Intravenous lignocaine pretreatment to prevent intraocular pressure rise following suxamethonium and tracheal intubation

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SUMMARY Intravenous lignocaine (1.5 mg kg⁻¹) was evaluated in patients undergoing intraocular surgery as a means of preventing the rise in intraocular pressure which accompanies tracheal intubation. In patients given either suxamethonium or pancuronium to facilitate tracheal intubation, lignocaine pretreatment conferred no benefit over placebo in preventing the intraocular hypertensive response.

Laryngoscopy and tracheal intubation induce potent cardiovascular pressor responses which may cause myocardial ischaemia and cardiac arrhythmias in susceptible patients. They also cause a significant increase in intracranial and intraocular pressures, which may have serious consequences in certain patients. Intravenous lignocaine has been used with some success in attenuating these responses. Hamill et al.4 found that intravenous lignocaine (1.5 mg kg⁻¹) reduced but did not completely abolish the haemodynamic effects of laryngoscopy and intubation. Donegan and Bedford5 found that intracranial pressure (ICP) fell following intravenous lignocaine and that the rise in ICP accompanying tracheal suctioning was also attenuated. Poulton and James6 found that intravenous lignocaine in a dosage of 1.5 mg kg⁻¹ could significantly suppress the cough reflex.

Little work has been done in assessing intravenous lignocaine for prevention of intraocular hypertension. A recent review of the effects of anaesthesia on intraocular pressure6 showed that no presently used method of preventing suxamethonium-induced intraocular hypertension was completely effective. In view of the success of intravenous lignocaine in attenuating the haemodynamic responses and ICP rise accompanying tracheal intubation it seemed worthwhile to assess its efficacy in preventing the rise in intraocular pressure which also accompanies this manoeuvre.

The aim of the present study was to evaluate the effect of lignocaine pretreatment on suxamethonium induced intraocular hypertension and its effect on intraocular pressure changes following tracheal intubation.

Patients and methods

Adult patients giving informed consent and undergoing cataract surgery under general anaesthesia were included prospectively into the study. Premedication was with oral diazepam 10 mg, given 90 minutes prior to surgery.

The patients were grouped by random number sequence into those who received suxamethonium (1.5 mg kg⁻¹) and those who received pancuronium (0.1 mg kg⁻¹) for muscle relaxation and subgrouped into those who received intravenous lignocaine (1.5 mg kg⁻¹) pretreatment and those who received placebo.

The anaesthesia sequence for patients given suxamethonium involved administration of lignocaine or placebo followed one minute later by thiopentone (4-5 mg kg⁻¹) and then suxamethonium (1.5 mg kg⁻¹) one minute after completion of thiopentone injection. Tracheal intubation was performed one minute after suxamethonium injection. In these patients intraocular pressure was measured just before suxamethonium administration, just before laryngoscopy, and one minute after tracheal intubation.

The anaesthesia sequence for patients given pancuronium involved administration of lignocaine or placebo followed one minute later by thiopentone
(4-5 mg kg⁻¹) and pancuronium. As spontaneous breathing ceased, ventilation was controlled to maintain normocapnia (Siemens Elma 130 Infrared CO₂ Monitor) with 70% nitrous oxide in oxygen via face mask. Five minutes later tracheal intubation was performed. In these patients intraocular pressure was measured just before laryngoscopy and one minute after tracheal intubation.

After the last measurement was made a volatile anaesthetic agent was introduced to maintain anaesthesia, and ventilation was controlled in all cases. Pancuronium was given if necessary to the patients who had been given suxamethonium only after this time. No patient received opiates or beta blockers, and local anaesthetic spray to the larynx was not used.

Measurements of intraocular pressure were made in the eye not undergoing surgery with a Digilab 30RT Pneuma-Tonometer/Tonographer, which was calibrated before and rechecked after each set of measurements. This instrument, designed exclusively for intraocular pressure measurement and using a pneumatic sensor, operates on the principle of applanation tonometry and records intraocular pressure continuously in mmHg, using a freely moving pen recorder and calibrated paper strip. Values for intraocular pressure for all groups were compared by Student’s t tests.

At the same time as intraocular pressure measurements were performed arterial blood pressure and heart rate were measured with a Vita-Stat 901-S blood pressure monitor.

**Results**

A total of 40 patients were studied, 10 in each subgroup. No significant differences between the subgroups were seen with regard to age, sex, and weight (Table 1).

In the suxamethonium subgroups the change in IOP (mean 0.35 mmHg±2.68 SD), following suxamethonium administration failed to reach statistical significance. However, in these subgroups the IOP was significantly increased (mean 4.05 mmHg±3.145 SD, p<0.001) following tracheal intubation (Table 2).

In the pancuronium subgroups a similar significant increase in IOP followed tracheal intubation (mean 5.4 mmHg±4.67 SD, p<0.001). The systemic arterial pressure and heart rate increased in parallel with the IOP in all cases (Table 3).

