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EFFECTS OF NEBULIZED LIGNOCAINE ON THE INTRAOCULAR PRESSURE RESPONSES TO TRACHEAL INTUBATION

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SUMMARY

We have examined the effect of preoperative administration of nebulized lignocaine or saline on the intraocular pressure (IOP) response to tracheal intubation in 20 adults. In the saline group, tracheal intubation was associated with a significant increase in IOP above control and preintubation values ($P < 0.01$); in the lignocaine group there was no change in IOP following intubation. After intubation, IOP was significantly less in the lignocaine group than in the saline group ($P < 0.05$).

KEY WORDS

Eye: intraocular pressure. Anaesthetics local: lignocaine.

Laryngoscopy and tracheal intubation induce an increase in arterial pressure and IOP [1]. The administration of nebulized lignocaine before induction of anaesthesia has been shown to reduce the pressor response to tracheal intubation [2–4], but the effect of this manoeuvre on IOP has not been studied. We have examined, therefore, the influence of nebulized lignocaine on the haemodynamic and IOP changes caused by tracheal intubation.

METHODS AND RESULTS

The study was approved by the District Medical Ethics Committee. Informed consent was obtained from 20 ASA class I and II patients (12 male) undergoing routine ophthalmic surgery. They were allocated randomly to receive either saline ($n = 10$) or lignocaine 6 mg kg^{-1} ($n = 10$). All patients were premedicated with oral diazepam. In the anaesthetic room, each patient was given 0.15 ml kg^{-1} (maximum dose 10 ml) of the test solution using an Acorn nebulizer (part No. 1049, Medic-Aid, U.K.). Anaesthesia was induced with thiopentone 5 mg kg^{-1} followed by atracurium 0.6 mg kg^{-1} to facilitate tracheal intubation. Anaesthesia was maintained by controlled ventilation with 65% nitrous oxide in oxygen via a Bain system. End-tidal carbon dioxide concentration was maintained at 5 ± 0.1% (ventilatory frequency about 12 b.p.m.) and tidal volume 10 ml kg^{-1} (monitored by infra-red carbon dioxide analyser and Wright's respirometer, respectively). After the study period, 1% enflurane was added and droperidol 2.5–5 mg given i.v. Residual neuromuscular block was antagonized with neostigmine and glycopyrrolate 50 and 10 μg kg^{-1}, respectively.

Heart rate and arterial pressure were monitored by electrocardiogram and automatic pressure device, respectively. IOP was measured in the unoperated normal eye by a Perkins applanation tonometer. Heart rate, arterial pressure and IOP were measured before nebulization of the test solution, after nebulization, before intubation, immediately after intubation, 2.5 and 5 min thereafter (table I). Venous blood samples were obtained at corresponding times and at 10 and 20 min after nebulization for measurement of plasma concentrations of lignocaine using high pressure liquid chromatography. Parametric data were analysed within and between groups by

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TABLE I. *Mean (SD) values of IOP, arterial pressure and heart rate. Significant change from *control and †preintubaton (P < 0.05–0.001). 1 = Before nebulization of test solution; 2 = after nebulization; 3 = before intubation; 4 = immediately after intubation; 5 = 2.5 min after intubation; 6 = 5 min after intubation*

	1	2	3	4	5	6
IOP (mm Hg)						
Saline	11.3	11.2	8*	19.3*†	15.2*†	13†
	(3.5)	(2.5)	(3.6)	(6)	(4.5)	(4.9)
Lignocaine	12.9	11.6*	10.6*	10.4	7.1*	6*†
	(2.8)	(2.8)	(4.4)	(4.9)	(3.5)	(3.3)
SAP (mm Hg)						
Saline	147.2	149.6	143	190*†	162*	143.6
	(20.5)	(20.5)	(32.6)	(29.7)	(26.1)	(31.5)
Lignocaine	165.5	160.8	142.4*	154.3	145.6	129.5*
	(40.7)	(36.3)	(36.3)	(36.3)	(28.9)	(23.1)
DAP (mm Hg)						
Saline	85.4	84.3	86.1	110*†	96.8	85.6
	(13.7)	(12.7)	(20.7)	(23.1)	(17.1)	(15.7)
Lignocaine	98.6	97.7	86.9	99.9†	84.6*	80.4*
	(17.9)	(19.7)	(13)	(15.3)	(7.41)	(9.5)
HR (beat min^{-1})						
Saline	73.8	68.1	87.4*	99.9*†	93.1*	86.1*
	(6.3)	(6.4)	(17.2)	(21)	(15.4)	(16.9)
Lignocaine	81.9	84.1	96*	94.8*	83	80.4
	(10.8)	(15.4)	(12.4)	(11.4)	(15.2)	(15.3)

