EFFECT OF VECURONIUM ON INTRAOCULAR PRESSURE*

J.-P. JANTZEN, G. H. HACKETT, K. ERDMANN AND G. EARNSHAW

Intraocular pressure (IOP) is a variable which varies minimally from its physiological value because, some believe, of the existence of a control centre in the hypothalamus (Schmerl and Steinberg, 1948; v. Sallman et al., 1956). The factors which regulate IOP include aqueous humour balance, and the values of arterial and venous pressures. While there is an almost direct correlation between IOP and venous pressure (Macri, 1961), the relationship between IOP and arterial pressure is qualitative rather than quantitative (Adams and Barnett, 1966). A transient increase in the arterial pressure results in an increase in IOP (Langrehr, Adelstein and L’Allemand, 1967), but persistent hypertension is rapidly counteracted by an autoregulatory adjustment of the aqueous humour balance (Heilemann, 1975).

Factors increasing intraocular pressure are hypoxia and hypercarbia, light anaesthesia, an increase in CVP and the administration of drugs such as suxamethonium, propanidid and atropine. The factors which decrease IOP include inspiration and hypotension. Volatile anaesthetic agents, barbiturates and narcotics will also induce a decrease in IOP.

In patients undergoing operations on the open eye, or those suffering from a perforating eye injury or glaucoma, those anaesthetic drugs which increase IOP should be avoided. As neither precurarization (Jansen and Hansen, 1979), “self-taming” (Myers, Singer and Otto, 1980) nor the administration of higher doses (Cook, 1981), prevent the increase in IOP associated with suxamethonium, there is a need for another rapidly-acting neuromuscular blocking agent. This study was undertaken to assess whether vecuronium could be used in this context.

PATIENTS AND METHODS

Twenty patients (14 male) undergoing major ENT surgery gave informed consent for inclusion in the study. The average age was 50.6 yr (range 19–82 yr) and the average weight was 62.8 kg (range 52–108 kg). Two patients were ASA I, 13 patients ASA II and five patients ASA III. No patient with ophthalmic disease, diabetes mellitus or hypertension was admitted to the study.

Premedication consisted of pethidine 1 mg kg\(^{-1}\) and promethazine 0.5 mg kg\(^{-1}\) i.m. 30–60 min before surgery. Anaesthesia was induced with diazepam 0.1 mg kg\(^{-1}\), fentanyl 0.005 mg kg\(^{-1}\) and etomidate 0.2 mg kg\(^{-1}\). Tracheal intubation was achieved after the administration of vecuronium 0.1 mg kg\(^{-1}\), and the lungs were subsequently ventilated with a volume controlled ventilator (Narkose-Spiromat, 650, Dräger), delivering a 2:1 mixture of nitrous oxide in oxygen at a rate of 10 b.p.m. The initial tidal volume of 10 ml kg\(^{-1}\) was adjusted to obtain an arterial carbon dioxide tension between 4.7 and 5.3 kPa. The airway pressure was monitored by a manometer in the breathing system (Precom, Dräger).

Anaesthesia was maintained with fentanyl 0.1 mg i.v. every 20 min. Intra-arterial pressure was monitored (radial artery) and displayed

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**SUMMARY**

Intraocular pressure decreased by 22.6% in association with neuromuscular blockade produced by vecuronium 0.1 mg kg\(^{-1}\). This appeared to be the result of an indirect action possibly via an effect on CVP. Vecuronium would be a suitable neuromuscular blocker for patients undergoing eye surgery in whom an increase in IOP would be undesirable.
TABLE I. Intra-ocular and central venous pressure in 20/18 patients before and after administration of vecuronium

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Baseline</th>
<th>5 min</th>
<th>10 min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IOP (mm Hg)</td>
<td>CVP (cm H₂O)</td>
<td>IOP (mm Hg)</td>
</tr>
<tr>
<td>1</td>
<td>9</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>14</td>
<td>6.5</td>
<td>11</td>
</tr>
<tr>
<td>3</td>
<td>19</td>
<td>—</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>1.9</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>17</td>
<td>13</td>
<td>19</td>
</tr>
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<td>6</td>
<td>10</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>7</td>
<td>14</td>
<td>7.5</td>
<td>12</td>
</tr>
<tr>
<td>8</td>
<td>14</td>
<td>10</td>
<td>14</td>
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<td>9</td>
<td>17</td>
<td>4</td>
<td>20</td>
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<td>10</td>
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<td>8</td>
<td>3</td>
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<tr>
<td>11</td>
<td>8</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>12</td>
<td>18</td>
<td>14.5</td>
<td>16</td>
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<tr>
<td>13</td>
<td>8</td>
<td>14</td>
<td>9</td>
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<tr>
<td>14</td>
<td>8</td>
<td>—</td>
<td>10</td>
</tr>
<tr>
<td>15</td>
<td>8</td>
<td>4.5</td>
<td>9</td>
</tr>
<tr>
<td>16</td>
<td>14</td>
<td>8.5</td>
<td>9</td>
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<tr>
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<td>18</td>
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<tr>
<td>19</td>
<td>12</td>
<td>15</td>
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<td>20</td>
<td>11</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td>Average</td>
<td>12.2</td>
<td>10.5</td>
<td>10.3</td>
</tr>
<tr>
<td>Average change</td>
<td>—</td>
<td>—</td>
<td>-14.1%</td>
</tr>
</tbody>
</table>

Continuous, together with lead II of the ECG. Central venous pressure was measured (18 patients) via a catheter inserted through the brachial vein. Arterial P<sub>co₂</sub> was measured intermittently. Neuromuscular blockade was assessed by depression of the twitch response to a supramaximal train-of-four stimulus (2 Hz over 2 s). The IOP measurements were performed by the same ophthalmologist using a hand-held Dräger applanation tonometer.

