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Impact of pre-spinal atropine on post spinal hemodynamic and cardiac output measured by electrical cardiometry in cesarean delivery, a randomized controlled trial

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ABSTRACT

Background: Spinal anesthesia is a widely used technique for cesarean delivery, but it often results in hypotension, bradycardia, and reduced cardiac output (CO). Atropine has a potent muscarinic receptor antagonist activity in the heart. It may be a good choice to prevent post-spinal bradycardia and minimize the marked CO reduction.

Methods: Sixty pregnant women between the ages of 18 and 40 who were ASA-PS II and planned for elective cesarean delivery were divided into two equal groups at random. Both groups received spinal anesthesia. Atropine group (I) ($n = 30$): patients received 0.01 mg/kg atropine, while control group (II) ($n = 30$): patients received the same volume of saline. CO measured by electrical cardiometry (EC) was the primary outcome where, heart rate (HR), mean blood pressure (MBP), stroke volume (SV), systemic vascular resistance (SVR), and neonatal outcomes were the secondary outcomes.

Results: CO after the intervention was higher in the atropine (group I) than in the control (group II). Also, CO reduction at 5 and 10 min following spinal anesthesia was less in the group I than in the group II. Except for baseline reading, HR was significantly higher in the atropine group versus the control group. MBP was higher in the atropine group than in the control group in all readings. SV and SVR were similar in both groups. Neonatal outcomes were equivalent in both groups.

Conclusion: Pre-spinal atropine was effective in preventing post-spinal bradycardia and minimized CO reduction in patients undergoing elective cesarean delivery under spinal anesthesia.

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1. Introduction

Worldwide, there are an estimated 23 million cesarean deliveries performed each year, making it the most common obstetric surgical procedure [1]. Single-shot spinal anesthesia is the most frequently employed anesthetic method for cesarean delivery all over the world [2]. Spinal anesthesia is practically simpler to apply than epidural anesthesia because it results in a dense blockade quickly, minimizing the need for additional intravenous analgesics or switching to general anesthesia [3].

As only a small volume of local anesthetic is required to establish a functional spinal blockade, spinal anesthesia is linked with negligible maternal risk for systemic local anesthetic toxicity and with minimal drug transfer to the fetus [4].

A frequent adverse effect of spinal anesthesia is hypotension, which, if severe and prolonged, can impair uteroplacental perfusion and result in fetal circulation hypo-perfusion, acidosis, and subsequent neonatal depression [5].

Strongly detrimental maternal outcomes from severe hypotension and bradycardia include disturbances in consciousness, pulmonary aspiration, apnea, and cardiac arrest [6,7], and [8].

Heart rate HR, SV, and SVR all influence CO, which is necessary for adequate systemic perfusion. To ensure that cellular metabolic oxygen demand is met, a complex interaction between HR, blood flow, SVR, and MBP exists [9–11].

The anticholinergic drug atropine is a broad-spectrum muscarinic receptors antagonist. Five muscarinic receptors have been identified (M_1-M_5).M2 receptors are expressed in the heart where their inhibition accelerates heart rate and nodal activity and increases atrial contractility. Atropine can treat bradycardia in both the mother and the fetus [12].

Also, atropine is used to minimize the muscarinic cardiovascular side effects of cholinesterase inhibitors [13].

Based on the Electrical Velocimetry model, EC is a more recent technology that continuously and

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non-invasively measures CO, SV, SVR, and other derived parameters [14,15].

The technique is based on evaluating the cyclic changes in the thoracic electrical bio-impedance throughout a cardiac cycle. Through the use of thoracic electrical bio-impedance (TEB), it is possible to calculate SV and subsequently CO by monitoring changes in the electrical conductivity of blood flow in the aortic arch. Nowadays, EC is reliable for performing advanced CO monitoring in critically ill patients [16,17], obstetric patients receiving regional anesthesia [18], and monitoring during hemodialysis [19].

This prospective, randomized study sought to demonstrate the effectiveness of pre-spinal atropine in improving post-spinal hemodynamics and minimizing CO reduction after Spinal anesthesia under continuous monitoring by electrical cardiometry in patients undergoing cesarean delivery.

2. Materials and methods

2.1. Study design

The study protocol had a code number (FMASU R216/2022) after being approved by the research ethics committee of the Faculty of Medicine at Ain Shams University, the protocol was then registered on ClinicalTrials.gov under the registration number NCT05658380. This prospective, single-center, randomized, double-blinded, controlled study enrolled 60 pregnant patients who were listed to undergo elective cesarean delivery under spinal anesthesia after obtaining informed consent. The study was carried out at the obstetrics and gynecology hospital, Ain Shams University.

