Effect of Combined Spinal-epidural Versus General Anesthesia on the Hemodynamic, Respiratory and Metabolic Changes Associated with Tourniquet Deflation

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Background: Tourniquet deflation is associated with profound hemodynamic, respiratory and other organ system derangements. These changes occur with both general and regional anesthesia. Since general and regional anesthesia impact the cardiovascular systems in different ways, we found it worthwhile to evaluate if the magnitude of the systemic effects of tourniquet deflation is differently affected by combined spinal-epidural anesthesia (CSEA) vs. general anesthesia (GA). We hypothesized that during total knee replacement surgery (TKR), CSEA when compared to GA produces less pronounced hemodynamic, respiratory and metabolic changes after tourniquet deflation.

Methods: In a parallel group clinical trial, 49 patients undergoing primary (no redo cases included), cemented TKR surgeries were randomly allocated to either CSEA (n=29) or GA (n=20).

Mean arterial pressure (MAP) and heart rate (HR) were recorded before anesthesia, 10 min before release of a tourniquet and 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 15 and 30 min after tourniquet release. Arterial pH, standard bicarbonate, base excess, lactate, PaCO₂ and PaO₂ were measured 10 min before and 10 and 30 min after tourniquet deflation.

Results: There was no difference in baseline MAP values. MAP with CSEA was higher vs. GA, 5, 7, 8, 9 and 15 min after tourniquet deflation – 89 mmHg (95% CI 85–93) vs. 82 mmHg (95% CI 78–87) respectively (p < 0.05). At 30 min after tourniquet deflation, the MAP was lower with CSEA vs. GA (p <0.05). There was an association between anesthesia type and MAP (F for Test of Between-Subjects Effects = 5.6; df = 1; p < 0.02). At 30 min after tourniquet deflation, HR with CSEA was significant lower compared to GA group. There were no associations between type of anesthesia and pH, standard bicarbonate, base deficit, lactate, PaO₂ and PaCO₂.

Conclusions: Type of anesthesia had a significant, dynamic effect on MAP following tourniquet deflation, but not on the respiratory or metabolic changes.

Keywords: tourniquet, hemodynamic, respiratory, metabolic changes, combined spinal-epidural anesthesia, general anesthesia

Introduction

Tourniquet deflation during limb surgery can produce significant metabolic, hemodynamic, respiratory and other organ system changes [1-6]. The severity of injury is closely related to the duration of tourniquet inflation [2,7-11] and the concurrent illnesses of the patients [7-11]. Tourniquet shock and acute renal failure following tourniquet deflation is a recognized clinical entity [1,2]. A decrease in mean arterial pressure (MAP) below 70 mmHg may reduce the blood flow to the capillaries and venous circulation, eventually leading in extreme situations to irreversible organ/tissue damages [2].

It has been estimated that during general anesthesia MAP significantly decreased after tourniquet deflation and remained in the lower range about 15 min [9,12-13]. Changes in arterial pH, PaO₂, PaCO₂, lactate, potassium and bicarbonate have been observed, with a return to the normal range 30 min after tourniquet deflation [9,10]. Hypotension, lactic acidosis and hypercapnia may be life threatening in high-risk patients [14,15]. We are not aware of published studies that compared the magnitude of tourniquet – deflation effects with combined spinal-epidural anesthesia (CSEA) versus general anesthesia (GA). The purpose of this study was to compare the hemodynamic, respiratory and metabolic effects of tourniquet deflation in patients undergoing total knee replacement surgery under general vs. CSEA.

Based on clinical observation we hypothesized that during total knee replacement surgery (TKR), CSEA when compared to GA produces less severe hemodynamic, respiratory and metabolic changes after tourniquet deflation.

