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SCIENTIFIC ARTICLE

The Effect of Levobupivacaine and Bupivacaine on QT, Corrected QT (Qtc), and P Wave Dispersions in Cesarean Section

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Anesthesia, Spinal;
Cesarean Section;
Bupivacaine/ levobupivacaine;
Electrocardiography.

Abstract

Background and Objectives: In our study we aimed to investigate the effect of bupivacaine and levobupivacaine on QT, corrected QT (QTc), and P wave dispersion durations during spinal anesthesia in cesarean section.

Methods: Sixty parturients scheduled for elective cesarean section in ASA I-II risk groups were included in the study. Baseline electrocardiographic (ECG) records of the patients were obtained in the operation room. Heart rate (HR), non-invasive blood pressure (NIBP), peripheral oxygen saturation (SpO_2) and respiration rates (RR) were recorded. Venous cannulation was performed with 18G cannula and fluid preload made with 10 $mL.kg^{-1.}$ Lactated Ringer solution. After fluid preload, second ECG recordings were taken and the patients were randomly separated into two groups. Group B (n = 30) received 10 mg of bupivacaine and Group L (n = 30) received 10 mg of levobupivacaine for spinal anesthesia. ECG recordings were repeated at 1, 5 and 10 minutes after spinal block. HR, NIBP, SpO_2, RR and sensory block levels were also recorded at the same time intervals. At predetermined time intervals of spinal anesthesia, P wave dispersion (Pwd), QT dispersion (QTd), and QTc dispersion (QTcd) durations were measured from ECG records. QT and QTc durations are calculated with Bazzett formula.

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Results: There was no difference between two groups according to block levels, hemodynamic parameters, Pwd, QTd, QTc and QTcd durations.
Conclusion: Bupivacaine and levobupivacaine may be preferred in spinal anesthesia in pregnant patients who have extended Pwd and QTcd preoperatively.

Introduction

Anesthetic agents may display proarrhythmic and antiarrhythmic activity by inducing electrical activity with various mechanisms [1]. Other than the anesthetic agents used, existing heart disease and other concomitant systemic diseases, surgical manipulation, procedures performed on the patient and medication may also cause arrhythmias in the intraoperative stage [2]. Many hormonal and hemodynamic changes that take place during pregnancy may also result in proarrhythmic effects. Pregnancy may trigger the development of new arrhythmia or exacerbate already existing ones. Left axis deviation may be present in ECG as a result of the shift in the position of the heart due to the enlargement of the uterus during pregnancy. Premature atrial and ventricular beats are common [3].

Regional anesthesia in cesarean surgeries has the advantages of allowing the mother to be awake during delivery, not neeeding airway manipulation, keeping mother's airway reflexes, decreasing blood loss, reducing the risk of drug-induced fetal depression, and carrying the need for analgesia over to the postoperative stage. Regional anesthesia is the most common method of anesthesia used in cesarean surgeries in developed countries [4-6]. One of the most commonly used local anesthetics in obstetrics is bupivacaine. A relatively new agent, levobupivacaine is also increasingly being used in obstetric patients [7]. Bupivacaine and levobupivacaine may increase the PR interval and QRS duration and prolong cardiac conduction [8]. Bupivacaine in spinal anesthesia had been reported to induce ECG changes [9,10]. However, levobupivacaine has been shown to be less cardiotoxic [11]. On the other hand, high sympathetic blockage and hemodynamic changes that occur due to regional anesthesia and the inotropic agents used may cause proarrhythmic effects [10]. However, this has not been investigated adequately through research.

In our study, we aimed to examine the effects of bupivacaine and levobupivacaine on QT, QTc and P wave dispersion in pregnant women.

Material and Method

This prospective randomized study was conducted in 2009-2010 at Zonguldak Karaelmas University's School of Medicine Research and Practice Hospital, Department of Anesthesiology and Reanimation, after obtaining the approval of the Hospital Ethics Board (06. 12. 2007, Meeting decision No.: 2007 /09 /04) and patient consents.

Sixty pregnant women aged between 16 and 50, height ≥ 1.60 cm, weight between 60 and 100 kg, placed in the ASA risk group I-II in their preanesthetic evaluation and scheduled for elective cesarean surgery were included in the study. They were randomly allocated into two groups: bupivacaine (Group B) and levobupivacaine (Group L) by using a randomized numbers table.