2.2. Recruitment

Patients who were full term, aged 18 to 40 yrs. with a body mass index (BMI) less than 30 kg/m^2, American Society of Anesthesiologists Physical Status (ASA-PS) II was involved in the study. Exclusion criteria included patients with BMI ≥30 kg/m^2, ASA-PS > II, polyhydramnios, abnormalities in coagulation, injection site infection, low fixed cardiac output states (stenotic valvular heart disease and hypertrophic obstructive cardiomyopathy), cardiac arrhythmias or heart block, disorders associated with hypertension during pregnancy as preeclampsia and eclampsia, thyrotoxicosis, cerebrovascular diseases, or had fetal malformation. Patients were also excluded if they refused to participate in the study, or had a hypersensitivity, allergy, or contraindications to the studied drugs.

2.3. Randomization and blinding

Computer-generated lists and a closed-envelope approach were used to randomly assign patients to one of the following groups in a 1:1 ratio: (30 patients each).atropine group (I) ($n = 30$): patients received 0.01 mg/kg intravenous atropine in 5 ml normal saline 0.9% before induction of spinal anesthesia. Control group (II) ($n = 30$): patients received the same volume of normal saline 0.9% before induction of spinal anesthesia.

An obstetric anesthesiologist who was unaware of the study's protocol prepared the study drugs. The group allocations were concealed from the patients, surgeons, and research personnel who recruited participants and gathered trial data.

2.4. Anesthetic management

2.4.1. Study procedures

Pre-anesthesia consultation was done that reviewed the maternal health and anesthetic history, important obstetric history, laboratory investigations, allergies, and baseline blood pressure and HR measurements. Physical examination of an airway, heart, and chest reliable with the (ASA-PS) guidelines was done.

In the operating room, insertion of functional intravenous access (18-gauge) was done and then lactated Ringer's solution was infused to all patients as a (co-load). Patients were turned to the supine position with a 15º left lateral tilt, basic monitoring consisting of maternal pulse oximetry (Spo2), electrocardiography (ECG), and noninvasive blood pressure (NIBP) was applied, and electrical cardiometry with 4 ECG electrodes (2 in the lateral side of the neck and 2 in the lateral chest wall) (ICON; Cardiotronic, Inc, LaJolla, CA92307; Osypka Medical Gmbh; Berlin, Germany) was applied. Baseline line measurements including HR, MBP, SV, CO, and SVR were reported.

Before induction of spinal anesthesia, Group I received 0.01 mg/kg of intravenous atropine in 5 ml of normal saline 0.9%, while Group II received the same volume of normal saline 0.9% as a control group. Under complete aseptic conditions, spinal anesthesia was performed at the L3–L4 interspace, while patients were in the sitting position, using the paramedian approach, a 27-gauge, noncutting, pencil-point, spinal needles (Penecan® BRAUN, Germany), was used to perform the block.

After documenting free droplets of cerebrospinal fluid, 10 mg hyperbaric bupivacaine plus 20 µg fentanyl was injected into the subarachnoid space. Patients were returned to the supine position with a 15 º left lateral tilt and 10 º head up. Hemodynamic parameters (HR, MBP) and electrical cardiometry-derived measures (CO, SV, and SVR) were recorded at specific times, baseline (T1), 5 minutes after injection of intervention drugs (T2), 5 minutes after spinal anesthesia (T3),), 10

minutes after spinal anesthesia (T4),5 minutes after delivery (T5), and at the end of surgery (T6).

Ephedrine increments (3–6 mg) were used to treat post-spinal hypotension, which is defined as MBP less than 80% baseline measurement.

The primary outcome was the changes in the cardiac output values at the specific time measurements. Values of HR, MBP, SV, SVR, and neonatal outcomes were the secondary outcomes.

2.4.2. Sample size calculation and Statistical methods. The G*Power program version 3.1.0 was used to determine the necessary sample size. Findings from a prior study [20], revealed that the control group's cardiac output during spinal anesthesia was 5.43 1. We estimated the sample size (effect size, d = 0.8) to identify a 15% difference in cardiac output between the control and atropine groups. With 80% power and a type I error of 0.05, a sample size of 26 cases in each group is required. For a total of 60 instances, we included 30 cases in each group to account for dropouts.

The statistical package for social sciences (SPSS) software from IBM Corp., Chicago, USA 2013, version 22.0 was used for data management and analysis. Following the Shapiro-Wilk test for normality, independent t-tests were used to compare quantitative normally distributed variables that are expressed as mean SD (standard deviation). The chi-square test is used to compare qualitative variables expressed as a number or percentage, and Fisher's exact test was used for variables with small expected numbers. If a p-value is less than 0.050, it was considered significant; otherwise, it was not.

3. Results

For the study's eligibility requirements, 74 pregnant patients underwent screening. Five patients did not want to participate, and nine patients were excluded because they did not meet the inclusion criteria. Following enrollment and informed consent, 60 patients were included in the study. In two groups of 30 patients each, they were randomly divided. (Figure 1).

In terms of patients' characteristics (age, BMI, weight, height, and ASA-PS), type of cesarean, induction delivery time, and duration of operation, there were no significant differences between the studied groups (Table 1).

