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# COMPARATIVE EVALUATION OF INTRAOCULAR PRESSURE CHANGES FOLLOWING INDUCTION WITH SEVOFLURANE AND THIOPENTONE IN CHILDREN

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***Keywords:***
*Proseal laryngeal mask airway, intraocular pressure.*

## *Abstract*

**Introduction:** Comparative evaluation of intraocular pressure changes following induction with sevoflurane and thiopentone in children

**Objective:** To evaluate and compare the changes in intraocular pressure following induction with sevoflurane and thiopentone in children.

**Methods:** Seventy American Society of Anesthesiologist (ASA) I-II children aged 2-8 years undergoing non ophthalmic surgery were randomly divided into two groups (Sevoflurane group and Thiopentone group) in this prospective randomised study. Anaesthesia was induced with decreasing sevoflurane concentration (8%-2%) in 100% oxygen or with intravenous thiopentone 5mgkg$^{-1}$. IOP in right eye, heart rate (HR), mean arterial pressure (MAP) and $EtCO_2$ were measured before and 1 and 3 minutes after PLMA placement.

**Results:** There was significant difference in terms of loss of eyelash reflex which was earlier in thiopentone group. The changes in IOP was significant at 1 and 3 minutes after PLMA placement ($p<0.001$). Changes in MAP was also significant at 1 and 3 minutes after PLMA placement ($p<0.05$). There are no significant changes in HR, $SpO_2$ and $EtCO_2$.

**Conclusion:** It was seen that IOP increased was less in sevoflurane group as compared to thiopentone group.

## Introduction

Intraocular pressure is defined as the pressure exerted by contents of eye against its containing wall.[1] It is mainly determined by coupling of production and drainage of aqueous humor mainly through trabecular meshwork located in anterior chamber angle. Tonometer is the device and tonometry is the method used to measure intraocular pressure. The normal intraocular pressure is 10- 21mmHg.[2]

During general anaesthesia elevation of intraocular pressure of shorter or longer duration may be due to multiplicity of factors acting from outside the globe eg. extraocular muscle contraction and pressure response to laryngoscopy and intubation.[2,3] During laryngoscopy and tracheal intubation intraocular pressure increases of the order of 10-20 mm Hg which is possibly dependent on cardiovascular sympathetic responses to tracheal intubation. In LMA (Laryngeal Mask Airway) placement there is no laryngoscopy and tracheal stimulation as seen in endotracheal intubation. Hence LMA does not increase the blood pressure and intraocular pressure to high level, that makes LMA

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useful in patients with hypertension, myocardial ischaemia and for patients undergoing ophthalmic surgery with raised intraocular pressure.[4]Besides laryngoscopy and tracheal intubation, various anaesthetic drugs may affect the intraocular pressure directly through action on central diencephalic control centres, through facilitation or inhibition of aqueous production and drainage, through relaxation or contraction of extraocular and orbicularis oculi muscle or indirectly through their effect on the cardiovascular or respiratory system.[2] Out of many factors, IOP (intraocular pressure) is directly affected mainly by changes in systemic arterial pressure.[5]

IOP measurements are difficult to obtain in children. Determination made on alert non-cooperative child overestimate IOP due to rise in central venous pressure, extraocular muscle contraction and change in choroidal blood volume. General anaesthesia allows repeated determination of intraocular pressure on quite child. Anaesthesia may influence intraocular pressure depending on the type of inhalational anaesthetic agent likesevoflurane and intravenous anaesthetic agent thiopentone which are commonly used induction agents in children that are known to influence hemodynamics and intraocular pressure.[6]

After literature search, we found that effects on intraocular pressure of various anaesthetic drugs like sevoflurane, etomidate, propofol, thiopentone etc. have been studied in adult population during induction phase of general anaesthesia. However, there are few studies comparing the effect on IOP during induction with sevoflurane and thiopentone in children. As we hypothesized that these agents may affect the IOP, hence we planned to evaluate the effect of sevoflurane and thiopentone on intraocular pressure in children during induction phase of general anaesthesia.

## Material and methods

After permission from institutional ethics committee and review board, the study was conducted in Pt B.D.Sharma, PGIMS Rohtak. Written informed consent was taken from guardians. This was prospective randomised study. Sample size was calculated as more than 30 in each group at alpha error 0.05and power 90%. For the study we took 35 in each group.

Patients with ASA grade 1 and 2 and 2-8 years of both sexes undergoing non ophthalmic surgery under GA were included in the study. Patients with history of eye infection/injury,previous eye surgery,uveitis, CNS diseases, allergy to study drugs, difficult airway and refusal to participate in present study were excluded from study.

After arrival in the operation theatre routine monitoring comprising of electrocardiography (ECG), pulse oximetry ($SPO_2$), non-invasive blood pressure (NIBP) was established. Baseline readings of all vital parameters was recorded at before induction ($T_0$). Intravenous line was secured with appropriate sized canula. Ringer lactate was used as maintenance fluid.

