Even higher pulse strength may be required in rare circumstances, but increasing pulse duration beyond 10 ms will not decrease threshold strength and is not necessary because the strength–duration relationship is already flat between 5 and 10 ms.

We conclude that transesophageal atrial pacing is safe and has few complications. We have not had complaints suggestive of esophageal burns in our subjects, and the absence of this potential complication is confirmed by the report of Burrack and Furman[3] and that of Shaw *et al.*[5] In our experience and in that of others, inadvertent ventricular capture and pacing of intrathoracic motor nerves are rare in adult patients.[11,12] Signs of tracheal irritation (*e.g.*, coughing in conscious subjects), however, are common when the pacing electrode is too far proximal to the site of minimum stimulation threshold. In this instance, advancing the electrode for 2–3 cm distally will increase its distance from the carina, decrease the current required to capture the atria, and eliminate this complication.

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The Effect of Different Methods of Inducing Anesthesia on Intraocular Pressure

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Patients with penetrating eye injuries frequently require induction of general anesthesia and rapid endotracheal intubation with a technique that does not produce increases in intraocular pressure (IOP). Previous studies have shown that thiopental,[1] narcotic analgesics,[2] and volatile inhaled anesthetics[3] all lower IOP, with the possible exception of ketamine.[4] The nondepolarizing neuromuscular blocking drugs also minimally affect IOP. Atracurium is a nondepolarizing neuromuscular blocking agent that has a rapid onset and shorter duration of action than pancuronium.[5,6] Atracurium has also demonstrated minimal cardiovascular effects, making it ideal for rapid-sequence endotracheal intubation. A comparison of IOP responses to rapid-sequence induction and endotracheal intubation with different anesthetics in various doses has not been described. The purpose of the present study, therefore, was to compare the IOP and cardiovascular changes during rapid-sequence induction employing atracurium-facilitated endotracheal intubation with various combinations of iv anesthetics.

Materials and Methods

The study was approved by the Human Investigation Committee of the hospital. The purpose and the procedure of this investigation were explained to all patients by one of the investigators prior to surgery, and an informed consent obtained. Seventy patients (39 females, 31 males) ages 15–76 yr, weighing 45–111 kg (ASA Class I and II), and scheduled for elective nonocular surgery

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TABLE 1. Anesthetic Agents and Dose Regimen for Patient Groups

Group	Order of Administration
1	Thiopental 5 mg/kg atracurium 0.6 mg/kg
2	Thiopental 7 mg/kg atracurium 0.6 mg/kg
3	Thiopental 5 mg/kg atracurium 0.8 mg/kg
4	Atracurium 0.6 mg/kg thiopental 5 mg/kg
5	Lidocaine 1.5 mg/kg thiopental 7 mg/kg atracurium 0.6 mg/kg
6	Etomidate 0.3 mg/kg atracurium 0.6 mg/kg
7	Ketamine 2 mg/kg atracurium 0.6 mg/kg

were studied. Premedication consisted of meperidine 1 mg/kg, or morphine sulphate 0.1 mg/kg or diazepam 0.15 mg/kg im, with atropine 6 μg/kg or glycopyrolate 3 μg/kg im, 1 h before induction of anesthesia.

Ten consecutive patients were assigned to one of seven groups (table 1). Induction of anesthesia was with thiopental, etomidate, or ketamine. Endotracheal intubation was facilitated with atracurium in all groups. IOP was measured with a Mueller® electronic tonometer (ophthalmodynamometer, Model OP-9100) using a weight of 5.5 g, after the application of a corneal analgesic (proparacaine 0.5%) in the patient's right eye. Intraocular tension measurements were done (by the same investigator [S.K.B.]) prior to and 15 s following injection of the anesthetic agent, 90 s following atracurium, and at 30, 60, and 120 s following endotracheal intubation. The disappearance of the muscle twitch was noted with the assistance of a peripheral nerve stimulator. The vocal cords were sprayed with 4 ml of lidocaine 4% prior to endotracheal intubation, except Group 5. Heart rate (HR) and arterial blood pressure (BP) were recorded at the same intervals. The quality of intubation (table 2) was subjectively assessed by vocal cord relaxation and the degree of coughing, bucking, or straining (if present) following endotracheal intubation. All patients breathed oxygen prior to induction of anesthesia and were lying supine when IOP was determined. Neomycin-gramicidin-polymixin B ophthalmic ointment was instilled in the examined eye following the tonometric readings.