Regarding hemodynamic parameters, no significant statistical differences between the study groups

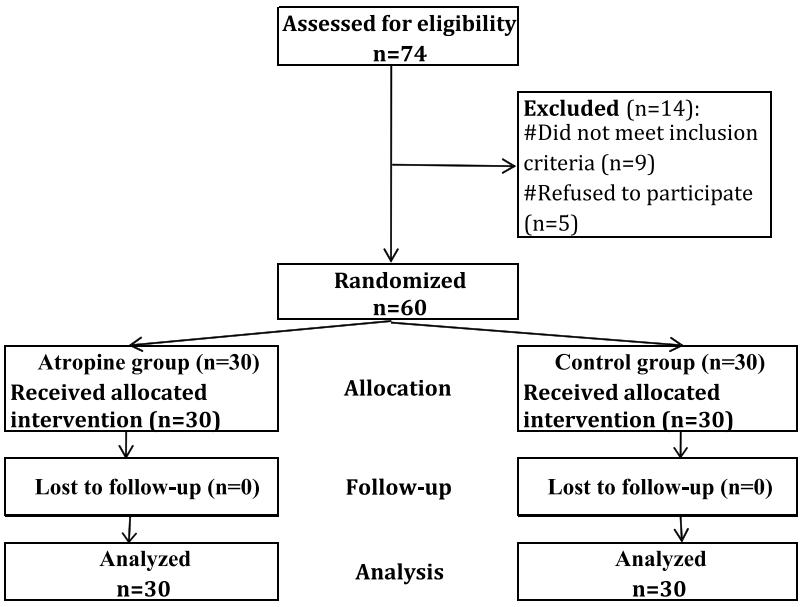

Figure 1. Flow chart of the studied cases.

Table 1. Demographic and operative characteristics of the study groups.

Variables		Group I (Total=30)	Group II (Total=30)	p-value
Age (years), Mean±SD		27.5±5.0	29.1±5.4	^.239
Weight (kg), Mean±SD		66.8±3.8	67.2±4.2	^.687
Height(cm)		172.1±5.0	171.9±5.3	^.861
BMI (kg/m^2), Mean±SD		22.6±1.0	22.8±1.0	^.456
The cesarean section number, (n, %)	First	12 (4.0%)	11 (36.7%)	#0.791
	Repeated	18 (6.0%)	19 (63.3%)	
Gestational age (weeks), Mean±SD		38.9±.6	38.8±.6	^.412
Induction-delivery time (min.), Mean±SD		18.1±3.2	17.9±2.7	^.828
Operation duration (min.), Mean±SD		83.2±13.9	81.7±13.7	^.676

BMI: Body mass index. ^Independent t-test. #Chi square test

Table 2. Comparison regarding heart rate, mean blood pressure, and total ephedrine consumption.

Time	Group I (Total=30)	Group II (Total=30)	^p-value	Relative effect (Atropine relative to control) Mean±SE	95% CI
Heart rate (beat/min.), Mean±SD					
Baseline (T1)	95.9±3.4	96.5±2.3	0.399	−0.6±0.7	−2.1–0.9
5 minutes after intervention (T2)	114.1±3.5	96.3±2.6	**<0.001***	17.8±0.8	16.2–19.4
5 minutes after spinal (T3)	86.0±2.7	73.1±6.4	**<0.001***	12.9±1.3	10.3–15.4
10 minutes after spinal (T4)	86.8±3.2	70.9±5.5	**<0.001***	15.9±1.2	13.6–18.2
5 minutes after delivery (T5)	117.9±3.4	98.6±2.9	**<0.001***	19.3±0.8	17.7–20.9
At the end (T6)	114.7±3.2	95.4±3.0	**<0.001***	19.3±0.8	17.7–20.9
Bradycardia (n, %)	0 (0.0%)	6 (20.0%)	**§0.024***	NA	NA
Mean Blood Pressure (mmHg), Mean±SD					
Baseline (T1)	92.7±7.1	91.8±6.0	0.626	0.8±1.7	−2.6–4.2
5 minutes after intervention (T2)	92.4±7.0	91.5±5.7	0.615	0.8±1.6	−2.5–4.1
5 minutes after spinal (T3)	80.1±6.0	69.8±5.4	**<0.001***	10.3±1.5	7.4–13.3
10 minutes after spinal (T4)	86.1±6.0	75.1±5.3	**<0.001***	11.1±1.5	8.1–14.0
5 minutes after delivery (T5)	92.3±6.1	84.2±5.5	**<0.001***	8.0±1.5	5.0–11.0
At the end (T6)	95.4±6.3	87.5±5.6	**<0.001***	8.0±1.5	4.9–11.1
Ephedrine consumption, Mean±SD					
Total Ephedrine dose (mg)	5.9±2.0	18.3±5.1	**<0.001***	−12.4±1.0	−14.4–-10.4

^Independent t-test. §Fisher's Exact test. NA: Not applicable. SE: Standard error. CI: Confidence interval. *Significant.

regarding baseline HR, then it became significantly higher in the atropine group from T2 to T6. No significant statistical differences between the study groups regarding baseline and T1 mean blood pressure, and then it became significantly higher in the atropine group from T3 to T6. Total Ephedrine consumption in every patient was significantly lower in the atropine group. (Table 2)(Figure 2)

Also, all patients in group II was given ephedrine in one and repeated doses starting with 3-6 mg per each dose, while 18 patients in group I required ephedrine and only single dose was given.