Preoxygenation was done with 100% $O_2$ for three minutes. Injection glycopyrrolate in a dose of 0.005 $mgkg^{-1}$ was administered. Analgesia was provided with intravenous fentanyl2$\mu gkg^{-1}$. Induction of anaesthesia was achieved with intravenous thiopentone 5$mgkg^{-1}$(Group T) or graded concentration of inhaled sevoflurane (Group S) in 100% oxygen via face mask. Clinical assessment for induction of anaesthesia was done by the loss of eyelash reflex. After that IOP was measured.

Schiotz tonometer was used to measure intraocular pressure in right eye after induction ($T_1$) while maintaining airway with bag and mask ventilation. Then, muscle relaxant inj. Atracurium in a dose of 0.5$mgkg^{-1}$ was administered to the patient and intermittent positive pressure ventilation (IPPV) was followed for next 2 minutes. PLMA of appropriate size was used to secure the airway. Intraocular pressure was measured at 1 minute ($T_2$) and 3 minutes ($T_3$) after PLMA placement. Mean arterial blood pressure and heart rate was recorded after induction ($T_1$), at 1 minute ($T_2$) and 3 minutes after PLMA placement ($T_3$). After monitoring of all the parameters, surgery was commenced and maintenance of anaesthesia was done with sevoflurane, nitrous oxide and muscle relaxant. IPPV

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was continued. At the end of surgery neuromuscular blockade was antagonized with 0.05 $mgkg^{-1}$ of neostigmine methylsulfate and 0.01$mgkg^{-1}$ of glycopyrrolate . Then patient was extubated and shifted to recovery room for further management.

## Results

Statistical testing was conducted with the statistical package for social science system version SPSS 17.0. Continuous variables was presented as mean ±SD or median if the data was unevenly distributed. Categorical variables were expressed as frequencies and percentages. The comparison of continuous variables between the groups was performed using Student's t test. Nominal categorical data between the groups was compared using Chi-square test or Fisher's exact test as appropriate. For all statistical tests, a p value less than 0.05 was taken to indicate a significant difference.

***Table I- Showing distribution of Age***

| | Group Allocation |
|---|---|
| | **Group S** |
| | **Mean ± SD** |
| Age (yrs) | 4.28 ± 2.15 |

Group S= Sevoflurane, Group T= Thiopentone

***Table II-Showing Sex distribution: Group S and Group T***

| **Sex** | **Group Allocation** | | | | **p Value** |
|---|---|---|---|---|---|
| | **Group S** | | **Group T** | | |
| | **Frequency** | **%** | **Frequency** | **%** | |
| F | 11 | 31.4% | 8 | 22.9% | 0.420 |
| M | 24 | 68.6% | 27 | 77.1% | |
| Total | 35 | 100% | 35 | 100% | |

***Table III- Showing distribution of weight***

| | Group Allocation | | **p Value** |
|---|---|---|---|
| | **Group S** | **Group T** | |
| | **Mean ± SD** | **Mean ± SD** | |
| weight (kgs) | 17.49 ± 6.64 | 18.77 ± 6.38 | 0.412 |

***Table IV- Showing distribution of ASA***

| **ASA** | **Group Allocation** | | | | **p Value** |
|---|---|---|---|---|---|
| | **Group S** | | **Group T** | | |
| | **Frequency** | **%** | **Frequency** | **%** | |
| I | 35 | 100.0% | 35 | 100.0% | – |

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| | | | | | |
|---|---|---|---|---|---|
| Total | 35 | 100% | 35 | 100% | |

***Table V- Showing baseline HR, $SpO_2$, SBP and DBP***

| | Group Allocation | | p Value |
|---|---|---|---|
| | **Group S** | **Group T** | |
| | **Mean ± SD** | **Mean ± SD** | |
| HR (bpm) | 120.74 ± 10.59 | 119.74 ± 13.05 | 0.783 |
| SpO2 (%) | 100.00 ± 0.00 | 100.00 ± 0.00 | – |
| SBP | 96.69 ± 6.37 | 98.09 ± 6.98 | 0.384 |
| DBP | 62.49 ± 4.12 | 63.49 ± 4.35 | 0.327 |

***Table VI: Showing time of loss of eyelash reflex in both groups***

| | Group Allocation | | p Value |
|---|---|---|---|
| | **Group S** | **Group T** | |
| | **Mean ± SD** | **Mean ± SD** | |
| Time of loss of eyelash reflex (sec) | 47.91 ± 1.63 | 19.40 ± 1.24 | <0.001 |

***Table VII: Showing IOP at different timings***

| | Group Allocation | | p Value |
|---|---|---|---|
| | **Group S** | **Group T** | |
| | **Mean ± SD** | **Mean ± SD** | |
| IOP During induction (mmHg) | 11.76 ± 1.19 | 12.29 ± 1.31 | 0.083 |
| IOP at 1 min after PLMA placement ( mmHg) | 13.15 ± 1.09 | 14.79 ± 1.35 | <0.001 |
| IOP at 3 min after PLMA placement (mmHg) | 11.51 ± 1.15 | 12.71 ± 1.38 | <0.001 |