TABLE 2. Quality of Intubation Assessment Scale

Score	Indicator
0	Bucking, straining, and coughing all present
1	Straining, coughing, no bucking, vocal cords not well abducted
2	No straining, may have some coughing, vocal cords abducted
3	No coughing, vocal cords well abducted

Intraocular tensions were converted to intraocular pressures (IOP) using the nomogram (Calibration Scale for Schiotz Tonometers—1955)[7] approved by the Committee on Standardization of Tonometers of the American Academy of Ophthalmology and Otolaryngology. IOP, mean BP, and HR data were analyzed using two-way analysis of variance (ANOVA) with repeated measures in one factor. Ages, weights, and time from atracurium administration to twitch disappearance were compared using a one-way ANOVA. Statistical differences between means were determined using the Tukey-a method. Endotracheal intubating conditions were compared using the Kruswall-Wallis H statistic. Significant differences between groups were determined using the Mann-Whitney U-test. A significance level of $P < 0.05$ was considered statistically significant.

RESULTS

Ages and body weights were similar in the seven groups. The control values of IOP, HR, and BP were similar in all groups (table 3).

IOP decreased significantly in all groups 15 s following thiopental, etomidate, atracurium–thiopental, and ketamine (table 3). Atracurium did not significantly change IOP in any group. IOP increased during tracheal intubation but returned toward preintubation values by 120 s in all groups except 4 and 7, where it remained significantly above the preintubation value but not significantly different from preinduction control.

The mean BP increased 30 s after tracheal intubation in Groups 1 and 3–7. HR increased at 15 s following induction in all groups except 6. Thirty seconds after intubation, HR had significantly increased from preinduction control in all groups except 1 and 6. HR remained significantly elevated at 120 s in these groups.

In Group 6, three patients complained of burning pain in their arm on injection of etomidate. One patient showed marked erythema localized on his chest and upper extremities that was treated with diphenhydramine 25 mg iv. Another demonstrated transient generalized increase in muscle tone, which subsided without additional treatment.

Intubating conditions (table 4) were found to be improved with increased doses of atracurium at the same dose of thiopental and with atracurium administration prior to thiopental. The other groups did not significantly differ in their intubating scores. No significant difference in the time to disappearance of muscle twitch was seen in any group (table 4).

DISCUSSION

The effect of anesthetics on IOP is important in the selection of anesthesia for patients with elevated IOP or

TABLE 3. Changes in IOP (mmHg), MAP (mmHg), and HR after Atracurium-facilitated Endotracheal Intubation