paired and unpaired Student's *t* test, respectively, and non-parametric data by the Mann–Whitney *U* test.

There were no significant differences in mean prenebulization values between the saline and lignocaine groups for age (70 and 62 yr, respectively), weight (65 and 70 kg) or sex distribution of patients (table I). Following intubation in the saline group, there were increases in IOP, heart rate and arterial pressures to values significantly greater than control ($P < 0.01$) and preintubation values ($P < 0.05$). In comparison, post-intubation IOP, heart rate and systolic pressures in the lignocaine group were not significantly different from control or preintubation values; indeed, IOP was significantly less than control. Although heart rate increased significantly above control following induction ($P < 0.01$), post- and pre-intubation values were not significantly different. Only a small, albeit significant ($P < 0.05$) increase in diastolic pressure was noted, immediately after intubation.

Following intubation, all IOP values in patients who received lignocaine were significantly lower than those given saline ($P < 0.001$). Post-intubation systolic and diastolic pressures in the lignocaine group remained less than those in the saline group immediately after intubation and at 2.5 min thereafter ($P < 0.05$). Mean peak plasma concentration of lignocaine after nebulization was 1.07 (SD 0.53) μg ml^{-1}, decreasing to 0.56 (0.21) μg ml^{-1} at the end of the period of study.

COMMENT

We have demonstrated that tracheal intubation produced a significant increase in IOP and haemodynamic state, confirming previous studies [1]. Several techniques have been used to modify the pressor response to intubation [5], but none is completely successful.

Previous studies [2, 3] have demonstrated that administration of nebulized lignocaine before operation did not abolish the pressor response to tracheal intubation. Our lignocaine group demonstrated a more stable haemodynamic response. This difference may be a reflection of the dose of lignocaine used and our technique of administration. Clay and Clarke [6] have reported that, during nebulization, 15% of the first part of a dose is deposited in the body and only 7–12% reaches the airways. Previous studies with nebulization involved smaller doses of lignocaine than that used here, a face mask or a short period of nebulization. Despite our use of a larger dose

and more efficient method of administration, the plasma concentrations were lower than toxic values (5–9 $\mu g\ ml^{-1}$). The greatest concentration recorded was 2.3 $\mu g\ ml^{-1}$, similar to those reported in other studies [3, 4].

Transient changes in IOP are probably of little importance in healthy individuals. However, such changes may be hazardous in patients with a lacerated globe or glaucoma. None of the studies involving lignocaine nebulization examined changes in IOP. In our study, mean IOP increased substantially above control after intubation in the control group, but there was no change in the group given lignocaine. A slight decrease in arterial pressure, caused possibly by lignocaine, was noted following nebulization and may account for the decrease in IOP after nebulization. However, this may have been caused by oxygen, which was used as the sole gas to nebulize lignocaine [5]. Reduction in IOP following induction and intubation may have resulted from the effects of thiopentone or absence of reflex pressor responses to the stimulus of intubation as a result of local anaesthesia of the airway.

Several factors affect IOP during anaesthesia. In our study, excessive inflation pressure was avoided, normocapnia and normoxia were maintained and the patient remained supine to prevent hydrostatic changes in IOP.

In a previous study [3] a mixture of viscous and aqueous lignocaine was administered via a modified Bird nebulizer. This technique is not suitable for routine use, and inhalation of viscous lignocaine may cause obstruction of small airways, hence it cannot be recommended at present. Our nebulization technique is simple, but acceptability to patients demands full explanation and reassurance regarding loss of pharyngeal reflexes.

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