Exclusion criteria were refusal to participate in the study, the existence of brain tumors, scalded skin syndrome (SSS infection), spinal cord and peripheral nervous system diseases (poliomyelitis, multiple sclerosis, demyelinating diseases), hemorrhagic and hypovolemic shock, severe anemia, increased intracranial pressure, aortic and valvular heart disease, cardiac decompensation, systemic infection (generalized sepsis and bacteremia), local infection (dermal infections in puncture site of spinal needle, etc.), congenital spinal anomalies, scoliosis, post-traumatic vertebral injuries, vertebral colon metastatic lesions, increased abdominal pressure, chronic severe headache, anticoagulant drug use and anatomic difficulties, electrolyte disturbances diabetes mellitus, hypothyroidism, hyperthyroidism, cardiomyopathy, atrial and/or ventricular hypertrophy on ECG, cardiomegaly, valvular disease, cardiac failure or chronic disease, patients with excessive smoking and alcohol consumption and used medication causing QT interval prolongation.

Premedication was not administered to our subjects. Following their admission into the operation room, ECG monitorization was performed and their control ECG (T0) records were taken. Heart rate, noninvasive blood pressure, peripheral oxygen saturation values and respiration rates were recorded.

Vascular access was obtained by using 18G catheter. Preloading was performed with a 10 $mL.kg^{-1}$ Lactated Ringer's solution. Following the preloading, second ECG (T1) records were taken and the patients were placed in lateral decubitus position. Dural puncture was performed from the L2-L3 or L3-L4 interval by using a 27G quincke spinal needle. After the flow of cerebrospinal fluid we administered 10 mg bupivacaine in Group B (n = 30) and 10 mg levobupivacaine in Group L (n = 30) in two minutes (at a speed of 1.5 $mL.min^{-1}$) intrathecal. We brought patients to a supine position after the injection.

Other ECG records were taken 1 (T2), 5 (T3), and 10 (T4) minutes after the block. Bromage scale (BS) scores, heart rate, blood pressure, peripheral oxygen saturation, respiration rate values and sensory block levels with pinprick test were recorded at minutes 1, 5 and 10 of spinal anesthesia and every 5 minutes thereafter.

Additional fluid loading and stabilization of hemodynamics with 5 mg ephedrine was planned for cases where blood pressure values fell 20% below control values; 0.5 mg IV atropine was planned for cases where heart rate fell below 55 $beats.min^{-1}$; and 10 mg IV metoclopramide was planned for

patients with nausea and vomiting. Standard 2 L.min^{-1} oxygen was delivered to all patients via nasal cannula. Sensory and motor block levels were identified and noted until 10 min and surgery started later.

Electrocardiography

Standard 12 derivation ECG recordings obtained with a paper speed of 25 mm.sec^{-1} and a deflection of 10 mm.mV^{-1} of patients participating in the study was analyzed (Hewlett Packard®, Pagewriter 300pı). We calculated heart rate using mean RR time.

Analysis of QT dispersion

The QT interval was defined as between the beginning of QRS complex and the point where T waves descend onto the TP isoelectric line. When a U wave interrupted the T wave before returning to baseline, the QT interval was measured to the nadir of the curve between the T and U waves [1]. The corrected QT interval (QTc) was calculated using the Bazett formula; QTc (ms) = QT measured/√RR (where RR is the RR interval). The QTd value was determined as the difference between the longest and shortest QT intervals in the 12 ECG leads. The QTc dispersion (QTcd) duration according to heart rate was identified with the Bazett formula; QTcd (ms) = QTd measured/√RR [1].

Analysis of P-wave dispersion

The beginning of P-wave was defined as positive deflection from the isoelectric line, and the end point when the positive deflection returned to the isoelectric line [1]. Derivations where the beginning and end of P-waves were not obvious were excluded from the study. Pwd was the difference between the longest and shortest P-wave durations [1].

Subjects who had less than 9 derivations assessed on the ECG were excluded from the study. All ECG measurements were evaluated three times by two experts who were not aware of which group the subject belonged to [1].

