Intraocular pressure following inhalation anesthesia and sodium thiopental

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A review of past literature reveals only one report concerning the effect of isoflurane on intraocular pressure (IOP) in pediatric patients. This article focuses on the results of a study of IOP performed on 30 adult patients under isoflurane, enfurane and halothane anesthesia following an intravenous induction with sodium thiopental.

With the introduction of any new drug into the anesthesia armamentarium, there are many parameters that require evaluation. In ophthalmology, the effects of anesthetics on intraocular pressure (IOP) are of considerable importance and the necessity for maintaining normal or reduced intraocular pressure has been well documented. This is particularly important in open globe injuries where a sudden increase in IOP may result in expulsion of vitreous and loss of vision.

Isoflurane, the most recent inhalation agent approved by the FDA, is now a well established drug in anesthesia. In the authors' institution (University of Texas Health Science Center) it is by far the most commonly utilized agent. Its popularity is based on rapid depression of laryngeal and pharyngeal reflexes. Levels of anesthesia may be altered quickly and recovery is rapid with a minimum of nausea and vomiting. It produces vasodilatation and dose-related decreases in systemic blood pressure, but cardiac output is maintained by an increase in heart rate. Lastly, it does not appear to sensitize the myocardium to ventricular dysrhythmias with the concomitant use of epinephrine.

In 1975, Ausinsch, et al. studied the effects of isoflurane and halothane on IOP in children. A search of the literature before and after this period did not reveal any further investigations of isoflurane and its effects on IOP. Runciman, et al., in 1978, published their findings on the effects of halothane and enfurane on IOP and concluded that enfurane caused a significant decrease. It seemed pertinent at this time to evaluate the effects of isoflurane on IOP in comparison with halothane and enfurane.

Method and materials of the present study
A total of 30 ASA I and II adult patients, ages 18 to 60 years, were studied. Pregnant females, neurosurgical patients and those with a prior history of convulsions were omitted. Also excluded were patients with known eye disease, those scheduled for ophthalmic surgery or those weighing over 85 kg. Ten patients each received isoflurane, enfurane or halothane on a random selection basis.*

Preoperative IOP was measured prior to the

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*Informed consent was obtained from all patients and the protocol was approved by the University of Texas Health Science Center Institutional Review Board.
induction of anesthesia. One drop of proparacaine hydrochloride (Alcain®) was instilled in each eye followed by measurement of IOP.

All patients were premedicated one hour prior to the induction of anesthesia with morphine sulfate 0.15 mg/kg. Morphine was selected on the basis that its effect on IOP is minimal. Blood pressure and heart rate were recorded, and the patients were monitored with a cardioscope. A temperature monitor and esophageal stethoscope were placed following induction.

D-Tubocurarine 3 mg and atropine 0.25 mg were administered 3 minutes prior to the induction of anesthesia, which was accomplished by the administration of sodium thiopental 5-7 mg/kg. A baseline IOP reading was obtained following loss of lash reflex and the advent of central positioning of the eyes. After this baseline IOP determination, patients were given either isoflurane (1.5-3.0%), enfurane (3.5-4.5%) or halothane (2-4%) in 100% O₂, followed immediately by the administration of succinylcholine 1.5 mg/kg to facilitate intubation.

After insertion of the tracheal tube, 50% N₂O+O₂ was administered with isoflurane 1.0-2.5%, enfurane 1.5-3.0% or halothane 0.5-1.0%. A peripheral nerve stimulator was utilized to determine the absence of the effects of succinylcholine after which IOP was measured at 10, 20 and 30 minute intervals following administration of each anesthetic. Vital signs were recorded every 5 minutes.

Ventilation was controlled and an end tidal CO₂ monitor (Instrumentation Laboratories—IL200) was connected to the tracheal tube to maintain PₐCO₂ within the normal range. IOP measurements were made with a Schiotz™ indentation tonometer using a 5.5 gm weight with patients in the supine position. All measurements were performed solely by the primary investigator under carefully controlled conditions.

Individual groups were compared using a two-way analysis of variance with repeated measures.

**Results**

Results of the study are summarized in Table I and depicted graphically in Figure 1. Mean anesthetic concentrations for isoflurane, enfurane and halothane were 1.68, 1.9 and 1.1 volume % respectively. Blood pressure readings prior to induction of anesthesia ranged between 120/70 and 130/100 with an average of 123/79 for the isoflurane group. The 10, 20 and 30 minute readings averaged 104/69, 100/65 and 108/69 respectively. For the enfurane group, preinduction values were between 90/50 and 140/98 with an average of 121/76. The 10, 20 and 30 minute values averaged 112/72, 105/67 and 104/69 respectively. Pre-induction blood pressures for the halothane group were between 100/70 and 140/90 with an average of 119/75. The 10, 20 and 30 minute values were 98/65, 100/71 and 104/68 respectively.

We were unable to monitor alveolar anesthetic concentration for these procedures at the time of this study. However, all patients were judged to be in a surgical plane of anesthesia as evidenced by the absence of movement or changes in vital signs at the onset of surgery.

