THE EFFECTS OF TOURNIQUET ON INTRAOCULAR PRESSURE DURING KNEE SURGERY

NEBAHAT GULCU BULUT*, KAZIM KARAASLAN*, KUTAY E. OZTURAN**, HUSAMETTIN CAKICI** AND HASAN KOCOGLU***

Abstract

Background: In this prospective study we aimed at examining the effects of pneumatic tourniquet on intraocular pressure during elective knee surgery.

Method: Twenty patients undergoing elective knee surgery under general anesthesia with tracheal intubation were included the study. Anesthesia was induced with pentothal, rocuronium and fentanyl, then maintained with sevoflurane and nitrous oxide in oxygen. Intraocular pressure measurements were performed at 7 time points; awake (baseline), following induction, following tracheal intubation, just before tourniquet inflation, after the inflation of tourniquet, before tourniquet deflation, after the deflation of the tourniquet.

Results: Baseline IOP was 15 ± 1 mmHg. Following the induction of anesthesia IOP was reduced significantly (12 ± 1 mmHg) (p <0.05), then increased to 16 ± 1 mmHg after tracheal intubation (p <0.05). IOP was significantly higher after tourniquet inflation compared with just before (13 ± 1 mmHg vs 16 ± 1 mmHg respectively) (p <0.05). There was no significant difference between the IOP measurements after the inflation and before the deflation of the tourniquet (p >0.05). The lowest value was 12 ± 0 mmHg measured after the tourniquet loosened and it was significant compared with the baseline and the measurement performed before deflation of the tourniquet (16 ± 0 mmHg) (p <0.05).

Conclusion: Pneumatic tourniquet may cause a significant IOP increase in patients performing knee surgery under general anesthesia.

Key words: intraocular pressure, tourniquet, knee surgery.

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Introduction

The ocular perfusion pressure is determined by the difference between mean arterial pressure (MAP) and intraocular pressure (IOP). The decreased perfusion may result in complications ranging from subtle vision changes to total blindness in both ophthalmic and non-ophthalmic surgery\(^1\). In the previous studies it was reported that, using the eye speculum\(^2\), Valsalva maneuver\(^3\), holding breath\(^4\), exercises\(^5,6\), the prone position\(^7\), acute volume loading\(^8\) and positive end expiratory pressure\(^9\), increased there was in the intraocular pressure inadvertently.

Pneumatic tourniquets are needed in knee surgery (arthroscopy or knee replacement surgery) to provide a bloodless field for the surgeon\(^10\). The placement of tourniquet on a lower limb causes shift of blood into the ‘systemic circulation’ in volume of 500-1000 mL and it is named as ‘tourniquet induced hypertension’\(^11\). In the literature we could not find information regarding the effects of tourniquet on intraocular pressure. Therefore we planned a prospective study to document IOP changes intraoperatively in patients scheduled for elective knee surgery with the use of pneumatic tourniquet.

Methods

Participants and procedures

After approval by our Ethics Committee and written informed consent from the patients, 20 ASA physical status I-II patients aged 40-60 yrs planned for elective knee surgery were included in the study. Surgery was performed early in the morning to avoid diurnal variations in IOP. The patients were unpremedicated before surgery.

Exclusion criteria included chronic diseases except regular hypertension, known allergies to tetracaine or any anesthetic drugs used in the study and eye disease or surgery in the medical history, and the expected tourniquet time was between 60-120 min. The tourniquet pressure was maintained at 350 mmHg in all cases.

In all cases anesthesia was induced with 5 mg.kg\(^{-1}\) thiopenthal, and 1 mcg.kg\(^{-1}\) fentanyl and 0.5 mg.kg\(^{-1}\) rocuronium for muscle relaxation and maintained with sevoflurane end tidal concentration <2%. Following the endotracheal intubation, mechanical ventilation was performed with the mixture of 33% oxygen and 66% nitrous oxide. Minute volume was set to adjust ET\(\text{CO}_2\) at 25-35 mmHg during the anesthesia. Fluid intake was restricted at 4 mL.kg\(^{-1}\).h\(^{-1}\) intraoperatively.