***Table VIII- showing changes in heart rate in both groups***

| | Group Allocation | | p Value |
|---|---|---|---|
| | **Group S** | **Group T** | |
| | **Mean ± SD** | **Mean ± SD** | |
| HR Before induction (bpm) | 119.60 ± 11.90 | 119.94 ± 13.03 | 0.909 |
| HR During induction (bpm) | 121.31 ± 11.19 | 121.63 ± 13.38 | 0.915 |
| HR at 1 min after PLMA placement (bpm) | 128.94 ± 11.19 | 131.83 ± 13.27 | 0.329 |
| HR at 3 min after PLMA placement (bpm) | 124.23 ± 10.95 | 126.26 ± 13.31 | 0.489 |

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***Table IX –Showing mean value of changes in mean arterial pressure***

| | Group Allocation | | p Value |
|---|---|---|---|
| | **Group S** | **Group T** | |
| | **Mean ± SD** | **Mean ± SD** | |
| MAP Before induction (mmHg) | 73.71 ± 4.38 | 74.77 ± 4.88 | 0.343 |
| MAP During induction (mmHg) | 74.37 ± 4.29 | 75.54 ± 4.99 | 0.296 |
| MAP at 1 min after PLMA placement (mmHg) | 73.06 ± 3.91 | 76.91 ± 5.43 | 0.001 |
| MAP at 3 min after PLMA placement (mmHg) | 71.69 ± 3.95 | 74.51 ± 4.90 | 0.010 |

***Table X- showing changes in $SpO_2$ in both groups***

| | Group Allocation | | p Value |
|---|---|---|---|
| | **Group S** | **Group T** | |
| | **Mean ± SD** | **Mean ± SD** | |
| $SpO_2$ Before induction (%) | 100.00 ± 0.00 | 99.97 ± 0.17 | 0.321 |
| $SpO_2$ During induction (%) | 100.00 ± 0.00 | 99.97 ± 0.17 | 0.321 |
| $SpO_2$ at 1 min after PLMA placement (%) | 100.00 ± 0.00 | 99.97 ± 0.17 | 0.321 |
| $SpO_2$ at 3 min after PLMA placement (%) | 100.00 ± 0.00 | 99.97 ± 0.17 | 0.321 |

***Table XI- showing changes in $EtCO_2$ in both groups***

| | Group Allocation | | p Value |
|---|---|---|---|
| | **Group S** | **Group T** | |
| | **Mean ± SD** | **Mean ± SD** | |
| $EtCO_2$ Before induction (mmHg) | 30.80 ± 1.43 | 30.89 ± 1.59 | 0.813 |
| $EtCO_2$ During induction (mmHg) | 31.80 ± 1.49 | 31.49 ± 1.63 | 0.404 |
| $EtCO_2$ at 1 min after PLMA placement (mmHg) | 32.06 ± 1.53 | 31.71 ± 1.23 | 0.305 |
| $EtCO_2$ at 3 min after PLMA placement (mmHg) | 31.09 ± 1.22 | 30.83 ± 1.20 | 0.378 |

***Table XII- showing adverse reaction in both groups***

| **Other adverse reaction** | **Group Allocation** | | | | **P Value** |
|---|---|---|---|---|---|
| | **Group S** | | **Group T** | | |
| | **Frequency** | **%** | **Frequency** | **%** | |

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| | | | | | |
|---|---|---|---|---|---|
| No | 35 | 100.0% | 35 | 100.0% | – |
| Total | 35 | 100% | 35 | 100% | |

## Discussion

There was no significant differences in baseline HR, SBP, DBP and $SpO_2$ in both the sevoflurane and thiopentone group.

In our study the mean IOP during induction and before PLMA placement was 11.76±1.19 mmHg in group S and 12.29±1.31 mmHg in group T and p value was > 0.05. The mean IOP at 1 minute after PLMA placement was 13.15±1.09 mmHg in group S and 14.79±1.35mmHg in group T and p value was <0.001. The mean IOP at 3 minute after PLMA placement was 11.51±1.15mmHg in group S and 12.71±1.38 mmHg in group T and p value was < 0.001. There was increase in IOP at 1 minute after PLMA placement in both the groups but the increase was slightly more in thiopentone group. In sevoflurane group the IOP returns to the pre PLMA placement level at 3 minute after PLMA placement while in thiopentone group it slightly remained less than the pre PLMA placement level.

## Conclusion

There was no significant difference between the groups in demographic profile.We concluded that the intraocular pressure initially raised following 1 minute after PLMA placement in both sevoflurane and thiopentone group but the increase is a little bit more in thiopentone group and the intraocular pressure returns to pre PLMA placement in sevoflurane group while in thiopentone group it does not touches the pre PLMA placement value at 3 minutes after PLMA placement. Hemodynamic stability, $SpO_2$ and $EtCO_2$levels was preserved during PLMA insertion in paediatric patients. Our study concludes that, with respect to IOP, sevoflurane can be preferred induction agent in small children over thiopentone in non ophthalmic surgeries.

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