Electrical cardiometry-derived measures showed no significant statistical differences between the study groups regarding baseline CO, then it became significantly higher in the atropine group after intervention (T2) then the reduction in the CO was significantly less in the atropine group than the control group at (T3 and T4), at (T5 and T6) the increase in the cardiac output was more significant

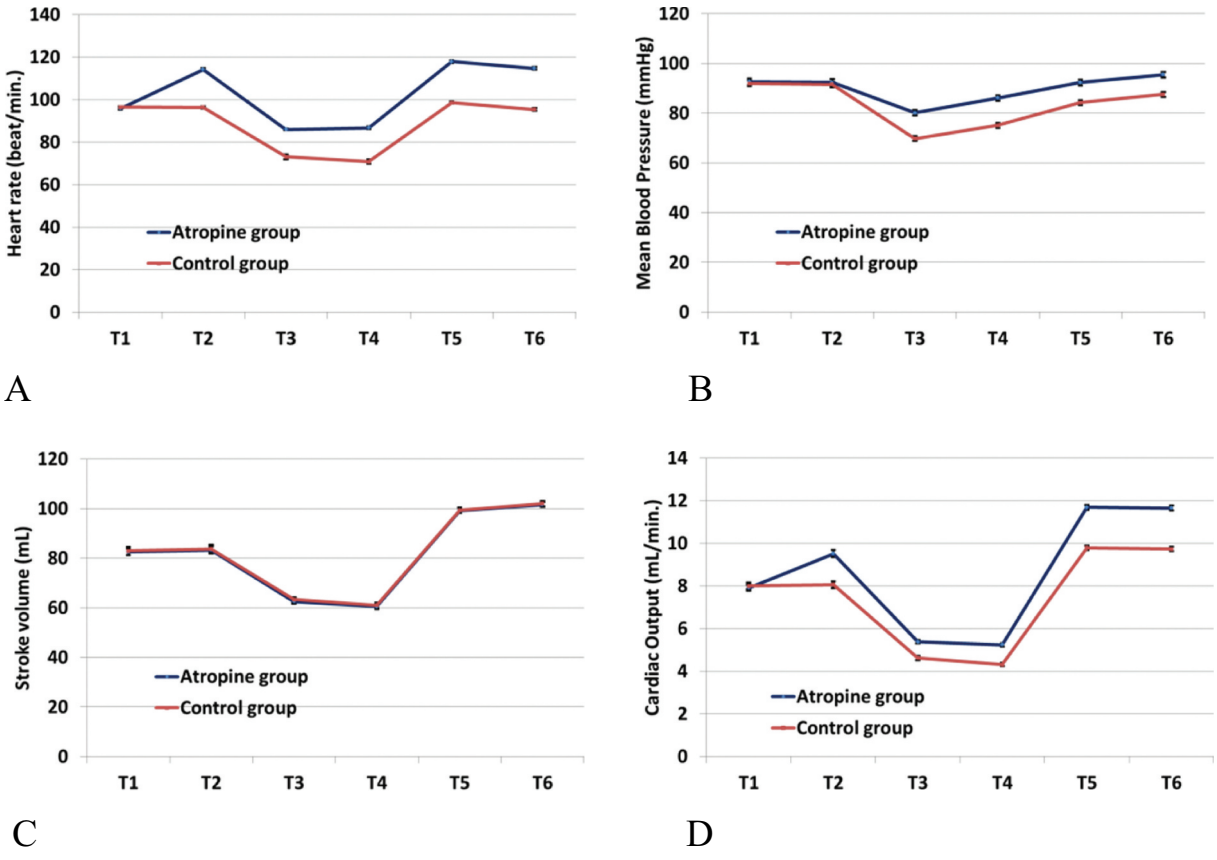

Figure 2. HR, MBP, SV and CO changes during specific times A-HR, B – MBP, C-SV, D-CO.

in the atropine group than the control group. (Table 3)(Figure 2)

There were no significant readings in both groups as regards SV (Figure 2) and SVR at all times of measurement (Table 3).