Assessment of IOP

IOP was measured by using a Schiötz tonometer. Topical tetracaine 0.5% was applied before the baseline measurement of IOP. Eyelid speculum was not used during measurements.

The measurements of systolic arterial pressure (SAP) and diastolic arterial pressure (DAP), heart rate, ET\(\text{CO}_2\) and IOP were performed throughout the anesthesia period and recorded at 7 time points in each subject; awake and supine (baseline) (t1), following anesthetic induction (t2), following tracheal intubation (t3), 10 min later, immediately before inflation of tourniquet (t4), after the tourniquet inflation (t5), just before deflation of the tourniquet (t6) and after deflation of the tourniquet (t7).

Statistical analysis

The data was analyzed with the simple paired test and Fisher’s exact test with ANOVA for repeated measurements. \(p<0.05\) was accepted as significant.

Table 1

<table>
<thead>
<tr>
<th>Demographic and surgical data</th>
<th>Study Group ((n = 20))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>54.0</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>7/13</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>166.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>67.5</td>
</tr>
<tr>
<td>Duration of operation (min)</td>
<td>84</td>
</tr>
<tr>
<td>Operation type (Replacement/arthroscopy)</td>
<td>8/12</td>
</tr>
</tbody>
</table>

Results

The demographic and surgical data was presented in Table 1. Baseline IOP was \(15 \pm 1\) mmHg. Following the induction of anesthesia IOP was reduced significantly (\(12 \pm 1\) mmHg) \((p <0.05)\), then increased
The effects of Tourniquet on intraocular pressure during knee surgery

Table 2
The mean values of IOP, HR, SAP and DAP at the 7 time points

<table>
<thead>
<tr>
<th>Time</th>
<th>IOP (mmHg)</th>
<th>HR (beat/min)</th>
<th>SAP (mmHg)</th>
<th>DAP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>t0</td>
<td>15 ±1</td>
<td>86 ±2</td>
<td>137 ±4</td>
<td>83 ±2</td>
</tr>
<tr>
<td>t1</td>
<td>12 ±1</td>
<td>81 ±2 #</td>
<td>128 ±4 #</td>
<td>79 ±2 #</td>
</tr>
<tr>
<td>t2</td>
<td>16 ±1</td>
<td>88 ±3</td>
<td>149 ±3 #</td>
<td>91 ±3 #</td>
</tr>
<tr>
<td>t3</td>
<td>13 ±1</td>
<td>79 ±2 #</td>
<td>128 ±3 #</td>
<td>79 ±2</td>
</tr>
<tr>
<td>t4</td>
<td>16 ±1 #§</td>
<td>76 ±2 #§</td>
<td>139 ±4 #</td>
<td>80 ±2</td>
</tr>
<tr>
<td>t5</td>
<td>16 ±0</td>
<td>78 ±2 #</td>
<td>138 ±3</td>
<td>80 ±2</td>
</tr>
<tr>
<td>t6</td>
<td>12 ±0 #§</td>
<td>75 ±2 #§</td>
<td>122 ±3 #§</td>
<td>76 ±2 #§</td>
</tr>
</tbody>
</table>

(#: p <0.05 compared with the value of t0, §: p <0.05 compared with the previous value)

to 16 ± 1 mmHg after tracheal intubation (p <0.05) (Table 2). IOP was significantly higher after tourniquet inflation compared with just before inflation (13 ± 1 mmHg vs 16 ± 1 mmHg respectively) (Table 2) (p <0.05). There was no significant difference between the IOP measurements after the inflation and before the deflation of the tourniquet (Table 2) (p >0.05). The lowest value was 12 ± 0 mmHg measured after the tourniquet was loosened and it was significant compared with the baseline and the measurement performed before deflation of the torniquet (16 ± 0 mmHg) (Table 2) (p <0.05).