	Control	15 s after Induction	90 s after Atracurium	30 s after Intubation	60 s after Intubation	120 s after Intubation
Group 1						
IOP	13.1 ± 1.0	8.3 ± 0.8*	7.2 ± 2.1*	11.6 ± 1.5†	10.2 ± 1.1	7.0 ± 0.7*
HR	91 ± 6.4	94 ± 5.1	97 ± 4.9	102 ± 5.2	95 ± 4.6	95 ± 5.1
MAP	98 ± 5.0	87 ± 4.5	100 ± 5.3	111 ± 6.8	107 ± 5.1	111 ± 5.7
Group 2						
IOP	14.1 ± 0.7	6.2 ± 0.8*	6.9 ± 0.7*	9.3 ± 1.1*	8.5 ± 1.0*	7.9 ± 0.8*
HR	82 ± 6.5	98 ± 5.8*	101 ± 6.1*	102 ± 6.6*	93 ± 6.6	97 ± 5.6*
MAP	113 ± 2.9	94 ± 5.6	107 ± 5.4	110 ± 2.7	110 ± 4.4	106 ± 3.6
Group 3						
IOP	14.4 ± 1.2	9.2 ± 1.2*	6.5 ± 0.7*	12.6 ± 1.3†	12.2 ± 1.4†	9.0 ± 1.3*
HR	86 ± 4.6	98 ± 3.5	110 ± 5.8*	119 ± 5.1*	115 ± 5.5*	107 ± 3.8*
MAP	95 ± 6.6	90 ± 4.9	85 ± 4.6	115 ± 7.4	114 ± 6.1	101 ± 5.7
Group 4						
IOP	14.3 ± 0.8	9.7 ± 0.6*	9.9 ± 1.2*	13.8 ± 1.0†	13.1 ± 1.1†	11.5 ± 1.1†
HR	89 ± 5.3	104 ± 5.1*	100 ± 4.2	113 ± 3.5*	106 ± 5.2*	106 ± 4.9*
MAP	106 ± 6.0	103 ± 6.0	101 ± 4.7	115 ± 4.8	115 ± 4.4	108 ± 4.1
Group 5						
IOP	15.0 ± 0.7	9.5 ± 0.9*	8.3 ± 0.8*	10.2 ± 0.7*	9.6 ± 0.5*	9.4 ± 0.6*
HR	81 ± 3.9	99 ± 5.9*	102 ± 4.5*	106 ± 3.0*	104 ± 3.9*	105 ± 4.2*
MAP	95 ± 2.7	88 ± 3.3	94 ± 3.2	105 ± 3.6	105 ± 3.8	102 ± 2.7
Group 6						
IOP	14.7 ± 0.8	10.7 ± 1.1*	8.6 ± 0.7*	13.9 ± 1.7†	12.1 ± 1.3†	10.2 ± 1.1*
HR	77 ± 6.7	77 ± 5.6	85 ± 6.8	93 ± 4.6	89 ± 5.5	88 ± 4.8
MAP	97 ± 7.9	99 ± 4.8	105 ± 8.1	119 ± 8.2	111 ± 7.7	103 ± 7.8
Group 7						
IOP	14.6 ± 0.9	9.5 ± 0.5*	8.5 ± 0.5*	15.0 ± 0.9†	13.3 ± 1.0†	13.2 ± 1.0†
HR	74 ± 4.2	83 ± 5.1	96 ± 4.1*	98 ± 3.9*	104 ± 5.7*	102 ± 5.9*
MAP	103 ± 5.0	109 ± 5.9	121 ± 6.9	120 ± 6.5	121 ± 7.6	116 ± 4.6

IOP = intraocular pressure; MAP = mean arterial pressure; HR = heart rate.

* Denotes statistical significance from control ($P < 0.05$).

† Denotes statistical significance from 90 s after atracurium pre-intubation value ($P < .05$).

open eye injuries. Additionally, other factors known to influence IOP include hypoxia, hypercarbia, coughing, straining, and depth of anesthesia.[1,8,9] Although the end-expired carbon dioxide concentration and central venous pressure were not measured, all patients were breathing oxygen and ventilated prior to and during measurement of IOP. The timing of measurements was devised to determine changes in ocular tension, HR, and BP following injection of each individual anesthetic and endotracheal intubation.

Previous studies with thiopental have demonstrated a dose-dependent reduction in IOP in the range from 3 to 5.5 mg/kg.[3,10] In Group 2 we observed that the IOP drop following induction was not only the largest but that the 30-s postintubation values of ocular tension also remained well below controls. It seems likely that higher induction doses of thiobarbiturates may incur a suppressive effect of greater magnitude on ocular tension *via* a pathophysiologic mechanism mediated through the diencephalic centers in the midbrain.[11] A higher dose probably also diminishes the intubation-triggered pharyngeal and laryngeal reflexes that may lead to increases in IOP.

