Effects of general anaesthesia on the intraocular pressure in man

Trichloroethylene in nitrous oxide and oxygen

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The intraocular pressure (IOP) is often measured under general anaesthesia in children and mentally defective adults. The most commonly used agent (halothane) has been shown to cause a marked fall in IOP (Al-Abrak and Samuel, 1974). It was therefore thought desirable to search for an anaesthetic agent which does not affect the IOP.

There are only two reports on the effect of trichloroethylene (TCE) on the IOP. Magora and Collins (1962) studied TCE among other non-explosive anaesthetics in man and they reported a fall in IOP with TCE and other anaesthetics that was proportional to the level of anaesthesia. However the IOP is affected by other factors that were not standardized in their study, notably carbon dioxide (Duncalf and Weitzner, 1963; Beaugié and Samuel, 1973), blood pressure (Adams and Barnett, 1966), and central venous pressure (CVP) (Duke-Elder, 1970). On the other hand Schreuder and Linssen (1972) studied the effect of TCE on the IOP in monkeys by direct needling of the anterior chamber and they reported a rise in IOP with TCE. However, animal experiments may not be applicable to man (Grant, 1969).

It was therefore decided to study the effect of TCE on the human IOP under anaesthesia while excluding other possible causes of change in IOP.

The IOP was measured with the Perkins hand-held applanation tonometer which can be used with the patient in the supine position (Perkins, 1965). The choice of this instrument was based on the fact that it uses the principle of applanation which is more accurate than indentation tonometry used in previous studies (Kaufman, 1972).

Material and methods

Eight otherwise healthy urological patients (all males aged 20–73 yrs: mean 52.38 ± 19.67) were studied before

surgery. They were not taking drugs known to affect the IOP. After the nature of the study had been explained, written consent was obtained from each patient.

The patients were not premedicated, and the methods were similar to those used in a previous study (Beaugié and Samuel, 1973). Anaesthesia was induced with thiopentone 5 mg./kg., pancuronium 0.1 mg./kg. and pethidine 0.5 mg./kg. The larynx was sprayed with 100 mg. lignocaine and a cuffed endotracheal tube was introduced.

The respiratory and cardiovascular status was stabilized by the method of Samuel, Grange, and Hawkins (1968). Pancuronium was substituted for tubocurarine since it does not affect the IOP (Al-Abrak and Samuel, 1974) and has fewer cardiovascular side-effects (Baird, 1968; Stoetling, 1972; Feldman, 1973). Respiration was controlled with a Manley ventilator set at constant pressure with a constant gas flow of 15 l./min. (nitrous oxide 10 l./min. and oxygen 5 l./min.) to minimize the effects of changes in intrathoracic pressure on the circulation. The patients lay supine to avoid postural changes in the intraocular and orbital venous pressures.

The end-tidal carbon dioxide concentration, which mirrors arterial carbon dioxide (PaCO_2) (Collier, Affeldt, and Farr, 1955), was maintained at 5 per cent. by adding carbon dioxide to the inspired gases as necessary. A corresponding volume was reduced from the nitrous oxide flow rate to keep the minute volume and the inspired oxygen concentration constant.

TCE was vaporized in a Tritec vaporizer and 1 per cent. was administered for 15 min. after cardiovascular stability has been achieved for at least 15 min. from induction of anaesthesia.

The end-tidal gases were analysed by continuous sampling from the trachea at the carina by a nylon catheter. The sample was analysed for carbon dioxide and oxygen by a Uras III infrared carbon dioxide analyser and a paramagnetic oxygen analyser. The end-tidal oxygen concentration was stable at 34 per cent.

In all patients a local anaesthetic (lignocaine 0.5 per cent.) was instilled into the conjunctival sac and the IOP measured before induction of anaesthesia. Further readings, together with the patient's pulse, blood pressure, and CVP were taken after induction of anaesthesia, after a 15 min. period of administration of TCE, and 15 min. after cessation of administration of TCE.

At the conclusion of the study, chloramphenicol ointment was applied to the eye.

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Results

These are shown in the Table. The figures in brackets are the percentage changes from the observations made after induction and stabilization of anaesthesia and before the administration of TCE. In every patient there was a rise in IOP when 1 per cent. TCE was administered. When the administration of TCE was discontinued, the IOP returned near to its original value. Thus each patient acted as his own control.