The HR decreased significantly compared with the baseline in all measurements (p <0.05) except t2 time point (after intubation) (Table 2) (p >0.05). The SAP decreased significantly at the t1, t2, t3 and t6 time points (Table 2) (p <0.05). It was found that the DAP decreased significantly during the t1, t2 and t6 time points (Table 2) (p <0.05). Both HR and SAP were significantly different between before and after the inflation of tourniquet (Table 2) (p <0.05).

The decreases in the SAP and DAP values were significant after tourniquet deflation compared with measurement performed just before the deflation (Table 2) (p <0.05).

The graphical presentation of the measurement results at the 7 time points was shown in Graphic 1.

Discussion

This prospective study was the first in the literature indicating that IOP increased significantly due to the use of tourniquet during knee surgery under general anesthesia. The normal range of IOP in awake humans was reported to be 16 ±5 mmHg, and the values greater than 24 mmHg were accepted as pathologic. In the present study the amount of increase in IOP after the tourniquet inflation was 3 mmHg and similar to the IOP increase measured after intubation.

It was reported that during surgery under anesthesia IOP is a result of various factors including; a direct effect of the anesthetic agents on central diencephalic control centres, reduction of aqueous
production and drainage, relaxation of extraocular muscle tone or the volumes of choroidal blood and vitreous humour\textsuperscript{12}.

Most anesthetic and hypnotic agents, including inhalational anesthetics, barbiturates, opioids, neuroleptics and benzodiazepines are capable to decrease IOP in relation to the depth of anesthesia\textsuperscript{12}. This is significant especially in ocular surgery\textsuperscript{13}. It was shown that IOP decreases after induction of anesthesia with both total intravenous anesthesia and by propofol and isoflurane\textsuperscript{14} during laparoscopic surgery. The isoflurane continued to increase IOP above the preinduction level but propofol not. The authors speculated that this difference may be a result of the effects that, propofol cause increase in the level of arginine vasopressin (AVP) during laparoscopic surgery with head down position but inhaled anesthetics do not affect the release of AVP\textsuperscript{14}. Previously it was shown that propofol decreases the diuresis by inhibiting the AVP release from the supraoptic nucleus located in the hypothalamus\textsuperscript{15}. It was found that sevoflurane is a practical maintenance agent for adults which decreases IOP at a constantly low level in patients undergoing non-ophthalmic surgery\textsuperscript{16}. Unfortunately we could not find enough information about the effects of thiopenthal used in the present study on supraoptic neurons.

Hvidberg et al.\textsuperscript{17} showed that arterial carbon dioxide tension affects inversely the IOP in the patients under general anesthesia. It was attributed to secondary passive changes in choroidal venous blood volume. When the carbon dioxide tension increases, via the respiratory air or hypoventilation, this reflects to general circulation, thereby the central venous pressure increases, this results in enhancing the IOP. In the same report it was suggested that a similar mechanism works during postural changes. Placing the head of the patient 15 degrees above or below at the horizontal axis with the constant $P_{CO_2}$ levels, hydrostatic factors trigger the central venous pressure. The observed IOP changes were parallel in form and magnitude to the increased carbon dioxide pressure in the arterial blood. Compatible with this findings, it was shown that the lateral decubitus position increased the IOP in the dependent eye compared with non dependent eye in anesthetized patients\textsuperscript{18}. In the present study we kept the carbon dioxide pressure between 25-35 mmHg with a 15 degree head up supine position in all patients. The above mentioned factors did not affect significantly our results. We did not measure the central venous pressure in our study due to ethical concerns related to the insertion of a central venous catheter.

Schafer et al.\textsuperscript{1} showed a positive correlation between mean arterial pressure and IOP but could not discriminate the effects of arterial pressure and anesthetic depth. Although both IOP and intracranial pressure have similar range of response in pressures to intraabdominal, intrathoracic and aortic arterial pressure, it was suggested that the IOP elevation was the reflection of retinal venous pressure mainly, due to obstruction of venous outflow during exercise\textsuperscript{5}. In this study the depth of anesthesia was evaluated with heart rate and blood pressure, we could not add the bispectral index measurement for determining the depth objectively as advised\textsuperscript{1}. However, we think that the drop after induction may be related to the depth of anesthesia.