4. Discussion

The findings of the current study show that pre-spinal intravenous atropine was associated with less reduction in CO after induction of spinal anesthesia till fetal delivery compared with saline. Also, post-spinal HR was higher after administration of atropine without bradycardia, unlike the control group with more incidence of bradycardia. In our study, SV and SVR were similar in both groups; the reduction in MBP was less in the atropine group compared with the control group. Compared to the atropine group, the control group consumed more total intraoperative ephedrine.

These findings support our hypothesis that the use of pre-spinal atropine, which has muscarinic receptor antagonist activity on the heart, can produce positive chronotropic effects with a compensatory increase in CO that could counteract the decrease in CO caused by supine hypotensive syndrome [21].

In parturients who have undergone spinal anesthesia, a decrease in SVR, MBP, and HR are typical hemodynamic changes, particularly if inferior vena cava obstruction is still present [22]. Bradycardia can develop via one of two speculated mechanisms: either blocking sympathetic cardiac accelerator fibers (T1-T5) or decreasing venous return through a reflex mechanism. When the cardiac accelerator sympathetic fibers are blocked, the heart's vagal input takes over and the HR drops sharply. This bradycardia, along with a drop in MBP and SVR, can lead to cardiac arrest very quickly [23].

After spinal anesthesia, hypotension can occur for two different reasons: first, increased blood pooling in capacitance vessels reduces venous return and CO, and second, dilated resistance arterioles reduce SVR. In our study, pre-spinal atropine could prevent the post-spinal reduction in heart rate but it did not prevent hypotension to occur. However, the degree of reduction in MBP was significantly less in the atropine group.

Table 3. Comparison regarding cardiac output, systemic vascular resistance, and stroke volume.

Time	Group I (Total=30)	Group II (Total=30)	^p-value	Relative effect (Atropine relative to control) Mean±SE	95% CI
Stroke volume (mL), Mean±SD					
Baseline (T1)	82.5±7.5	83.0±9.0	0.840	-0.4±2.1	-4.7–3.8
5 minutes after intervention (T2)	83.3±7.7	83.7±9.0	0.854	-0.4±2.2	-4.7–3.9
5 minutes after spinal (T3)	62.6±5.0	63.3±5.8	0.621	-0.7±1.4	-3.5–2.1
10 minutes after spinal (T4)	60.4±5.1	61.0±5.9	0.692	-0.6±1.4	-3.4–2.3
5 minutes after delivery (T5)	99.2±5.5	99.3±5.8	0.927	-0.1±1.5	-3.0–2.8
At the end (T6)	101.6±5.6	102.0±5.7	0.784	-0.4±1.5	-3.3–2.5
Systemic Vascular Resistance (dyn/cm^2), Mean±SD					
Baseline (T1)	816.8±72.8	806.4±64.5	0.563	10.3±17.8	-25.2–45.9
5 minutes after intervention (T2)	819.4±72.9	808.2±65.7	0.534	11.2±17.9	-24.7–47.1
5 minutes after spinal (T3)	665.0±17.3	663.2±15.1	0.664	1.8±4.2	-6.6–10.2
10 minutes after spinal (T4)	684.0±23.1	679.8±24.4	0.500	4.2±6.1	-8.1–16.5
5minutes after delivery (T5)	700.4±25.1	690.5±27.7	0.153	9.9±6.8	-3.8–23.5
At the end (T6)	678.3±25.8	669.3±26.7	0.190	9.0±6.8	-4.6–22.6
Cardiac Output (mL/min.), Mean±SD					
Baseline (T1)	7.9±0.8	8.0±0.8	0.662	-0.1±0.2	-0.5–0.3
5 minutes after intervention (T2)	9.5±0.9	8.1±0.8	**<0.001***	1.4±0.2	1.0–1.9
5 minutes after spinal (T3)	5.4±0.4	4.7±0.4	**<0.001***	0.6±0.1	0.4–0.9
10 minutes after spinal (T4)	5.2±0.4	4.4±0.3	**<0.001***	0.9±0.1	0.7–1.1
5 minutes after delivery (T5)	11.7±0.6	9.8±0.6	**<0.001***	1.9±0.2	1.6–2.2
At the end (T6)	11.6±0.6	9.7±0.6	**<0.001***	1.9±0.2	1.6–2.2

^Independent t-test. SE: Standard error. CI: Confidence interval. *Significant.

There was no significant difference between the two groups regarding APGAR score at 1 and 5 min. also, umbilical artery ABG values were equivalent in both groups. (Table 4).

Table 4. Comparison regarding neonatal APGAR score and umbilical artery ABGs.

Variables	Group I (Total=30)	Group II (Total=30)	^p-value	Relative effect (Atropine relative to control) Mean±SE	95% CI
APGAR 1	7.5±0.9	7.7±0.7	0.343	−0.2±0.2	−0.6–0.2
APGAR 5	9.0±0.6	9.1±0.4	0.445	−0.1±0.1	−0.4–0.2
PH	7.32±0.00	7.32±0.01	0.779	0.00±0.00	0.00–0.00
Base deficit	0.77±0.06	0.78±0.07	0.430	−0.01±0.02	−0.05–0.02
Bicarbonate level	22.8±0.4	23.0±0.4	0.056	−0.2±0.1	−0.4–0.0

^Independent t-test. SE: Standard error. CI: Confidence interval.

