INTRA-OCULAR PRESSURE CHANGES DURING HALOTHANE AND ENFLURANE ANAESTHESIA

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SUMMARY

We have measured intra-ocular pressure (i.o.p.) in 20 patients anaesthetized with halothane or enflurane (0.6 MAC in oxygen) for repair of trauma to the eye. The changes in i.o.p. with halothane 0.5% were unpredictable, whereas enflurane 1% reduced intra-ocular tension consistently in all the patients studied. Enflurane is recommended as a possible alternative anaesthetic for surgery on the open eye.

The effect of any new anaesthetic agent on intraocular pressure (i.o.p.) should be assessed before the drug can be recommended for routine use in ophthalmic anaesthesia. Trichloroethylene has been shown to cause an undesirable increase in i.o.p. (Al Abrak and Samuel, 1975), whereas halothane 1–2% is known to produce a decrease (Al Abrak and Samuel, 1974a). The non-depolarizing neuromuscular blocking drugs tubocurarine and pancuronium also have differing effects on i.o.p., the former decreasing and the latter having no effect on eyeball tension (Al Abrak and Samuel, 1974b).

The effect of enflurane on i.o.p. has been investigated by Radtke and Waldman (1975), but other factors which may alter intra-ocular pressure (Al Abrak and Samuel, 1974a; Duncalf, 1975; Macdiarmid and Holloway, 1976), were not strictly controlled. In order to minimize the effect of these factors, we have adopted the recommendations of Al Abrak and Samuel (1974a) and standardized the anaesthetic technique used in this study.

PATIENTS AND METHODS

Twenty healthy adult patients (age range 18–41 yr) gave informed consent for this study. They were allocated to two groups on the cohort principle, to avoid circuit contamination by the inhalation agent not currently in use. Anaesthesia was supplemented under controlled conditions with either halothane 0.5% (group 1) or enflurane 1% (group 2). These concentrations are approximately equivalent to 0.6 MAC in oxygen, and represent those commonly used to augment anaesthesia with nitrous oxide, oxygen and controlled mechanical ventilation. The anaesthetic agents were vaporized from two new vaporizers (Fluotec and Enfluratec: Cyprane Ltd, Mk III models).

Thirteen of the patients studied were to undergo evisceration and seven, attempted repair of a severely traumatized eye. The operations were performed electively, and the patients were in a normal state of hydration during investigation. After instillation of local anaesthetic solution into the conjunctival sac, the intra-ocular pressure of the patient's normal eye was measured before induction of anaesthesia, using a Perkins hand-held appplanation tonometer (Perkins, 1965).

Premedication comprised morphine 10–15 mg i.m. according to weight, together with diazepam 5 mg i.m. 1 h before operation. Anaesthesia was induced with thiopentone 4.5–5.5 mg kg⁻¹ and neuromuscular blockade was produced with pancuronium 0.1 mg kg⁻¹. Pancuronium was chosen to prevent significant alteration of either i.o.p. (Al Abrak and Samuel, 1974b) or the haemodynamic state (Coleman et al., 1972).

After manual ventilation with 40% oxygen in nitrous oxide by mask until adequate neuromuscular blockade had been achieved, the larynx was sprayed with 4 ml of lignocaine 4%, and the trachea was intubated. Thereafter, ventilation was controlled using a Manley ventilator delivering a minute volume of 120 ml kg⁻¹. A constant inflation pressure of 2.5 kPa was used throughout the study.

The inspired oxygen concentration was maintained constant at 40% by monitoring the fresh gas flow with a Beckman OM 11 oxygen analyser and adjusting
the flow rate of nitrous oxide and oxygen. The end-
tidal carbon dioxide concentration was monitored 
continuously with a Beckman LB2 infra-red carbon 
dioxide analyser and maintained at 5% by the addition 
of carbon dioxide to the fresh gas supply if necessary.

Central venous pressure (c.v.p.) was measured 
using a 16-gauge i.v. catheter which was inserted into 
the internal jugular vein after the patient had been 
aanaesthetized. The surface of the operating table was 
used as the zero reference point for all c.v.p. 
measurements. Surgery was conducted with the patient in 
the horizontal position.