To document the mechanism that elevates IOP after acute hydration (14 mL.kg\textsuperscript{-1} h\textsuperscript{2}o) it was speculated that because of the ocular osmotic pressure gradient did not change neither vitreous hydration nor increased aqueous ultrafiltration, the result might be explained by the factors affecting aqueous drainage\textsuperscript{8}. It was shown that regional lymphatic stasis reduces the aqueous humour outflow and leads to a bilateral secondary IOP elevation\textsuperscript{19}. We provided a 15° horizontal angle for position of head-neck axis therefore we do not think that the reduced drainage pose significant effect on our results.

The depolarizing relaxants may cause intraocular hypertensive effect due to tonic contraction of the extraocular muscles, choroidal vascular dilatation or relation of orbital smooth muscle whereas non depolarizing agents are associated with a reduced intraocular pressure\textsuperscript{12}. In a study comparing rocuronium, atracurium and succinylcholin, they were all found to achieve successful intubation without serious increase in IOP\textsuperscript{20}. It was emphasized in that study that regardless of the relaxant type, adequate depth of anesthesia at the time of intubation was most important concern to maintain IOP stability. We preferred rocuronium as the muscle relaxant in our study based on the results of previous reports.
It was suggested that some conditions may be a source of error in clinically normal cornea during IOP measurement; accommodation, the Valsalva manoeuver, vertical gaze, corneal resistance and a decline due to repeated tonometry\textsuperscript{21}. The resistance to measurement or forced squeezing of the eyelids may cause the Valsalva effect thereby an increase in IOP\textsuperscript{22}. In a study whether the pediatric eyelid speculum altered the IOP indicated that IOP raised by an average of 4 mmHg in pediatric patients undergoing extra ocular eye surgery\textsuperscript{2}. We performed all measurements under general anesthesia, after seeing pupil centralization and without eyelid speculum to avoid definite errors.

Chen et al.\textsuperscript{23} showed that right unilateral forced nostril breathing decreased (UFNB) in IOP by the effects on the ciliary muscle of the eye controlled by autonomic nervous system whereas left UFNP did not. These findings suggested that IOP may be influenced by ipsilateral sympathetic system with side asymmetry in favour of right over the level of neck. It was indicated\textsuperscript{24} that UFNB decreased IOP only in men compared with women without side difference in contrast to the former study. We performed the measurements on right side in all cases not to take consideration of side asymmetry.

Arai et al.\textsuperscript{10} hypothesized that because of pneumatic tourniquet inflation facilitated hyperdynamic circulatory response due to systemic sympathetic activity, stellate ganglion blockade may alleviate this response by supressing the stellate ganglion nerves. They concluded that the ipsilateral stellate ganglion block effectively alleviated the parameters including SAP, DAP and HR indicating the circulatory response by tourniquet inflation at the lower extremity under general anesthesia.

It was reported that cluster headache attacks were associated with increased IOP predominantly on the dependent side. Barriga et al.\textsuperscript{25} reproduced an acute increase in IOP with a provocative test (the Valsalva manoeuvre) in patients with cluster headache. They found that Valsalva manoeuvre elicited an asymmetric increase in IOP significantly higher than the asymptomatic side. Because of the increment took place within only a few seconds they concluded that it was due to intraocular vasodilatation thus pointing to a latent increase in vascular reactivity. In the present study, no patient have had known autonomic disorders but higher values in some patients may be attributed to the predictor of an occult vascular sensitisation. We think that the increase in IOP in our study was mediated partially by stimulated sympathetic system. Further studies are needed to determine the exact role of acute sympathetic activation on IOP during knee surgery with tourniquet.

Conclusively, pneumatic tourniquet causes a significant IOP increase in patients during knee surgery under general anesthesia.


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References