Our findings are consistent with an experimental study by Akhtar et al showing that MBP is better preserved with the use of prophylactic pre-spinal atropine 10 μ g/kg compared with saline [24].

Another non-obstetric study by Lim et al. showed the benefit of prophylactic IV atropine given before spinal anesthesia with an increase in HR in a dose-dependent manner and decreased the need to use vasopressors for significant hypotension [25].

Also, another study conducted by Ahn et al revealed that pre-spinal atropine reduces the incidence of bradycardia in patients with dexmedetomidine sedation. Also, MBP showed a significant increase in patients when pre-spinal atropine was given [26].

In our study, we discovered that the MBP reduction following spinal anesthesia was significantly less in the atropine group. Additionally, in the atropine group, significantly less ephedrine was required overall to normalize maternal MBP.

In their investigation of the anticholinergic medication glycopyrrolate for maintaining MBP following spinal anesthesia for cesarean delivery, Ure et al. discovered that pre-spinal glycopyrrolate reduced the need for overall ephedrine [27]. Also, Nagan et al concluded that pre- spinal glycopyrrolate in a dose of 4 mg/kg increased both maternal

HR and CO and decreased phenylephrine dose [28]. In contrast to our findings, Yentis et al. discovered that prophylactic glycopyrrolate administration did not affect the amount of ephedrine required or the severity of hypotension [29]. This discrepancy may be due to different methodology and glycopyrrolate doses.

Electrical cardiometry EC was used in our study to measure CO non-invasively. When compared to the control group, the reduction of CO following pre-spinal atropine administration was considerably less. Both groups experienced similar SV and SVR changes following spinal anesthesia. Atropine's beneficial chronotropic effect was primarily responsible for the atropine group's lesser CO reduction.

According to the current research, the atropine group experienced higher levels of HR and CO in all measurements following spinal anesthesia. According to earlier research, there is a significant correlation between changes in HR and CO [30].

Atropine is the most widely used anticholinergic drug because it is inexpensive and readily available. However, in the atropine group, self-limited negative effects like dry mouth, a feeling of rapid heartbeats, and facial flushing were reported.

In our study, to lessen aortocaval compression, all patients were placed in the supine position with the left uterine tilted 15° and the head raised 10° until delivery. In a prior study, Lee et al. measured CO, SV, and SVR in parturients with four levels of left uterine tilt (0°, 7.5°, 15°, and 90°), demonstrating that a left uterine tilt of at least 15° effectively reduced aortocaval compression [31].

On the other hand, Sonnino et al. revealed that, under continuous hemodynamic monitoring, CO did not significantly change after the removal of the left uterine displacement throughout spinal anesthesia for cesarean delivery [32].

Most research on maternal hemodynamics has focused on MBP. This parameter is utilized frequently because it is trustworthy and simple to reproduce. Non-invasive CO monitoring devices are expensive and hard to come by, and CO measurements are not frequently taken during elective cesarean deliveries. Contrary to blood pressure, which may or may not accurately reflect fetal perfusion, changes in peripheral resistance that occur during pregnancy make CO a better indicator of fetal perfusion [33].

Neonatal outcomes were equivalent in both groups regarding Apgar score at 1 and 5 minutes and umbilical artery blood gases for PH, base deficit, and bicarbonate level.

There may be some limitations to our study. First, the study cannot be generalized to all patients scheduled for cesarean delivery because the exclusion criteria involved a wide range of patients, particularly cardiac patients whose cardiac conditions could not tolerate atropine-induced tachycardia. Secondly, electrical velocimetry was used to measure CO. This method, which has been approved for use in pregnant patients, allows for continuous CO monitoring. The technique's dependence on an estimation of the electrical impedance in the aortic arch, which is established using an algorithm based on the patient's height and weight, is, however, a disadvantage. When detecting the absolute values, this technique carries the risk of bias and errors. We made an effort to get around this by relating derived parameters to baseline readings. Our study's failure to continue past the operative period is an additional limitation.

5. Conclusion

According to our findings, in parturients having an elective cesarean section, pre-spinal atropine in combination with left uterine tilt was more effective than control at preventing maternal bradycardia, minimizing the reduction of CO, and reducing the total amount of intraoperative ephedrine needed.

List of abbreviations

ASA-PS: American Society of Anesthesiologists Physical Status, BMI: Body mass index, CO: Cardiac output, EC: Electrical cardiometry, ECG: Electrocardiography, SV: Stroke volume, SVR: Systemic vascular resistance, MBP: Mean blood pressure NIBP: Non-invasive blood pressure, SpO2 Oxygen saturation, TEB: thoracic electrical bio-impedance.