Statistical Analysis

Statistical analyses were performed by using the Statistical Package for the Social Sciences (SPSS) 13.0 (SPSS Inc., Chicago, IL, USA). Descriptive statistics included arithmetic mean ± standard deviation for numerical data, and numbers and percentages for categorical data. We used the Kolmogorov-Smirnov test to examine compatibility between measured variables and normal distribution. We used a significance test when parametric test assumptions were met for intergroup differences between the measured variables, and the Mann-Whitney U test when they were not. We analyzed the differences between groups for categorical variables by using Chi-Square analysis. For measured variables, we analyzed differences between groups and time-dependent changes by two-way analysis of variance in repeated measurements. When we found a difference as a result of two-way analysis of variance in repeated measurements, comparisons between pairs were made with the Bonferroni test. For ordinary variables, differences between groups and time-dependent changes were analyzed by using analysis of variance in repeated measurements. The results were evaluated at 95% confidence interval and $p < 0.05$ was accepted as statistically significant difference.

Results

Our subjects were allocated randomly into two groups. Groups were similar in terms of age, body mass, height and *American Society of Anesthesiologists* risk class (Table 1). When we compared the groups with respect to sensory block levels, we found no statistically significant difference between groups at any time ($p > 0.05$) (Table 2).

Compared with respect to systolic and diastolic arterial blood pressure values at all times, Groups B and L did not display a statistically significant difference ($p > 0.05$). When we compared T3 and T4 times against control values, we saw a significant decrease in systolic and diastolic arterial blood pressure values in both Groups B and L ($p < 0.05$) (Table 3). When the mean heart rates in the two groups were compared, a statistically significant difference did not exist between or within groups at any time ($p > 0.05$) (Table 3).

When the groups were compared with regard to maximum P-wave values, no statistically significant difference was found between the values measured in Groups B and L at any time ($p = 0.146$). When T4 time was compared with the control value, a significant decrease was detected in the maximum P-wave value of Group L ($p = 0.015$) (Table 3).

Table 1 Demographic and anthropometric data of groups.

	Group B (n = 30)	Group L (n = 30)	p
Age (year)	28.0 ± 5.34	26.8 ± 4.2	0.323
Weight (kg)	79.1 ± 11.35	77.07 ± 9.0	0.446
Height (cm)	163.4 ± 2.5	163.8 ± 3.2	0.531
	n (%)	n (%)	
ASA			
I	28 (%93.3)	26 (%80)	
II	2 (%6.7)	4 (%20)	0.254

ASA: American Society of Anesthesiologists.

Table 2 Sensory block levels of groups.

Time	Group B (n = 30)	Group L (n = 30)	p
T0	-	-	-
T1	-	-	-
T2	L1 (Th10-5)	L1 (Th11-L5)	0.150
T3	Th8 (Th4- T12)	Th8 (Th6-Th11)	0.106
T4	Th4 (Th2-Th6)	Th4 (Th3-Th5)	0.327

T0: Control, T1: After the preloading, T2: 1 minute after spinal anesthesia, T3: 5 minutes after spinal anesthesia, T4: 10 minutes after spinal anesthesia.

Table 3 Hemodynamic and Electrocardiographic data of groups.

