CHANGES IN INTRAOCULAR PRESSURE ASSOCIATED WITH THE ADMINISTRATION OF PROPOFOL

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Propofol (2,6-diisopropylphenol) is formulated currently as an oil-in-water emulsion (Cumings et al., 1984). This investigation was undertaken to define the effects of propofol on intraocular pressure when given as a single bolus dose of 2 mg kg\(^{-1}\) i.v. to induce anaesthesia, and then subsequently as an infusion at a rate of 9 mg kg\(^{-1}\) h\(^{-1}\) to maintain anaesthesia.

PATIENTS AND METHODS

Twenty-five patients aged 20–60 yr were studied. All were graded as ASA I or II and were without known ophthalmic abnormalities. They presented for elective general or orthopaedic surgery during which intraocular pressure (IOP) was measured (in both eyes) with a Perkins applanation tonometer (Perkins, 1965). Each patient gave informed consent and the study was approved by the Hospital Ethical Committee.

Patients were premedicated with lorazepam 1 mg given by mouth 1 h before the induction of anaesthesia. In the anaesthetic room a 20-gauge cannula was placed in a vein on the dorsum of the hand or forearm. IOP was measured bilaterally on arrival in the operating room (T1); anaesthesia was induced with a bolus dose of 1% propofol 2 mg kg\(^{-1}\) i.v. injected over a mean time of 36.4 s (SEM ± 1.99). One minute later, vecuronium 0.1 mg kg\(^{-1}\) was administered and the lungs were inflated gently with an oxygen-nitrous oxide mixture (\(F_{\text{In}}\) 0.33). One minute later, vecuronium 0.1 mg kg\(^{-1}\) was administered and the lungs were inflated gently with an oxygen-nitrous oxide mixture (\(F_{\text{In}}\) 0.33). Two minutes after the injection of the vecuronium, a continuous infusion of propofol 9 mg kg\(^{-1}\) h\(^{-1}\) was started using an Ivac 630 volumetric pump.

IOP was measured 1 min after induction (T2), 2 min after the injection of vecuronium (T3) and 2 min after the start of the continuous infusion of propofol (T4). After the larynx and the trachea were sprayed with 4% lignocaine, tracheal intubation was performed (between times T3 and T4). In order to maintain normal values of end-tidal carbon dioxide, ventilation was controlled with a Servo ventilator. Further measurements of IOP were made at 15 (T5) and 45 min (T6) after the start of anaesthesia. If the patient moved, the continuous infusion was supplemented by one or more bolus doses of propofol (up to 1 mg kg\(^{-1}\)). After the last IOP measurement (45 min) fentanyl 1–2 \(\mu g\) kg\(^{-1}\) was administered. Ten minutes before the end of surgery the infusion of propofol...
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Table I. Physical characteristics of the patients

<table>
<thead>
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<th>Propofol</th>
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<td>$n$</td>
<td>25</td>
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<tr>
<td>Sex ratio (M:F)</td>
<td>10:15</td>
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<tr>
<td>Age (yr) (mean ± SEM)</td>
<td>38.8±2.4</td>
</tr>
<tr>
<td>Weight (kg) (mean ± SEM)</td>
<td>69.2±2.0</td>
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was discontinued, and when the operation was finished, the nitrous oxide was discontinued and the patients allowed to breathe 100% oxygen.

Heart rate and arterial pressure were recorded at the time of IOP measurements and any side-effects occurring during the induction and maintenance of anaesthesia were noted. Statistical analyses were performed by Student’s $t$ test ($P < 0.05$).

RESULTS

Twenty-five patients (15 female) aged 20–60 yr and weighing 56–86 kg were studied (table I).

Figure 1 shows the changes in intraocular pressure at the different time points after induction, and during maintenance of anaesthesia, with propofol. One minute after the injection of propofol (T2), there was a significant decrease in IOP ($P < 0.001$) compared with the pre-induction value (T1). Administration of vecuronium resulted in a further decrease in IOP (T3). After intubation (T4) IOP increased, but the value remained less (statistically significant) than the pre-induction value. During maintenance (T5, T6), IOP was less than the baseline value, although the differences were not significant.

The administration of propofol 2 mg kg$^{-1}$ was associated with significant decreases in arterial pressure (T2, T3). Tracheal intubation increased arterial pressure above the baseline value (T4) and also caused a significant increase in heart rate. Changes in arterial pressure and heart rate were minimal during the maintenance of anaesthesia with propofol (fig. 2).

Side-effects noted in the study are presented in table II. A cutaneous flush was observed in nine patients and seven patients experienced discomfort on injection. Spontaneous movement occurred in five patients and hiccup in three.

In all patients recovery after anaesthesia with propofol was rapid and smooth.