Authors' contributions

AMA designed the work and reviewed the manuscript. MAE revised the literature, performed the analysis, revised the statistical analysis, and wrote the manuscript. MMA design of the work revised literature and collected the data. AMA followed the patients and collected the data. All authors approved the final version of the manuscript. All authors have contributed intellectually to the manuscript and the manuscript has been read and approved by all the authors. The manuscript has not been published, simultaneously submitted, or accepted for publication elsewhere.

Disclosure statement

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Availability of data and material

The datasets generated and/or analyzed during the current study are not publicly available due [publishing the clinical data about any study conducted in our hospitals and approved by the institutional ethical committee is against the policy of the Faculty of Medicine, Ain Shams University unless there is a reasonable request] but are available from the corresponding author on reasonable request.

Trial registration

Ethical committee approval of Faculty of Medicine, Ain-Shams University (FMASU R 216/2022)), and a Clinical Trials Registry (ID NCT05658380).

References

[1] Antoine C, Young BK. Cesarean section one hundred years 1920-2020: the Good, the Bad and the Ugly. J Perinat Med. 2020;49(1):5–16. doi: 10.1515/jpm-2020-0305

[2] Reynolds F, Seed PT. Anesthesia for caesarean section, and neonatal acid-base status: A meta-analysis. Anesthesia. 2005;60(7):636–653. doi: 10.1111/j.1365-2044.2005.04223.x

[3] Garry M, Davies S. Failure of regional blockade for cesarean section. Int J Obstet Anesth. 2002;11 (1):9–12. doi: 10.1054/ijoa.2001.0903

[4] Bryson GL, Macneil R, Jeyaraj LM, et al. A small dose of spinal bupivacaine for Cesarean delivery does not reduce hypotension but accelerates motor recovery. Can J Anaesth. 2007;54(7):531–537. doi: 10.1007/BF03022316

[5] Knigin D, Avidan A, Weiniger CF. The effect of spinal hypotension and anesthesia-to-delivery time interval on neonatal outcomes in planned cesarean delivery. Am J Obstet Gynecol. 2020;223(5):.e747.1–.e747.13. doi: 10.1016/j.ajog.2020.08.005

[6] Cyna AM, Andrew M, Emmett RS, et al. Techniques for preventing hypotension during spinal anesthesia for cesarean section. Cochrane Database Syst Rev. 2006;4:1–335.CD002251.

[7] Lee A, Ngan Kee WD, Gin T. Prophylactic ephedrine prevents hypotension during spinal anesthesia for cesarean delivery but does not improve neonatal outcome: A quantitative systematic review. Can J Anaesth. 2002;49(6):588–599. doi: 10.1007/BF03017387

[8] Macarthur A, Riley ET. Obstetric anesthesia controversies: Vasopressor choice for post spinal hypotension during cesarean delivery. Int Anesthesiol Clin. 2007;45 (1):115–132. doi: 10.1097/AIA.0b013e31802b8d53

[9] Kobe J, Mishra N, Arya VK, et al. Cardiac output monitoring: Technology and choice. Ann Card Anaesth. 2019;22(1):6–17. doi: 10.4103/aca.ACA_41_18

[10] Kamel YA, Elmoniar MM, Fathi YI, et al. Monitoring haemodynamic changes during transjugular portosystemic shunt insertion with electric cardiometry in sedated and spontaneous breathing patients. A diagnostic test accuracy study. J Anaesthesiol Clin Pharmacol. 2023;39(1):127–133. doi: 10.4103/joacp.joacp_198_21

[11] Funk DJ, Moretti EW, Gan TJ. Minimally invasive cardiac output monitoring in the perioperative setting. Anesthesia & Analgesia. 2009;108(3):887–897. doi: 10.1213/ane.0b013e31818ffd99

[12] Kivalo I, Saarikoski S. Placental transmission of atropine at full-term pregnancy. Br J Anaesth. 1977;49 (10):1017–1021. doi: 10.1093/bja/49.10.1017

[13] Jakubik J, El-Fakahany EE. Current Advances in Allosteric Modulation of Muscarinic receptors. Biomolecules. 2020 18;10(2):325. doi: 10.3390/biom10020325

[14] Sanders M, Servaas S, Slagt C. Accuracy, and precision of non-invasive cardiac output monitoring by electrical cardiometry: a systematic review and meta-analysis. J Clin Monit Comput. 2020;34 (3):433–460. doi: 10.1007/s10877-019-00330-y

[15] Trinkmann F, Berger M, Michels JD, et al. Influence of electrode positioning on accuracy and reproducibility of electrical velocimetry cardiac output measurements. Physiol Meas. 2016;37(9):1422–1435. doi: 10.1088/0967-3334/37/9/1422

[16] Zoremba N, Bickenbach J, Krauss B, et al. Comparison of electrical velocimetry and thermodilution techniques for the measurement of cardiac output. Acta Anaesthesiol Scand. 2007;51(10):1314–1319. doi: 10.1111/j.1399-6576.2007.01445.x