Recordings of heart rate and systolic arterial 
pressure were made at 5-min intervals. Both e.c.g. 
(standard lead I) and heart rate (photoelectric finger 
transducer) were displayed continuously on a Data-
scope 610 module. Systolic arterial pressure was 
measured using a sphygmomanometer.

The i.o.p., heart rate and systolic arterial pressure 
were recorded initially with the patient awake. These, 
together with c.v.p. measurements, were repeated 10 
and 15 min after the injection of thiopentone. Stable 
conditions were produced in all patients within 15 
min of the induction of anaesthesia.

Thereafter, either enflurane 1% or halothane 
0.5% vapour was added to the inspired gas mixture 
and i.o.p., heart rate, systolic arterial pressure and 
c.v.p. were recorded at 5, 10 and 15 min following the 
introduction of the volatile anaesthetic. Stable 
conditions were achieved again in every patient 
within 15 min.

After completion of the study surgery was com-
menced on the traumatized eye. At the conclusion of 
the operation, residual neuromuscular blockade was 
antagonized with neostigmine 3.75 mg, i.v. preceded 
by atropine 1.2 mg.

The results were analysed using a Student’s paired 
t test for significant difference between two sample 
means, computed on a Hewlett-Packard (9815 A) 
desk calculator.

RESULTS
The two groups of patients studied were similar with 
respect to age (halothane group 29.2 yr ± 7.7 (mean 
± SD), enflurane 27.8 yr ± 7.3) and body weight 
(halothane group 55.6 kg ± 13.9, enflurane 61.0 kg 
± 11.2).

Induction of anaesthesia and controlled ventilation 
decreased i.o.p. in both groups, but the changes were 
statistically significant only in group 1 (figs 1, 2). The 
addition of 0.5% halothane to the inspired gas 
mixture produced, after 15 min, a further but 
insignificant 14% decrease (0.19 kPa) in the mean 
i.o.p., whereas 15 min of nitrous oxide and enflurane 
anesthesia reduced mean i.o.p. by 0.48 kPa (40% 
decrease; P<0.01).

The mean heart rate before anaesthesia was 
slightly greater in group 2, a pattern which persisted 
throughout the study. Heart rate increased in both 
groups following induction of anaesthesia, but the 
change was significant only in group 1. The addition 
of a volatile anaesthetic to the inspired gas reduced 
heart rate significantly in both groups (figs 1, 2).

Mean arterial pressure changed little following 
induction of anaesthesia, but both halothane and 
enflurane caused significant decreases in mean arterial 
pressure (P<0.05 and P<0.01 respectively).

Central venous pressure was unchanged throughout 
anesthesia in both groups.
**DISCUSSION**

The effects of halothane on i.o.p. are well documented (Al Abrak and Samuel, 1974a), but similar studies with enfurane are limited in their scope. A previous investigation of enfurane (Radtke and Waldman, 1975) failed to standardize the various factors which influence i.o.p. during general anaesthesia. These include alterations in arterial carbon dioxide tension, inspired oxygen concentration, arterial and central venous pressures and the premedication and anaesthetic technique used (Al Abrak and Samuel 1974a; Duncalf, 1975; Macdiarmid and Holloway, 1976).

The present study followed the design of that by Al Abrak and Samuel (1974a) and utilized pancuronium which is known not to influence i.o.p. significantly (Al Abrak and Samuel, 1974b), or cardiovascular haemodynamics (Coleman et al., 1972). Applanation tonometry was used to measure i.o.p. as this technique has been found to be both accurate and reliable in previous studies of i.o.p. changes during general anaesthesia (Al Abrak and Samuel, 1975; Radtke and Waldman, 1975).