Succinylcholine produces the most rapid onset of neuromuscular blockade. However, it may increase IOP for up to 6 min following injection and may be contraindicated in patients with open eye injuries and during intraocular procedures.[12,13] Paralytic doses of *d*-tubocurarine and its congeners either decreases or does not change IOP.[10,14] In emergency situations the delayed onset of

TABLE 4. Time to Disappearance of Muscle Twitch and Intubation Score

	Twitch Suppression*	Intubation Score†
Group 1	76.4 ± 9.9	2 (0–3)
Group 2	73.6 ± 9.9	2 (1–3)
Group 3	76.4 ± 9.2	2 (2–3)‡
Group 4	73.6 ± 9.3	3 (2–3)‡
Group 5	68.9 ± 10.8	3 (2–3)
Group 6	59.7 ± 10.1	2 (0–3)
Group 7	69.4 ± 11.3	2 (2–3)

* Time from atracurium administration to disappearance of muscle twitch (mean ± SD).

† Median intubation scores; range in parentheses.

‡ Denotes statistical significance ($P < .05$) between Groups 3 and 4.

sufficient paralysis for tracheal intubation makes nondepolarizing muscle relaxants less suitable due to the long interval when the airway is unprotected. Our study demonstrates that atracurium 0.6–0.8 mg/kg allows for tracheal intubation within 90 s with little effect on IOP. Atracurium injection given prior to thiopental (Group 4) showed neither the decrease or sustained lower IOPs as did Group 2, which received the thiopental prior to atracurium. The variation in response of ocular tension in this group may be due to altered pharmacodynamics of thiopental when it is administered after the intermediate-acting neuromuscular blocking agent.

The onset of complete neuromuscular block is shortened by increasing the dose of atracurium from 0.2 to 0.6 mg/kg.[5] In the present study, increasing the dose of atracurium from 0.6 mg/kg to 0.8 mg/kg did not alter the time to maximum twitch suppression. We did, however, find a significant improvement in intubating scores with the increased dose of atracurium. The improvement in intubating scores in Group 3 is a likely result of the completeness of neuromuscular block when a dose of 0.8 mg/kg of atracurium is given.

Intratracheal administration of lidocaine spray may attenuate the hemodynamic changes[15] or modify aspects of endocrine response to the stress of laryngoscopy and endotracheal intubation.[16] We did not, however, find that this prevented the effects of laryngotracheal stimulation upon IOP. Lidocaine given iv prior to tracheal intubation tends to suppress the abrupt hypertensive reactions to laryngoscopy and intubation[17] by obtunding the laryngeal reflexes, vasodilating the peripheral circulation, and also by its negative chronotropic effect on myocardium. A recent study on IOP responses has demonstrated an attenuation of intraocular hypertension following pancuronium-facilitated intubation with iv lidocaine pretreatment.[18] The results (Group 5) of our study are in agreement with the findings of these investigators, where pretreatment with iv lidocaine prevented increase in IOP similar to the effect seen with larger doses of thiopental (Group 2).

The effect of ketamine on IOP is not well defined, as previous studies involved noncomparable groups of patients. In one study[4] children ages 4–7 yr received ketamine as the sole anesthetic. In another study of children[19] conditions were not standardized. In adults, under standardized conditions,[20] changes in IOP were neither large nor uniform in direction. Our data demonstrated a significant decrease in IOP 15 s following ketamine. This also conforms to the time period when the state of dissociation is said to be achieved by this nonbarbiturate agent.[21] The quantitative changes in IOP after ketamine injection in our study are similar to those described by Peuler *et al.*[20] with an early decrease in IOP at 2 min after ketamine and a return toward control in a further 2 min.