The typical changes in one patient are shown in the Figure. The mean IOP after administration of TCE was 17.75 ± 2.82 . The mean percentage rise in IOP after administration of TCE was 31.38 ± 8.27 , and it was statistically highly significant ($P < 0.001$) when subjected to Student's *t*-test. After cessation of administration of TCE for 15 min., the IOP returned to its initial level. The mean IOP was 14 ± 2.56 . This corresponded to a mean percentage change of 3.21 ± 5.36 which was not significant ($P < 0.2$).

The changes in blood pressure during the administration of 1 per cent. TCE were not statistically significant ($P < 0.3$).

In every patient the CVP was observed to rise markedly during the administration of TCE. The mean pressure rose from 10.25 to 17.25 cm. saline. The mean percentage increase in CVP in the whole group after 15 min. administration of TCE was 68.35 ± 32.86 which was statistically highly significant ($P < 0.001$). Within 15 min. of cessation of TCE administration, the CVP fell back to near its initial

FIGURE Effect of trichlorethylene on the intraocular pressure, central venous pressure, systolic blood pressure, and heart rate

Table Changes in intraocular pressure, blood pressure, heart rate, and central venous pressure after administration of TCE anaesthesia in 8 cases

Patient no.	Awake	I (after induction and stabilization of anaesthesia)				II (15 min. after 1 per cent. TCE)				III 15 min. after cessation of TCE)			
		IOP	IOP	BP	HR	CVP	IOP	BP	HR	CVP	IOP	BP	HR
1	11	10	130	94	8	13 (+30)	130 (0)	98 (+4.26)	12 (+50)	11 (+10)	130 (0)	98 (+4.26)	8 (0)
2	16	15	115	64	7.5	19 (+26.67)	120 (+4.35)	70 (+9.38)	12 (+60)	16 (+6.67)	120 (+4.35)	68 (+6.25)	9 (+20)
3	17	16	135	88	11	20 (+25)	135 (0)	88 (0)	15 (+36.36)	17 (+6.25)	130 (-3.7)	84 (-4.55)	10.5 (-4.55)
4	12	12	120	80	12	17 (+41.67)	125 (+4.17)	76 (-5)	18 (+50)	13 (+8.33)	130 (+8.33)	76 (-5)	12 (0)
5	12	11	120	88	9	16 (+45.45)	120 (0)	100 (+13.64)	19 (+111.11)	11 (0)	125 (+4.17)	84 (-4.55)	9 (0)
6	16	15	140	72	12	19 (+26.67)	140 (0)	68 (-5.56)	16 (+33.33)	15 (0)	130 (-7.14)	68 (-5.56)	12 (0)
7	12	12	110	84	10	16 (+33.33)	110 (0)	72 (-11.29)	19 (+90)	12 (0)	120 (+9.09)	68 (-19.05)	12 (+20)
8	18	18	120	64	12.5	22 (+22.22)	150 (+25)	104 (+62.5)	27 (+116)	17 (-5.56)	120 (0)	72 (+12.5)	17 (+36.0)
Mean level ±S.D.	14.25 ±2.76	13.63 ±2.77	123.75 ±10.26	79.25 ±11.41	10.25 ±1.93	17.75 ±2.82	128.75 ±12.75	84.5 ±14.76	17.25 ±4.83	14.00 ±2.56	125.63 ±4.96	77.25 ±10.74	11.19 ±2.83
Mean change (%) ±S.D.						+31.38 ±8.27	+4.19 ±8.63	+8.49 ±23.31	+68.35 ±32.86	+3.21 ±5.36	+1.89 ±5.66	-1.96 ±9.58	+8.93 ±14.53
Probability						<0.001	<0.3	<0.4	<0.001	<0.2	<0.4	<0.6	<0.2

IOP = Intraocular pressure (mm.Hg)

BP = Systolic arterial blood pressure (mm.Hg)

HR = Heart rate (beats per min.)

CVP = Central venous pressure

level. The mean was 11.19 cm. saline. This corresponded to a mean percentage change of 8.9 which is not significant statistically ($P < 0.2$).