Radtke and Waldman (1975), in a study conducted on young, healthy patients breathing 3–5% enfurane spontaneously, reported a significant decrease in mean i.o.p. of 4 mm Hg (0.53 kPa). In this investigation, controlled ventilation with 1% enfurane reduced i.o.p. in every patient of group 2, thus resulting in a significant decrease in mean i.o.p. of 0.48 kPa ($P<0.01$). In contrast, halothane failed to alter mean i.o.p. significantly (group 1) and produced varying effects in individual patients (i.o.p. increased in two, did not alter in three and decreased in five patients). The degree of change in i.o.p. induced by enfurane was thus greater than that caused by an equipotent concentration of halothane (approximately 0.6 m.a.c. in oxygen).

The possible mechanisms whereby anaesthetic drugs reduce i.o.p. under controlled conditions include relaxation of intra-ocular and extra-ocular muscles, facilitation of the flow of aqueous humour, or decrease in production of aqueous humour (Duncalf, 1975). Both halothane and especially enfurane actively inhibit neuromuscular conduction. Furthermore, all inhalation anaesthetic agents are alleged to reduce i.o.p. by facilitation of aqueous flow (Duncalf, 1975), and enfurane may be expected to possess a similar action.

Before drawing any conclusions from this study, we should comment on certain findings. The initial decrease in i.o.p. from pre-induction control values before the administration of the volatile agent was greater in group 1 than in group 2. However, mean i.o.p. 15 min after the introduction of the volatile agent was significantly less ($P<0.005$) in group 2 (enfurane) compared with group 1 (halothane), despite similar i.o.p. values before the induction of anaesthesia. Other factors which may have influenced our results include the presence of a slightly greater mean systolic arterial pressure during anaesthesia in the halothane group, but heart rates were faster in the patients receiving enfurane.

Finally, the exact concentrations of halothane and enfurane delivered by their respective vaporizers were not measured. The vaporizers used were relatively new and recently calibrated, but their accuracy during anaesthesia was not established (Al Abrak and Samuel, 1974a).

However, we can conclude from this study that enfurane 1%, in combination with nitrous oxide,
oxygen, muscle relaxation and controlled mechanical ventilation, decreases i.o.p. significantly. Enflurane appears to offer a reasonable alternative to halothane for use in ophthalmic anaesthesia.

REFERENCES


VARIATIONS DANS LA PRESSION INTRAOCULAIRE PENDEANT UNE ANESTHESIE A L’HALOTHANE OU A L’ENFLURANE

**RESUME**

Nous avons mesuré la pression intraoculaire (IOP) de 20 malades anesthésiés à l’ aide d’halothane ou d’enflurane (0.6 MAC dans l’oxygène) pour réfection chirurgicale d’un traumatisme de l’œil. Les variations dans l’IOP avec l’halothane à 0,5% ont été imprévisibles, alors qu’avec l’enflurane à 1% on a réduit d’une manière continue la tension intraoculaire sur tous les malades que l’on a observés. L’enflurane est recommandée comme alternative anesthésique possible pour la chirurgie sur l’oeil ouvert.

INTRAOKULARE DRUCKVERÄNDERUNGEN WAHREND HALOTHAN- UND ENFLURANNARKOSE

**ZUSAMMENFASSUNG**

Der intraokulare Druck (i.o.p.) wurde gemessen bei 20 Patienten, die zwecks Beseitigung eines Augenartus mit Halothan oder mit Enfluran (0,6 MAC in Sauerstoff) narkotisiert wurden. Die i.o.p.-Veränderungen bei 0,5% Halothan waren unberechenbar, während bei Enfluran (1%) die intraokulare Spannung konsistent bei allen beobachteten Patienten verringert wurde. Enfluran wird als alternatives Narkotikum für chirurgische Eingriffe im Auge empfohlen.

Cambios en la presión intraocular durante anestesia de halotano y enflurano

**SUMARIO**

Hemos medido la presión intraocular (i.o.p.) en 20 pacientes anestesiados con halotano o enflurano (0,6 MAC en oxígeno) para la reparación de trauma al ojo. Los cambios en la i.o.p. con halotano 0,5% fueron impredecibles mientras que el enflurano 1% redujo la tensión intraocular consistentemente en todos los pacientes estudiados. Se recomienda el enflurano como una posible anestesia alternativa para cirugía de ojo abierto.