic technique, arterial carbon dioxide concentrations, inspired oxygen content and MAP [40]. Therefore, anesthetic management was carefully standardized in all patients in both groups, MAP was maintained between 55 and 65 mmHg, end-tidal carbon dioxide ($ETCO_2$) tension at 30–35 mmHg and BIS readings representing the depth of anesthesia, there was no significant difference within group comparison in both group or in between groups comparison in different time intervals reflecting stable depth of anesthesia in both groups all through the anesthesia course.

Brain oxygenation can be measured invasively by jugular bulb oximetry to measure jugular venous oxygen saturation ($Sjvo_2$) as an index of cerebral blood flow/metabolism coupling [15], $Sjvo_2$ is generally accepted as a method for the monitoring of global cerebral oxygen saturation and rS_cO_2 as a method for monitoring regional oxygen saturation [19].

NIRS measures the ratio of oxyhemoglobin to total hemoglobin in a field beneath the sensor, and this ratio is expressed as a percentage of rS_cO_2 on the right and left side [41]. rS_cO_2 has been reported to be more sensitive than jugular venous oxygen saturation $Sjvo_2$ to brain ischemia. Also, McLeod and his colleagues found that NIRS correlated more closely with changes in $Sjvo_2$ [42]. So, rS_cO_2 may be an alternative method for detecting brain ischemia during induced hypotension [43].

In the present study, there was significant decrease in both $Rt.rs_co_2$ and $Lt.rs_co_2$ with BCP after induction of general anesthesia in both groups, however Fuchs and his colleagues [27] found that rS_cO_2 values in the BCP were not different from those in the supine position in non-anesthetized volunteers. However, rS_cO_2 values in the sitting position were significantly lower than those in the supine position in anesthetized patients, although their MAP was maintained. These results were explained by the role of general anesthesia in impairment of regulatory pathways of peripheral circulation and cerebral autoregulation and thus indicate that changes in $rScO_2$ related to BCP cannot be only explained by changes in hemodynamic parameters as MAP.

In our study $Rt.rs_co_2$ and $Lt.rs_co_2$ values were influenced also by deliberate hypotension in both groups. Our results were agreed with Lee and his colleagues [44] who found that the BCP combined with induced hypotension significantly decreases rSO_2 in patients undergoing shoulder arthroscopic surgery under general anesthesia; however they used remifentanil and nitroglycerine in deliberate hypotension. Also, Endoh and his colleagues [45] who found decreased rs_co_2 in controlled hypotension induced by nicardipine. But in our work it was found that there was significant decrease in $Rt.rs_co_2$ and $Lt.rs_co_2$ in E group compared with D group at T5, but this was in contrast to the results of Heinke and his colleagues [46] who stated that moderate esmolol doses do not change cognitive performance and had no direct effects on CBF, so this excludes regional or global effects of esmolol on CBF. However, this study did not use higher esmolol doses which may cause a greater reduction of cardiovascular variables. Moreover, this study was done in awake volunteers, so whether these data can be applied under general anesthesia with BCP, needs to be clarified in future investigations.

The incidence of CDEs in patients monitored with NIRS undergoing elective shoulder surgery in the beach chair position in many previous studies is quite variable and ranges from 8% to 80% [47,48,44,49,28]. Despite the frequent occurrence of CDEs in these patients, no clear neurologic insults were documented [48]. This can be explained by limited duration of the surgical procedure, as severity and duration of cerebral hypoperfusion are critical determinants of tissue damage, so, viability-time thresholds usually not exceeded to cause neurologic deficits [50].

The normal range for rS_cO_2 values was variable in different studies, Madsen and his colleagues [51] stated that the normal range for rS_cO_2 values in 39 healthy resting subjects is 55–78, Similarly, Casati and his colleagues [14] reported baseline rS_cO_2 values 63 ± 8 in healthy elderly patients undergoing general surgery, also Kim and his colleagues [52] reported that mean baseline rS_cO_2 value was 71 ± 6 in healthy volunteers aged 20–36 years.

In the present study we defined cerebral desaturation events (CDEs), as a reduction of rS_cO_2 less than 75% of baseline for more than 15 s in agreement with Casati and his colleagues [22] and Iglesias and his colleagues [53], however other literature defined a CDE as a drop in rS_cO_2 of 20% or greater from baseline or an rS_cO_2 of less than 55% for any time period [18] and this definition depend on the fact that in conscious patients, a 20% decrease in frontal lobe oxygenation is associated with clinical symptoms and signs of cerebral ischemia [54]. Also during carotid endarterectomy, 20% or greater drop in rS_cO_2 during clamping of carotid artery is associated with a 20-fold increase in obvious signs and symptoms of cerebral ischemia [55].

In the present study, the MMSE was used for evaluating cognitive function as it has many advantages include high validity and reliability, ease of use, brevity and suitability for bedside use. Also, MMSE is very appropriate for repeated cognitive assessments over time [22]. There are other sensitive and specific scores as regards the assessment of cognitive functions evaluating different components of cognitive function; however, they have major drawback of lengthy, complex administration guidelines and long time in its application [56].

In our study, as regards the MMSE score there was no significant difference in both groups within group comparison or in-between group comparison. This was coinciding with Williams-Russo and his colleagues [57] who found no POCD after hypotensive epidural anesthesia in elderly patients. Also, Townes and his colleagues [58] reported there were no neuropsychological changes or decrease in MMSE score after induced hypotension in healthy young adults.

The absence of a significant difference between preoperative and postoperative MMSE values could be explained by proper management of any intra-operative CDEs. Also, proper monitoring of rS_cO_2 values after BCP and induced hypotension preserve intra-operative cerebral perfusion and oxygenation which may protect the CNS from any insult.

6. Conclusion

In patients undergoing shoulder arthroscopic surgery under general anesthesia, the beach-chair position significantly decreases rS_cO_2, with further slight decrease of rS_cO_2 with dexmedetomidine and esmolol induced hypotension with no affection of postoperative cognitive function with both drugs.

Dexmedetomidine and esmolol are safe drugs for induced hypotension as rS_cO_2 values are well maintained during hypotension with better safety of dexmedetomidine over esmolol which had more incidences of intraoperative CDEs.

Conflict of interest

None.

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