Based on the results of this investigation, we feel that increases in IOP associated with endotracheal intubation may be minimized by induction of a deeper level of anesthesia in addition to using nondepolarizing muscle relaxants. It appears that the selection of an individual iv anesthetic does not matter if an appropriate level of anesthesia is accomplished. Atracurium, as demonstrated in this study, and vecuronium[22] do not increase IOP. As the time to onset of paralysis is somewhat shorter and the duration of action considerably less than observed with *d*-tubocurarine or pancuronium, they may indeed be the best choice of muscle relaxants for rapid-sequence intubation in patients for open eye surgery. Furthermore, the onset of neuromuscular blockade may be shortened with the use of a priming dose,[23] and this technique may prove to be superior to the ones used in the present study. It should be pointed out that although these nondepolarizing muscle relaxants offer advantages over the older nondepolarizing agents for rapid-sequence intubation, the depolarizing muscle relaxant succinylcholine is still the best muscle relaxant for a rapid-sequence technique in the absence of an open eye injury. A recent study in patients with open-eye injuries suggested that succinylcholine, while theoretically contraindicated, may not be associated with increases in IOP under the proper anesthetic conditions.[24] We feel this area needs further clarification.

In summary, anesthesia induced with large doses of iv anesthetics in conjunction with atracurium or vecuronium provides excellent intubating conditions and is associated with minimal changes in IOP. Therefore, we feel these techniques presently represent the best approach in the anesthetic management in patients with open eye injuries.

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Spurious Pulse Oximeter Desaturation with Methylene Blue Injection

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Pulse oximetry allows reliable and noninvasive monitoring of arterial oxygenation. Arterial oxygen saturations of 65–100% are accurately recorded.[1] However, several clinical conditions interfere with the accuracy of the oximeter. These include peripheral vasoconstriction caused by vasopressors, peripheral vascular disease, hypothermia, hypotension, dyshemoglobinemias,[2] as well as the placement of a sphygmomanometer cuff on the ipsilateral extremity and the application of high-intensity heat lamps.[3] We report a case of iv administration of methylene blue dye causing a pulse oximeter to give a spurious reading of hemoglobin desaturation.

REPORT OF A CASE

A 64-yr-old man was scheduled for cystoscopy, transurethral resection of the prostate gland, and right testicular mass excision. The past medical history was unremarkable. Past surgical history included a left nephrectomy under spinal anesthesia without complications. The patient had no known drug allergies, took no medications, and did not smoke tobacco.

Physical examination revealed an arterial blood pressure of 130/70 mmHg, a heart rate of 60 beats/min, height 176 cm, and weight 91 kg. The airway and teeth were normal. Examination of the heart and lungs was unremarkable. All laboratory data, including hemoglobin concentration, serum electrolytes, chest radiogram, and electrocardiogram, were normal. The physical status was ASA Class I.

The patient received intraoperative monitoring that included electrocardiogram, oral temperature, end-tidal carbon dioxide tension, right brachial indirect blood pressure, and left index finger pulse oximetry. After breathing oxygen, general anesthesia was induced with sufentanyl 20 μg and thiopental 250 mg iv. After adequate airway control had been verified, succinylcholine 120 mg was given iv to facilitate oro-tracheal intubation. Anesthesia was maintained with 1.0% enflurane, in a 50% nitrous oxide/50% oxygen mixture. Neuromuscular blockade was maintained with intermittent doses of atracurium and monitored with a peripheral nerve stimulator applied to the left ulnar nerve. Ventilation was controlled.

Throughout the procedure the pulse oximeter indicated a hemoglobin oxygen saturation of 99%. On completion of surgery, methylene blue 100 mg was administered iv to check ureteral urine flow. Within 30 s the oxygen saturation alarm sounded and the monitor indicated a hemoglobin oxygen saturation of 65% with a satisfactory pulse search signal. All other clinical and monitoring signs were stable, bilateral breath sounds were good over both lung fields, and the patient was not cyanotic. The oxygen saturation rose to 97% over the next 5 m. Twenty minutes later, a second bolus of methylene blue was administered iv with identical results. After the anesthetic had been discontinued, the neuromuscular blockade reversed with neostigmine, and glycopyrolate and the trachea extubated, recovery from anesthesia and surgery was uneventful.

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