	T0	T1	T2	T3	T4
HR (beats.min^{-1})					
Group B (n = 30)	90.6 ± 13.7	91.5 ± 15.9	95.6 ± 15.7	91.9 ± 21.6	96.0 ± 23.7
Group L (n = 30)	90.6 ± 12.9	90.1 ± 13.3	92.2 ± 20.0	88.2 ± 17.8	87.6 ± 18.5
SAP (mm Hg)					
Group B (n = 30)	122.6 ± 13.5	128.7 ± 14.4	123.2 ± 15.2	104.6 ± 21.4*	110.4 ± 21.7*
Group L (n = 30)	127.8 ± 9.1	131.5 ± 10.9	121.1 ± 15.3	111.4 ± 18.6†	113.1 ± 20.8†
DAP (mm Hg)					
Group B (n = 30)	75.1 ± 11.2	79.0 ± 9.2	72.9 ± 14.2	58.2 ± 16.1*	64.3 ± 15.1*
Group L (n = 30)	79.6 ± 8.2	80.3 ± 7.5	72.9 ± 13.0	58.2 ± 16.2†	64.4 ± 15.7†
Max. P-wave duration (ms)					
Group B (n = 30)	99.0 ± 16.5	96.0 ± 18.0	96.3 ± 15.2	96.3 ± 13.3	93.3 ± 13.5
Group L (n = 30)	99.7 ± 14.5	94.6 ± 15.0	92.7 ± 13.1	92.0 ± 14.7	90.3 ± 11.6†
Min P-wave duration (ms)					
Group B (n = 30)	42.9 ± 16.0	42.3 ± 13.8	42.7 ± 13.9	41.3 ± 12.8	42.7 ± 11.4
Group L (n = 30)	38.3 ± 13.2	35.6 ± 12.2	33.3 ± 9.2	35.7 ± 10.4	34.3 ± 11.0
P-wave dispersion (ms)					
Group B (n = 30)	56.3 ± 19.2	53.7 ± 20.4	53.7 ± 17.7	55.7 ± 16.1	52.7 ± 17.2
Group L (n = 30)	62.0 ± 16.7	59.0 ± 19.0	59.3 ± 11.1	56.3 ± 13.0	54.3 ± 14.5
QT interval (ms)					
Group B (n = 30)	352.7 ± 21.4	348.0 ± 20.2	339.3 ± 22.4	340.0 ± 32.1*	346.0 ± 30.1*
Group L (n = 30)	341.0 ± 29.0	344.1±28.5	339.3±23.4	340.1±26.3	350.7±36.8
QTc interval (ms)[a]					
Group B (n = 30)	428.0 ± 26.0	419.0±19.1	422.7±23.5	422.1±30.6	422.8±29.3
Group L (n = 30)	422.1 ± 19.1	421.7±25.1	418.1±23.0	422.4±24.2	420.6±32.8
QTd interval (ms)					
Group B (n = 30)	55.0 ± 10.7	49.3±13.8	49.0±11.2	53.3±15.8	55.0±11.9
Group L (n = 30)	55.3 ± 19.7	55.0±19.6	54.6±16.7	57.0±15.5	56.0±19.5
QTcd interval (ms)[b]					
Group B (n = 30)	67.2 ± 16.1	64.7±17.3	71.2±36.1	65.2±35.0	69.5±17.5
Group L (n = 30)	66.7 ± 26.7	66.0±27.8	67.7±21.0	68.0±18.0	70.3±23.0

T0: Control, T1: After the preloading, T2: 1 minute after spinal anesthesia, T3: 5 minutes after spinal anesthesia, T4: 10 minutes after spinal anesthesia; Max: maximum; Min: minimum; HR: Heart Rate; SAP: Systolic Arterial Pressure; DAP: Diastolic Arterial Pressure
Values are mean ± SD.
* $p < 0.05$ (compared control value in Group B); † $p < 0.05$ (compared control value in Group L); *a:* QTc reflects the heart rate adjusted QT interval by using Bazett's formula; b: QTcd reflects the heart rate adjusted QTd interval by using Bazett's formula.

When the two groups were compared with respect to minimum P-wave values, no statistically significant difference was seen between or within groups at any time ($p > 0.05$) (Table 3).

Similarly, when we studied the groups' P-wave dispersion values, no statistically significant difference was found between or within groups at any time ($p > 0.05$) (Table 3).

When we compared the groups with respect to QT values, we did not detect a statistically significant difference between Groups B and L at different measurements ($p > 0.05$). When we compared T3 and T4 times to the control value within Group B, we found a decrease in the QT value. This decrease was statistically significant ($p < 0.05$). When we compared the values obtained at all times in Group L to control values, no statistically significant difference existed in the QT value ($p > 0.05$) (Table 3).

When we compared the groups with respect to QTd, QTc and QTcd values, we found a statistically significant difference both between and within groups at all times ($p > 0.05$) (Table 3).

Ephedrine consumption was 13.3 ± 14.7 mg in Group B and 6.5 ± 6.2 mg in Group L. No statistically significant difference existed between the total ephedrine, atropine and metoclopramide consumption in the two groups ($p > 0.05$).