Material and methods

Following our Institutional Review Board’s approval and patient’s informed consent, 49 patients have been recruited to this randomized, parallel group clinical trial. Patients aged 40-80 yrs, scheduled for elective, unilateral, primary TKR surgery at Wolfson Medical Center were randomly allocated to either GA group or CSEA group. Randomization was performed with the opaque, closed envelope technique.
Excluded from the study were patients with contraindications to CSEA such as those in whom enoxaparin (clexane) was administered within 12 hours before anesthesia, those receiving enoxaparin twice daily with the last dose given less than 24 hours before surgery, or those receiving a dose higher than 1.5 mg/kg body weight/day. Patients receiving other anticoagulants except for aspirin (< 300 mg daily dose) and those with abnormal coagulation tests (PT, PTT, INR and platelets count) were also excluded. Also excluded were patients with a history of congestive heart failure or a documented ejection fraction < 40% and patients belonging to ASA class > 3.

Protocol

Chronic medication was continued until surgery. Patients were premedicated with 0.25 mg sublingual brotizolam, 30 min before anesthesia. Routine ASA monitoring was applied. Blood pressure was measured directly through an intraarterial cannula.

Before induction of anesthesia, patients from both groups received a bolus of 500 ml warm (37°C) lactated Ringer's solution, over 20 min. This was followed by a continuous infusion at a rate of 10 ml/kg/hr. Additionally a bolus of 200 ml was rapidly infused just before tourniquet deflation. CSEA was performed at L3-L4 or L2-L3. According to the protocol, the first priority was L3-L4. CSEA was performed with an 18-G epidural needle, 20-G multipor catheter and 27-G pencil point spinal needle (B.Braun, Melsungen AG, Germany). Two ml of 0.5% isobaric bupivacaine were injected into the CSF 15 min after the subarachnoid injection, an additional dose of 5 ml of 0.5% bupivacaine was given through the epidural catheter after confirming its correct position. We reduced by one third the local anesthetic dose injected into the spinal space with the CSEA technique, compared to the dose proposed in a previous study [16], therefore, only 10 mg instead of 15 mg of isobaric bupivacaine was administered.

In the GA group, following 3 min of preoxygenation, anesthesia was induced with 1 mg midazolam, 1 µg/kg fentanyl, 1-2 mg/kg propofol (‘sleep’ dose) and 0.6 mg/kg rocuronium or 1 mg/kg succinylcholine (in patients at risk for aspiration of gastric content). Anesthesia was maintained with N₂O in O₂ – 70:30%, isoflurane 0.5-1.0 MAC, aliquots of fentanyl 50 mcg when deemed necessary and rocuronium 10-20 mg in repeated doses to enable a muscle relaxation degree of one twitch out of the TOF. At the end of surgery the neuromuscular blockade was reversed with neostigmine and the patient’s trachea was extubated on the table.

A decrease in mean arterial pressure (MAP) below 70 mmHg was treated with a bolus of 200 ml lactated Ringer's solution in both groups. With a decrease in MAP below 70 mmHg, for > 5 min, 5 mg of iv ephedrine was administered. Bradycardia < 50 beats/min was treated with 0.5 mg iv atropine. Forced-air and fluid warmers were used from the start of surgery. The radial artery was cannulated before anesthesia induction in both groups. The tourniquet cuff was inflated to a pressure equal to the patient’s preoperative systolic blood pressure plus 100 mmHg. Cemented prosthesis was implanted in each patient.

Measurements

In the CSEA group, the height of the sensory block was assessed using the pin-prick test every 5 min during the first 30 min of anesthesia and afterwards, every 15 min up to the end of surgery. The level of autonomic block was assessed with a pad soaked in alcohol at the same time points. With two sensory segment regression, the CSEA patients received 5 ml of 0.5% bupivacaine through the epidural catheter. Patients who complained of discomfort from tourniquet received iv fentanyl 50 µg and midazolam 1 mg.

Statistical analysis

Statistical analysis of the results was done using SPSS-PC program.

Variables (age, BMI, duration of surgery and tourniquet placement, pH values, standard bicarbonate, base excess, lactate, PaO₂, PaCO₂ and MAP at different time points) are presented as standard deviation (SD) or confidence interval 95% (CI).

The difference between study groups in regard to pH, base excess, standard bicarbonate, lactate, PaO₂, PaCO₂, MAP and HR, indexes of fluid input and output were measured by Mann-Whitney Test. The difference between study groups considering gender and ASA class was evaluated by Chi Square test.