All three groups were similar in that baseline readings following an induction dose of thiopental with subsequent loss of lash reflex were lower than those in the awake patient. The most profound decrease in IOP was 10 minutes following intubation with a steady rise in pressure between the 10 and 20 minute readings and the 20 and 30 minute read-

<table>
<thead>
<tr>
<th>Table I</th>
<th>Mean (± SEM) intraocular pressure measurements (mmHg)</th>
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<tr>
<td></td>
<td>Mean anesthetic concentration (Vol. %)</td>
</tr>
<tr>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>Group 1 — Isoflurane</td>
<td>11.36 ± 1.02</td>
</tr>
<tr>
<td>Group 2 — Enflurane</td>
<td>11.09 ± 0.94</td>
</tr>
<tr>
<td>Group 3 — Halothane</td>
<td>10.81 ± 1.06</td>
</tr>
</tbody>
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A = Immediately following thiopental induction
B = Ten minutes post intubation
C = Twenty minutes post intubation
D = Thirty minutes post intubation
ings. All of these differences were statistically significant, \( P = 0.05 \). At the end of 30 minutes, all patients had returned to near baseline (after sodium thiopental) or higher levels with no significant difference between groups, \( P = 0.05 \).

Although differences between groups were not statistically significant, IOP under enflurane anesthesia was slightly lower than the baseline thiopental reading at the end of 30 minutes and the halothane group was higher than the baseline. This is in agreement with the findings of Runciman where a comparison was made between halothane and enflurane. Isoflurane was approximately the same as the baseline thiopental reading.

**Discussion**

This study was designed to evaluate the effect of isoflurane on IOP in comparison with enflurane and halothane. All other adjuvants that might possibly affect a change in IOP were eliminated. The only exception was succinylcholine which was necessary to facilitate intubation of the trachea. The rise in IOP following the administration of succinylcholine has been well documented in the past by many authors. Pandey, in 1972, demonstrated that the rise in IOP did not persist for more than 6 minutes.

In the present study, the first inhalation anesthetic measurement was made 10 minutes post-intubation and a peripheral nerve stimulator was utilized to determine the absence of any effect of succinylcholine. No additional muscle relaxants were administered prior to the completion of the last IOP measurement.

The results of this study were surprising in that it had been theorized that isoflurane would possibly lower IOP to a greater extent than enflurane or halothane. This theory was based on the fact that isoflurane produces more profound muscle relaxation than the other inhalation agents and

![Figure 1](image_url)

*Figure 1 Intraocular pressure following inhalation anesthesia and sodium thiopental.*

Preinduction intraocular pressure (awake state) and intraocular pressure following an induction dose of thiopental (baseline) and 10, 20 and 30 minutes post-intubation under isoflurane, enflurane or halothane anesthesia.
one of the factors involved in lowering IOP is believed to be relaxation of intra- and extraocular muscles. However, enflurane patients exhibited the lowest 30 minute reading.

In a review of anesthesia for ophthalmic surgery by Donlon, Smith and other authors, it can be concluded that adequate sedation and general anesthesia all cause a decrease in IOP from the awake state. The present investigation also substantiates these conclusions.

Our most important finding indicates that none of the inhalation agents demonstrated an appreciable effect on IOP as compared to sodium thiopental. This agrees with the work done by Ausinsch, et al. where halothane and isoflurane were compared with pre-induction values in sedated pediatric patients.

Of particular interest was the 10-minute post-intubation reading, which was the lowest in the study and statistically significant. Many authors in the past have demonstrated a profound decrease in IOP following the use of sodium thiopental; and Everett, et al. found as much as a 30% reduction of IOP. This reduction in IOP has been attributed to the increased facility of aqueous outflow and perhaps, in part, to relaxation of extraocular muscles. In a study of 408 normal eyes in infants and children, utilizing seven different anesthetic techniques, Dominguez, et al. found that the IOP levels were not related to the blood concentration of the anesthetic.

The low IOP occurring 10 minutes post-intubation coincides with a fairly high brain concentration of thiopental plus a maintenance concentration of either isoflurane, enflurane or halothane. Following this low 10 minute reading, there was a steady rise in IOP up to sodium thiopental baseline levels at the end of 30 minutes. It should be emphasized that the lowest IOP may not have occurred at exactly 10 minutes, because readings were only taken at 10, 20 and 30 minutes post-induction with thiopental.

The rise in IOP following the 10-minute reading was probably due to the redistribution of thiopental. Initially the brain, liver, heart and kidneys receive the highest concentration of anesthetic because of their high perfusion rates. Following the rapid uptake of thiopental by the brain, 20 to 30 minutes is required for redistribution to muscle and poorly perfused tissues. The 10 minute low IOP correlates with figure results on the uptake and distribution of thiopental as illustrated by Saidman and Brodie.

In summary, 30 healthy patients undergoing general anesthesia with isoflurane, enflurane or halothane were studied for changes in IOP. No muscle relaxant was utilized during pressure readings.

Thirty minutes of isoflurane, enflurane or halothane anesthesia did not demonstrate any further reduction in IOP following a generous induction dose of sodium thiopental 5-7 mg/kg. There was also no significant difference between agents. The greatest decrease in IOP was found to occur 10 minutes after induction with thiopental; and following redistribution of this drug, the IOP rose steadily to thiopental baseline levels under inhalation anesthesia with the above agents.

This rise in IOP may occur during surgery on the eye which could adversely affect the outcome of the procedure. It would be of primary importance in the traumatized eye. A second dose of thiopental after approximately 10 minutes could be effective in preventing this rise in intraocular pressure.

REFERENCES


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