Discussion

In our study we examined the effects of using bupivacaine and levobupivacaine in spinal anesthesia for cesarean surgeries on P-wave in ECG, QT, and QTc dispersion times, and we found no significant difference between the p wave, QT and QTc dispersions of the levobupivacaine and bupivacaine groups.

In pregnancy, a dramatic hormonal and hemodynamic change can be observed in the organism, which may lead to a proarrhythmic effect. Pregnancy may trigger the development of new arrhythmia or exacerbate existing ones. Hemodynamic changes, increased cardiac output and circulating blood volume cause an arrhythmogenic effect by increasing end diastolic volume and myocardial regression. In addition, elevated catecholamine levels also cause the development of arrhythmia. Arrhythmia and tachycardia are very common occurrences among pregnant women. Heart rate may increase by 20% during pregnancy and left axis deviation shows up in ECG due to the enlargement of the uterus. Premature atrial and ventricular beats are also very common [3].

Even though spinal block may be a safe anesthesia technique, severe tachycardia, cardiac arrest and other arrhythmia are reported during spinal anesthesia practices. In an ASA study of closed claims project, sudden cardiac arrest was reported during spinal anesthesia performed on 14 hemodynamically stable, young and healthy patients. In another study of 952 patients who received spinal anesthesia, risk factors for bradycardia and hypotension during spinal anesthesia were defined as being female, a control heart rate below 60, use of beta-blockers, and a sensory block above T5 [12].

In a multiple center study conducted on over 17,000 patients, Youngs PJ et al. [13] studied the effects of spinal anesthesia that cause arrhythmia. Of their subjects, 70.2% had tachycardia, bradycardia or arrhythmia. Most of these were spontaneously recovering minor arrhythmia. Sinusal arrhythmia was found in 30.3% of the patients, premature beats in 27.2%, and bradycardia in 13.8%.

The incidence of arrhythmia and hypotension among pregnant women who receive spinal anesthesia in cesarean surgeries is greater than expected. Most of these incidents are spontaneous and temporary. They may occur suddenly and require urgent treatment. In a study of 254 healthy pregnant women who underwent cesarean surgery, Shen CL et al. [14] administered 10 mg bupivacaine + 0.2 mg morphine for spinal anesthesia, and observed 1st degree atrioventricular block in 9 patients (3.5%), 2nd degree atrioventricular block in 9 (3.5%), severe bradycardia in 17 (heart rate < 50 beat.min^{-1}) (6.7%) and multiple ventricular premature complexes in 3. During cesarean surgeries, one should take care about arrhythmia and handle monitoring attentively. Among our subjects, we observed premature ventricular beats in one pregnant woman in the bupivacaine group and in two women in the levobupivacaine group.

Many clinical studies have shown both bupivacaine and levobupivacaine to have equal effectiveness in spinal anesthesia over doses of 10 mg [15]. Alley EA et al. [15] used 4 mg, 8 mg and 12 mg hyperbaric bupivacaine and hyperbaric levobupivacaine for spinal anesthesia in 18 healthy volunteers and observed similar sensory and motor block levels in the two groups. Similarly, in our study we observed no significant difference between the sensory block levels, systolic blood pressure, diastolic blood pressure, heart rate changes, and amounts of atropine used in the two groups until minute 10.

In spinal anesthesia, cardiovascular effects are related to the sympathetic blockage that develops with spinal anesthesia rather than the systemic absorption of local anesthetics [9].

However, there are a limited number of studies that explore the effects of spinal anesthesia on QT and QTc intervals, the results of which seem to vary [10,16,17]. In 20 adult males who did not receive premedication, Owczuk et al. [10] performed spinal anesthesia with 3 or 4 mL 5% hyperbaric bupivacaine and calculated the QTc interval from the ECG records taken at minutes one, three, five and 15, and observed a significantly prolonged QTc interval starting from minute 1 after spinal anesthesia induction and in later measurements. With mean values, they detected no significant difference at the onset of spinal block between QTc intervals and heart rate. However, starting from min three, a significant decrease was observed in systolic, diastolic and mean blood pressure as compared to control values. The QTc interval exceeded 440 msec in a total of eight patients, and QTc interval > 440 msec occurred once in two patients, twice in five patients, and three times in two patients. Prolonged QTc interval was seen at min one following spinal anesthesia in five patients, after mins three and five in three patients, and after min 15 in two patients. QTc interval > 500 msec occurred only in one patient, but severe arrhythmia or conduction did not. Ventricular ectopic beats were observed in one patient who had normal QTc interval.