Changes in pH, standard bicarbonate, base excess, lactate, PaO₂, PaCO₂, MAP and HR were analyzed by repeated measures one-way analysis of variance. Values less than 5% (P < 0.05) were considered as statistically significant.

Results

Forty-nine consecutive patients were recruited to the study. Demographic data are summarized in Table I. No statistically significant differences between the groups were found in regard to age, gender, ASA class, BMI, tourniquet time and fluid input/output (Table I). However, duration of surgery in GA group was significantly shorter compared to CSEA group.

Table 2 shows the overall values of MAP, HR, pH, bicarbonate, base excess, lactate, PaO₂ and PaCO₂ measured before anesthesia, before tourniquet deflation and at all measurement time points after tourniquet deflation. No difference was found between the groups in the MAP before anesthesia. For each repeated measurement of MAP during the study period, Levene’s Test of Equality of Error Variances was not statistically significant. MAP was different between the groups in relation to different stages of tourniquet placement (F for Test of Between-Subjects Effects = 5.6; df = 1; p < 0.02). At minutes 5, 7, 8, 9, and 15, MAP in the CSEA group was significantly higher compa-
Table I. Demographic data. Results are expressed as mean±SD or absolute numbers (n)(percentage); *p < 0.05

<table>
<thead>
<tr>
<th>Variable</th>
<th>GA (n = 20)</th>
<th>CSE (n = 29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender – females, n(%)</td>
<td>15 (41.7)</td>
<td>2 (58.3)</td>
</tr>
<tr>
<td>Age, years</td>
<td>66 (9)</td>
<td>71 (8)</td>
</tr>
<tr>
<td>ASA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I, n(%)</td>
<td>6 (54.5)</td>
<td>5 (45.5)</td>
</tr>
<tr>
<td>II, n(%)</td>
<td>13 (54.5)</td>
<td>20 (60.6)</td>
</tr>
<tr>
<td>III, n(%)</td>
<td>1 (25.0)</td>
<td>3 (75.0)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>29.7 (3.8)</td>
<td>29.8 (3.7)</td>
</tr>
<tr>
<td>Tourniquet time, min (SD)</td>
<td>73 (17)</td>
<td>81 (22)</td>
</tr>
<tr>
<td>Operation time, min</td>
<td>91 (16)*</td>
<td>106 (30)</td>
</tr>
<tr>
<td>Intraoperative urine output - mL/h</td>
<td>130 (80)</td>
<td>170 (90)</td>
</tr>
<tr>
<td>Intraoperative total fluid input - mL</td>
<td>1500 (300)</td>
<td>1600 (300)</td>
</tr>
<tr>
<td>Intraoperative blood loss - mL</td>
<td>90 (40)</td>
<td>80 (50)</td>
</tr>
<tr>
<td>Postoperative 4 hours total fluid input - mL</td>
<td>1300 (400)</td>
<td>1300 (500)</td>
</tr>
<tr>
<td>Postoperative blood loss - mL</td>
<td>130 (60)</td>
<td>130 (60)</td>
</tr>
</tbody>
</table>

Table II. Hemodynamic and metabolic measurements. Results are expressed as average (range; 95% confidence interval)

<table>
<thead>
<tr>
<th>Variable</th>
<th>GA (n = 20)</th>
<th>CSE (n = 29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean arterial pressure – mmHg</td>
<td>82 (78-87)</td>
<td>89 (85-93)</td>
</tr>
<tr>
<td>Heart rate – beats/min</td>
<td>65 (62-68)</td>
<td>62 (59-64)</td>
</tr>
<tr>
<td>pH</td>
<td>7.41 (7.36-7.46)</td>
<td>7.39 (7.34-7.43)</td>
</tr>
<tr>
<td>Standard bicarbonate – mEq/L</td>
<td>26 (25-27)</td>
<td>26 (26-27)</td>
</tr>
<tr>
<td>Base excess – mEq/L</td>
<td>0.9 (0-2)</td>
<td>1.3 (0.8-1.9)</td>
</tr>
<tr>
<td>Lactate – mmol/L</td>
<td>1.3 (1.2-1.5)</td>
<td>1.3 (1.2-1.5)</td>
</tr>
<tr>
<td>PaO₂ - mmHg</td>
<td>144 (125-162)</td>
<td>144 (129-160)</td>
</tr>
<tr>
<td>PaCO₂ - mmHg</td>
<td>41 (38-43)</td>
<td>41 (39-43)</td>
</tr>
</tbody>
</table>