In both the suxamethonium subgroups and the

### Table 1 Demographic data for patient subgroups (mean±standard deviation)

<table>
<thead>
<tr>
<th></th>
<th>Suxamethonium subgroups</th>
<th>Pancuronium subgroups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Placebo</td>
<td>Lignocaine</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
<td>Lignocaine</td>
</tr>
<tr>
<td>Age</td>
<td>72.8±11.8</td>
<td>70.8±10.7</td>
</tr>
<tr>
<td>Sex M:F</td>
<td>3:7</td>
<td>2:8</td>
</tr>
<tr>
<td>Weight</td>
<td>61.2±5.2</td>
<td>59.0±8.0</td>
</tr>
<tr>
<td></td>
<td>73.7±6.3</td>
<td>66.2±10.7</td>
</tr>
<tr>
<td></td>
<td>4:6</td>
<td>2:8</td>
</tr>
<tr>
<td></td>
<td>62.9±10.5</td>
<td>67.9±12.7</td>
</tr>
</tbody>
</table>

### Table 2 Intraocular pressure (mmHg), blood pressure (mmHg), and heart rate (/min) with and without lignocaine pretreatment. Mean (standard deviation)

<table>
<thead>
<tr>
<th></th>
<th>Presuxamethonium</th>
<th>Preintubation</th>
<th>Postintubation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Placebo</td>
<td>Lignocaine</td>
<td>Placebo</td>
</tr>
<tr>
<td>Intraocular pressure</td>
<td>16.2 (5.0)</td>
<td>19.1 (5.4)</td>
<td>16.9 (4.7)</td>
</tr>
<tr>
<td>Systolic arterial pressure</td>
<td>134.8 (14.7)</td>
<td>135.0 (17.9)</td>
<td>143.0 (19.0)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>79.6 (8.8)</td>
<td>80.9 (9.4)</td>
<td>82.0 (10.7)</td>
</tr>
</tbody>
</table>

### Table 3 Intraocular pressure (mmHg), arterial pressure (mmHg), and heart rate (/min) with and without lignocaine pretreatment. Mean (standard deviation)

<table>
<thead>
<tr>
<th></th>
<th>Preintubation</th>
<th>Postintubation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Placebo</td>
<td>Lignocaine</td>
</tr>
<tr>
<td>Intraocular pressure</td>
<td>18.7 (7.9)</td>
<td>14.8 (3.5)</td>
</tr>
<tr>
<td>Systolic arterial pressure</td>
<td>131.5 (27)</td>
<td>146.8 (18.3)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>86.0 (22.9)</td>
<td>81.5 (16.9)</td>
</tr>
</tbody>
</table>
pancuronium subgroups lignocaine pretreatment failed to have any effect on the rise in IOP accompanying tracheal intubation (p > 0.05).

**Discussion**

With improved anaesthetic agents and techniques, general anaesthesia is used increasingly in ophthalmic surgery in an attempt to optimise conditions for intraocular surgery. Controlled hyperventilation significantly reduces intraocular pressure (IOP) but is preceded by a significant rise in IOP due to tracheal intubation. In most patients intraocular hypertension following tracheal intubation is of short duration and is inconsequential. In patients undergoing cataract surgery the IOP is likely to have returned to normal owing to controlled hyperventilation before the eye is opened. In patients with perforating eye injuries, however, any rise in intraocular pressure irrespective of duration may be hazardous, as it may precipitate extrusion of intraocular contents and may further endanger sight in that eye. For this reason also the administration of suxamethonium to patients with perforating eye injuries is controversial, since it is known to increase intraocular pressure.

Many drugs have been used in an attempt to obtund the undesirable effects of suxamethonium and tracheal intubation, including small doses of non-depolarising neuromuscular blocking drugs, 'self-taming' with small doses of suxamethonium and with preinduction doses of intravenous diazepam, hexafluorenium, and acetazolamide. No method of pretreatment has been found to be completely satisfactory. A review of the literature showed that, though intravenous lignocaine pretreatment has been the subject of a number of studies, only one evaluated its effect on intraocular pressure. Smith et al.1 used 1 or 2 mg kg⁻¹ of intravenous lignocaine and found it to be 'ineffective in preventing the rise in intraocular pressure following succinylcholine and/or tracheal intubation.'

In the present study, lignocaine pretreatment was not found to have any effect on IOP following suxamethonium. It may be inferred that this would also be the case in patients with perforating eye injuries.

It is a reasonable criticism of this study that tracheal intubation was performed under light (thiopentone) anaesthesia and that resultant haemodynamic responses may have contributed to the changes in IOP observed. Since this technique is likely to compare with that used to anaesthetise a patient with a post-traumatic eye injury, however, this is most likely to compare with relevant conditions.

Of greater significance was the fact that changes in IOP following suxamethonium were unimportant compared with the major changes in IOP that occurred with tracheal intubation. Much has been written about the potentially hazardous use of suxamethonium but little about the dangers of responses to intubation in patients with eye injuries. It would seem from the present study that the rise in IOP from tracheal intubation is of such magnitude as to render the suxamethonium effect unimportant. The aim of future studies in our opinion should be directed at inhibiting responses to intubation rather than suxamethonium. Clearly intravenous lignocaine pretreatment is not the answer.

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**References**


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