At the same time, many case studies report that spinal anesthesia can be used safely in pregnant women with prolonged QT syndrome [16-18]. No complications were reported

during combined spinal-epidural anesthesia with 9 mg bupivacaine and 100 mg lidocaine in a pregnant woman with asymptomatic idiopathic prolonged QT interval syndrome who underwent elective cesarean surgery. Kameyam et al. [19] also emphasized that spinal anesthesia with bupivacaine is a safe option for patients with asymptomatic idiopathic prolonged QT syndrome.

Şen et al. [9] studied the effects of spinal anesthesia on the QTc interval of preeclamptic patients. They reported that even though preeclamptic patients have higher QTc intervals than the control group prior to spinal anesthesia, their interval shortens following spinal anesthesia while no change occurs in that of patients without preeclampsia. They also wrote that the sympathetic blockage effect of spinal anesthesia might normalize patients with prolonged QTc interval.

In our study, we found that both levobupivacaine and bupivacaine for spinal anesthesia shortens the QT intervals of patients, but no statistically significant difference existed between the groups. Even though a significant difference did not exist among the subjects in the levobupivacaine group, the QT interval of patients who received bupivacaine for spinal anesthesia were significantly shorter than control values at minutes five and 10 after spinal anesthesia. We are of the opinion that this shortening in the QTc interval may be related to the sympathetic suppression caused by spinal anesthesia. Supporting findings were observed in a previous study of Owczuk et al. [20], where they had compared lumbar and thoracic epidural block by using isobaric bupivacaine and found out that QTcd was significantly shorter in patients who received thoracic epidural block, suggesting the role of higher symphathetic block level.

Pregnancy may also affect QTd and QTcd times. Lechmanova et al. [21] compared QTds of 37 healthy pregnant women in their late pregnancy and postnatal days, and found out that parturients had significantly longer QTd in late pregnancy. In our study, we found that the QTd and QTcd interval of patients who received spinal anesthesia with levobupivacaine and bupivacaine were prolonged after spinal anesthesia, but a statistical difference did not exist within groups or in comparison with control values.

Anesthetic substances may affect P wave dispersion (Pwd). The general anesthetic sevoflurane has been reported to prolong Pwd, desflurane to have no effect on it, and propofol to shorten it [22-24]. At the same time, our literature survey found no study that investigated the effects of local or spinal anesthesia on Pwd. However, in one study that evaluated Pwd changes in pregnant women, Pwd was reported to prolong due to the shortening of minimum P wave time [25]. In our study, we found no significant difference between the Pwd, Pmax and Pmin times of the groups following spinal anesthesia with either local anesthetic.

Our study has several limitations. First, P wave, QT, QTc dispersions were manually calculated from the ECG record. Even though there are many studies stating that these parameters can be measured manually with a minimal room for error [26,27], others mention about the reliability of this type of measurement [26,28]. The second limitation of our study is that it has lasted until 10 minutes after spinal anesthesia application and ECG records were taken with five-minute intervals. However, there are studies that show in ECG that changes can occur in P-wave, QT times after minute 10 [10,13]. We are of the opinion that using continuous Holter monitorization in addition to intermittent ECG records in future studies may help longer-term and more detailed identification of perioperative arrhythmia and ECG changes. Third, effects of fluid therapy and vasopressors on ECG records cannot be excluded in this study, because one has to administer these therapies due to ethical reasons.

In conclusion, our double blind randomized prospective study which compared the electrocardiographic effects of spinal anesthesia by using 10 mg of bupivacaine and 10 mg of levobupivacaine in cesarean surgeries showed that P wave, QT and corrected QT dispersion values were not affected. For this reason, levobupivacaine and bupivacaine may be preferred for spinal anesthesia of the pregnant women with prolonged P wave and QT dispersion determined in the preoperative stage.

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