Discussions

The purpose of this study was to evaluate the effect of two types of anesthesia CSEA and GA on the hemodynamic, respiratory and metabolic changes associated with tourniquet release in patients undergoing primary TKR surgery. We hypothesized that during TKR surgery the hemodynamic, respiratory and metabolic changes caused by tourniquet release are less pronounced with CSEA as compared to GA.

The results of the study only partially confirmed our hypothesis in that patients operated under CSEA had higher MAP after tourniquet release compared to the GA patients group. Heart rate was lower with CSEA, 30 min after release.

However there were no differences between the two types of anesthesia in regard to the respiratory and metabolic changes.

In both groups, MAP decreased during the 5 to 15 minutes interval after tourniquet release and then increased until min 30.

The main finding of this study indicates that, despite similar MAP ten min prior tourniquet release, MAP was lower with GA group after tourniquet release. However there were no MAP drops in either of the groups to values< 70 mmHg necessitating pharmacological intervention.
The decrease in BP after tourniquet release was explained by a decrease in peripheral resistance related to ischemia-associated hyperemia and return of blood volume to the limb after tourniquet release [9].

A significant decrease in BP after tourniquet release may occur with both epidural and epidural-general anesthesia, both in children and in adult patients [9, 17-19]. These changes are more pronounced with a tourniquet time > 75 min [18].

Townsend HS et al [9] described the effect of tourniquet release on blood pressure with the peak of blood pressure decrease occurring at 3 min return to normal 15 min after the release.

When comparing the tourniquet release related blood pressure changes in CSEA vs. GA, we presume that the sharper decrease in MAP in our GA group could be explained by the fact that blood vessels in patients with CSEA were already dilated prior to the tourniquet release, as a result of the sympatholysis produced by CSEA. Therefore, in the CSEA group the hemodynamic effects of tourniquet release were less dramatic.

This possible mechanism could also explain the significant difference in heart rate at min 30, indicating a less sustained increase in heart rate with CSEA vs. GA, as a response to the decrease in blood pressure.

It seems that heart rate does not change significantly after tourniquet release [18]. However, in our CSEA group HR was significantly lower at min 30 after release compared to that in GA patients presumably attributed to sympatholysis and reflecting a less sustained increase in heart rate with CSEA vs. GA, as a response to the decrease in blood pressure. Several authors reported a decrease in pH, standard bicarbonate and BE and increase in lactate values after the tourniquet release, presumably reflecting limb ischemia. These findings were recorded in both adult and pediatric patients undergoing epidural or general anesthesia. The pH values peaked at 3-5 min and returned to those before tourniquet release at min 30 after release [9,10,19,21].

The current study did not reveal any correlation between type of anesthesia and tourniquet related changes in pH, standard bicarbonate and BE.

Modig et al reported about decrease of PaO₂ values immediately after tourniquet release among 15 patients undergoing epidural anesthesia [19].

After tourniquet release, the most significant decrease was found at min 1 and 2 while at min 30 the PaO₂ returned to pre-tourniquet release values.

In the current study there was no correlation between the type of anesthesia and the perioperative changes of PaO₂ values. Modig et al [19] also found an increase in PaCO₂ immediately after tourniquet reaching the peak level the peak level at min 3. The return to the pre-released values occurred 30 min after the release [9].

In the current study there was no correlation between the type of anesthesia and the perioperative changes of PaCO₂ values.

**Conclusions**

In conclusion, patients undergoing TKR under CSEA had less pronounced decreases in MAP as compared to their GA counterparts. The type of anesthesia did not affect the respiratory and metabolic changes.

**References**