[17] Chaiyakulsil C, Chantra M, Katanyuwong P, et al. Comparison of three non-invasive hemodynamic monitoring methods in critically ill children. PLoS One. 2018 18;13(6):e0199203. doi: 10.1371/journal.pone.0199203

[18] Liu Y, Pian-Smith MC, Leffert LR, et al. Continuous measurement of cardiac output with the electrical velocimetry method in patients under spinal anesthesia for cesarean delivery. J Clin Monit Comput. 2015;29 (5):627–634. doi: 10.1007/s10877-014-9645-8

[19] Wilken M, Oh J, Pinnschmidt HO, et al. Effect of hemodialysis on impedance cardiography (electrical velocimetry) parameters in children. Pediatr Nephrol. 2020;35(4):669–676. doi: 10.1007/s00467-019-04409-1

[20] Hasanin A, Soryal R, Kaddah T, et al. Hemodynamic effects of lateral tilt before and after spinal anesthesia

during cesarean delivery: an observational study. BMC Anesthesiol. 2018 15;18(1):8. doi: 10.1186/s12871-018-0473-0

[21] Lee AJ, Landau R. Aortocaval Compression Syndrome: Time to Revisit Certain Dogmas. Anesth Analg. 2017;125(6):1975–1985. doi: 10.1213/ANE.0000000000002313

[22] Langesæter E, Dyer RA. Maternal hemodynamic changes during spinal anesthesia for cesarean section. Curr Opin Anaesthesiol. 2011;24(3):242–248. doi: 10.1097/ACO.0b013e32834588c5

[23] Caplan RA, Ward RJ, Posner K, et al. Unexpected cardiac arrest during spinal anesthesia: a closed claims analysis of predisposing factors. Anesthesiology. 1988;68(1):5–11. doi: 10.1097/00000542-198801000-00003

[24] Akhtar FM, Pirzada A, Ashraf V. Prevention of hypotension in cesarean delivery under spinal anesthesia; the combination of modified supine wedged position, crystalloid co-hydration, and prophylactic atropine. PAFMJ-Pakistan Armed Forces Med J. 2008;58:429–436.

[25] Lim HH, Ho KM, Choi WY, et al. The use of intravenous atropine after a saline infusion in the prevention of spinal anesthesia-induced hypotension in elderly patients. Anesth Analg. 2000;91(5):1203–1206. doi: 10.1213/00000539-200011000-00029

[26] Ahn EJ, Park JH, Kim HJ, et al. Anticholinergic premedication to prevent bradycardia in combined spinal anesthesia and dexmedetomidine sedation: a randomized, double-blind, placebo-controlled study. J Clin Anesth. 2016;35:13–19. DOI:10.1016/j.jclinane.2016.07.012

[27] Ure D, James KS, McNeill M, et al. Glycopyrrolate reduces nausea during spinal anesthesia for cesarean section without affecting the neonatal outcome. Br J Anaesth. 1999;82(2):277–279. doi: 10.1093/bja/82.2.277

[28] Ngan Kee WD, Lee SW, Khaw KS, et al. Haemodynamic effects of glycopyrrolate pre-treatment before phenylephrine infusion during spinal anaesthesia for caesarean delivery. Int J Obstet Anesth. 2013 Jul;22(3):179–187. doi: 10.1016/j.ijoa.2013.03.008

[29] Yentis SM, Jenkins CS, Lucas DN, et al. The effect of prophylactic glycopyrrolate on maternal hemodynamics following spinal anesthesia for elective cesarean section. Int J Obstet Anesth. 2000;9(3):156–159. doi: 10.1054/ijoa.1999.0376

[30] Dyer RA, Reed AR, van Dyk D, et al. Hemodynamic effects of ephedrine, phenylephrine, and the coadministration of phenylephrine with oxytocin during spinal anesthesia for elective cesarean delivery. Anesthesiology. 2009;111(4):753–765. doi: 10.1097/ALN.0b013e3181b437e0

[31] Lee SW, Khaw KS, Ngan Kee WD, et al. Haemodynamic effects from aortocaval compression at different angles of lateral tilt in non-laboring term pregnant women. Br J Anaesth. 2012;109(6):950–956. doi: 10.1093/bja/aes349

[32] Sonnino C, Frassanito L, Piersanti A, et al. Impact of maternal lateral tilt on cardiac output during cesarean section under spinal anesthesia: a prospective observational study. BMC Anesthesiol. 2022 11;22(1):103. doi: 10.1186/s12871-022-01640-6

[33] Robson SC, Boys RJ, Rodeck C, et al. Maternal and fetal hemodynamic effects of spinal and extradural anesthesia for elective cesarean section. Br J Anaesth. 1992;68(1):54–59. doi: 10.1093/bja/68.1.54