DISCUSSION

This study has demonstrated that propofol, in a dose of 2 mg kg$^{-1}$, produces a significant and
FIG. 2. Mean (±SEM) arterial pressure and heart rate changes at different times after administration of propofol: 1 = Pre-induction values; 2 = 1 min after induction with propofol 2 mg kg⁻¹ (+ bolus injection of vecuronium 0.1 mg kg⁻¹); 3 = 3 min after induction with propofol (+ start of continuous infusion of propofol 9 mg kg⁻¹ h⁻¹); 4 = 5 min after induction with propofol—intubation between 3 and 4; 5 = 15 min after start of anaesthesia; 6 = 45 min after start of anaesthesia. *P < 0.05; ***P < 0.001.

TABLE II. Side effects

<table>
<thead>
<tr>
<th>Side effect</th>
<th>Propofol (n = 25)</th>
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<tbody>
<tr>
<td>Discomfort on injection</td>
<td>7</td>
</tr>
<tr>
<td>Cutaneous flush</td>
<td>9</td>
</tr>
<tr>
<td>Spontaneous movement</td>
<td>5</td>
</tr>
<tr>
<td>Hiccup</td>
<td>3</td>
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</table>

useful decrease in IOP. Other i.v. anaesthetic induction agents have been shown to cause decreases in IOP. Couch, Eltringham and Magauran (1979) observed a decrease in IOP after the induction of anaesthesia with thiopentone. The decrease in IOP was substantially the same as in our study. There was also a decrease in mean arterial pressure after the injection of thiopentone. From these results it cannot be clearly defined whether the observed decrease in IOP was attributable to the effects of propofol itself or was the result of the decrease in arterial pressure. In our study of patients younger than 60 yr, the injection of propofol was associated with significant decreases in arterial pressure, and this may have contributed to the reduction in IOP (Self and Ellis, 1977). When etomidate was used as an induction agent, a decrease in IOP was observed by Oji and Holdcroft (1979); however, they were not able to demonstrate a decrease in systemic arterial pressure.

In all these studies the applanation tonometric method was used (Perkins, 1965). This instrument was chosen because it uses the principle of applanation, which is more accurate than that of indentation tonometry with the Schiotz tonometer (Kaufman, 1972). The end-point utilizes corneal patterns that must be properly centred to obtain an accurate reading. Applanation methods read directly the weight (in grams) needed to produce flattening of a given corneal area, and are directly convertible to mm Hg. The weight applied to the eye (0.5 g) is very small, and induces minimal increase in intraocular pressure. The result is not affected by the rigidity of the ocular coats (Schmidt, 1961).

In ophthalmic surgery, patients are ventilated electively; the administration of vecuronium immediately after the induction dose of propofol may contribute to the further decrease in IOP. These results confirm the study of Jantzen and co-workers (1986), but contrast with the increase in IOP recorded by Sia and Rashkovsk (1981) after injection of vecuronium. However, measurements were taken with a Schiotz tonometer in that study. The use of suxamethonium should be avoided in intraocular surgery, as IOP increases for 10 min or more after injection of this depolarizing agent (Cook, 1981). Patients were normoventilated with a nitrous oxide–oxygen mixture, as measured by end-tidal carbon dioxide (Smitt, Aass and Elnemoto, 1981). Although the inhalation of nitrous oxide is not associated with any change in IOP (Holloway, 1980) the use of nitrous oxide should be avoided in patients in whom intravitreal injection of gas is planned (Wolf, Kapuano and Harding, 1983).

Following intubation there were increases in IOP in all the patients studied, but the values were still lower than the pre-induction control values. In our study a small dose of lorazepam was given as premedication, because large doses of benzodiazepines decrease IOP. Thus, the control value could be influenced if higher doses are used.
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(Al-Abrak, 1979). Better control of arterial pressure and heart rate at the time of tracheal intubation might have been obtained by the administration of fentanyl before the induction of anaesthesia. Low-dose fentanyl blunts the circulatory responses to tracheal intubation (Martin et al., 1982). During the continuous infusion of propofol, IOP did not exceed the control pre-induction values. Volatile anaesthetic agents were avoided in this study. Spontaneous movements and hiccup in lighter levels of anaesthesia might induce small increases in IOP in the patients (Greaves, 1958).

Induction of anaesthesia with propofol was associated with a significant and useful reduction in intraocular pressure. During i.v. anaesthesia with propofol, IOP did not exceed control values. In the context of this study propofol—when used to induce and maintain anaesthesia—appeared safe. However, it will be remembered that the patients studied did not have any ophthalmological pathology. Consequently, further studies may be indicated before these results can be extrapolated to such patients.

REFERENCES