Once the train-of-four ratio had returned to 95% of its initial value, and provided the cardiovascular and ventilatory variables were stable, a baseline measurement of IOP was obtained. A further dose of vecuronium 0.1 mg kg<sup>-1</sup> was given. At 5 and 10 min after the administration of this second dose of vecuronium, IOP was measured again, along with measurements of the cardiovascular variables and blood-gas tensions.

Statistical evaluation was by the Wilcoxon signed ranks test.

RESULTS

Although a small increase in IOP was observed in five patients following the administration of vecuronium, there was a marked decrease in the other 15. The mean IOP at 5 and 10 min were 10.3 mm Hg (SD±4.25) and 9.3 mm Hg (SD±4.57), respectively—values significantly lower (P < 0.05 and < 0.01) than the baseline mean value of 12.2 mm Hg (SD±3.72) (table I, fig. 1).

In all patients, neuromuscular blockade resulted in a slight decrease in the pressure generated by the ventilator. While the average mean arterial pressure and heart rate remained unchanged.
VECURONIUM AND INTRAOCULAR PRESSURE

100-
MAP (mmHg)

70-

60-

50-

40-

30-

20-

10-

0-

5 10

Time (min)

FIG. 2. Circulatory and ventilatory variables. Baseline (0) and 5 and 10 min after administration of vecuronium 0.1 mg kg\(^{-1}\).

*\(P < 0.01\) (Wilcoxon).

throughout the study, CVP decreased significantly at 5 and 10 min (fig. 2). Blood-gas analyses showed no significant differences with respect to pH, \(PCO_2\), \(PO_2\) and BE in the different time periods.

**DISCUSSION**

Accurate measurement of IOP depends upon stable conditions of arterial pressure, central venous pressure and arterial \(PCO_2\). Anaesthetic drugs known to effect IOP such as the volatile agents should be avoided and a pharmacodynamic steady state should exist in respect of any drugs being used.

The way in which neuromuscular blocking drugs influence IOP is still unclear. It has been shown that an increase in choroidal perfusion, rather than fasciculation of the striated eye muscles, is the cause of the increase in IOP which accompanies the administration of suxamethonium (Adams and Barnett, 1966). Al Abrak and Samuel (1974) cast doubt on the hypothesis that the relaxation of extraocular muscles contributes significantly to the decrease in IOP following neuromuscular blockade with non-depolarizing agents. They suggested that the decrease in IOP following the administration of tubocurarine was secondary to the decrease in systemic arterial pressure, which was also the mechanism suggested by Kalff and Linzen (1969) in respect of alcuronium. Giving pancuronium to spontaneously breathing volunteers, Litwiller, DiFazio and Rushia (1975) concluded that pancuronium exerted a direct effect on the eye, as the observed decrease in IOP was not related to any neuromuscular blocking effect of the small amount of drugs used in their study.

In 1981, Sia and Rashkovsky reported a small and transient increase in IOP following vecuronium 0.1 mg kg\(^{-1}\). However, Vilardi and colleagues (1983) described a decrease in IOP after a dose of 0.12 mg kg\(^{-1}\). Interpretation of these results is difficult as only patients undergoing eye surgery were included and measurements were taken with a Schiötz indentation tonometer. Central venous pressure and arterial \(PCO_2\) were not monitored, and no clinical steady-state was established.

The 22.6% decrease in IOP after 10 min in the current study and its correlation with the decrease in CVP would seem to indicate an indirect mechanism for the observed change in IOP following the administration of vecuronium (fig. 3). Neuromuscular blocking drugs reduce skeletal muscle tone and this leads to venous pooling. The resultant decrease in central venous pressure

**FIG. 3.** Percent changes from baseline values for heart rate, mean arterial pressure, carbon dioxide partial pressure, central venous pressure and intraocular pressure following administration of vecuronium.
improves the venous drainage from the choroid and increases the transscleral venous pressure gradient, facilitating the outflow of aqueous humour. Relaxation of the abdominal and thoracic muscles reduces resistance to ventilation, allowing a lower inflation pressure for a given tidal volume (Holloway, 1980). This mechanism further enhances drainage from the eye and decreases IOP. This is in agreement with our measurements of the CVP, which decreased in all patients in whom the IOP decreased. Conversely, none of the five patients (in four of whom central venous pressure was measured) in whom there was no decrease in IOP displayed such a decrease in CVP.

ACKNOWLEDGEMENTS
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REFERENCES