All the patients had normal IOP before induction of general anaesthesia. The mean value was 14.25 ± 2.76 mm. Hg, which is within the normal range of 10.5 to 20.5 quoted by Leydhecker, Akiyama, and Neumann (1958). Although the IOP rose significantly after administration of TCE, the mean value was still within this range.

Discussion

The rationale of the method was discussed in a previous paper (Beaugié and Samuel, 1973). The present observation that the administration of TCE causes a rise in IOP does not agree with that of Magora and Collins (1962) who claimed a fall. However, they made no attempt to control other factors that affect the IOP. They gave heavy premedication to the group of patients studied for TCE and it is known that premedication affects the IOP (Leopold and Comroe, 1948). They did not control end-tidal carbon dioxide or measure arterial carbon dioxide tension, and it is known that changes in carbon dioxide concentration profoundly affect the IOP. In addition, they measured IOP with a Schiötz tonometer, which uses the principle of indentation tonometry and is not accurate enough either for clinical use or for research (Duke-Elder, 1970; Kaufman, 1972).

On the other hand, our results support those of Schreuder and Linssen (1972), who reported a rise in IOP in monkeys after the administration of TCE. They measured IOP by direct needling of the anterior chamber. They measured end-tidal carbon dioxide, CVP, and arterial blood pressure. These authors, however, reported that the CVP did not change during the administration of TCE. However, they administered only 0.5 per cent. TCE from an inaccurate vaporizer, and it is known that the pulmonary circulation in man may react to drugs differently and even in an opposite way to animals (Aviado, 1960).

The cause of the rise in IOP after administration of TCE is probably due to the marked rise in CVP associated with administration of TCE, since it is known that the IOP is affected by changes in CVP (Duke-Elder, 1970).

Ellis (1966) found more loss of blood during extraocular surgery under TCE than under halothane anaesthesia. The underlying cause may be this marked rise in CVP which we have observed with TCE. In a previous study of the effect of halothane on IOP in man, it was noted that the mean rise in CVP was not more than 1 cm. saline which corresponded to a mean percentage rise of 11 per cent. (Al-Abrak and Samuel, 1974).

Jennett, Barker, Fitch, and McDowall (1969) reported that TCE causes a rise in intracranial pressure even when the arterial carbon dioxide is low, and that this rise is associated with cerebral vasodilatation and increased cerebral blood flow. It might be assumed that the IOP would be similarly affected. However, Duke-Elder (1970) stated that "vasodilatation does not necessarily lead to an increased IOP, the contrary is very frequently the case". This we also observed in a previous study of the effect of halothane on IOP in which we demonstrated a marked fall in IOP with halothane, in spite of its marked vasodilating properties and in spite of the rise in intracranial pressure which is known to accompany halothane anaesthesia (McDowall, Barker, and Jennett, 1966).

This study was planned to stabilize all other possible factors that affect the IOP. To achieve this neither premedication nor depolarizing relaxants used. Changes in intrathoracic pressure were minimized by keeping the minute volume and inflation pressure constant. The end-tidal oxygen concentration was maintained at 34 per cent. to avoid possible effects of changes in oxygen tension on the blood vessels of the eye, and on the pulmonary circulation and the CVP. The patients were studied before surgery and kept in the supine position so that postural changes did not affect the orbital and central venous pressures. The end-tidal carbon dioxide was maintained at 5 per cent. as described above. The blood pressure did not alter significantly.

The CVP changed only during the administration of TCE and was constant and stable before and after its administration.

From this study it appears that TCE is not a suitable anaesthetic for intraocular surgery or for the glaucomatous patient. However, although TCE caused a statistically highly significant rise in every patient, the mean IOP for the whole group during the administration of TCE remained within the accepted normal range quoted by Leydhecker and others (1958). Thus TCE is probably a better anaesthetic for the measurement of IOP in children than agents such as halothane which cause a marked fall in IOP.

Summary

The effect of TCE on the intraocular pressure was investigated in eight patients under thiopentone nitrous oxide/oxygen anaesthesia, using a muscle relaxant (pancuronium bromide). Pulmonary ventilation was controlled and the end-tidal carbon dioxide was maintained at 5 per cent. In every patient the intraocular pressure was observed to rise as TCE was administered and then to return to the initial level when the administration of TCE ceased.

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