Desflurane maintains intraocular pressure at an equivalent level to isoflurane and propofol during unstressed non-ophthalmic surgery

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Summary

We have investigated the effects of desflurane compared with isoflurane and propofol on intraocular pressure (IOP) in 48 ASA I–II patients undergoing elective non-ophthalmic surgery. Anaesthesia was induced with thiopental 3–5 mg kg⁻¹, fentanyl 2–4 µg kg⁻¹ and vecuronium 0.1 mg kg⁻¹. Patients were allocated randomly to receive propofol (n = 16) 4–8 mg kg⁻¹ h⁻¹, isoflurane (n = 16) or desflurane (n = 16) for maintenance of anaesthesia. Fentanyl was added if necessary. The lugs were ventilated with 70% nitrous oxide in oxygen. Arterial pressure, electrocardiography, heart rate and end-tidal carbon dioxide were measured throughout anaesthesia. IOP was measured before surgery, during maintenance and after emergence from anaesthesia with applanation tonometry by an ophthalmologist blinded to the anaesthetic technique. There was a significant decrease in IOP after induction of anaesthesia which did not differ between groups. Desflurane maintained IOP at an equivalent level to isoflurane and propofol. (Br. J. Anaesth. 1998; 80: 243–244)

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One aim of anaesthetic management during ophthalmic surgery is to provide good control of intraocular pressure (IOP). An increase in IOP may be catastrophic in patients with glaucoma or a penetrating open-eye injury. Anaesthesia in these patients must prevent an increase in IOP and provide a “soft” eye which is suitable for surgery and free of haemorrhage, particularly expulsive choroidal haemorrhage. IOP is determined by the rate of production of aqueous humour and vitreous volume, choroidal blood volume, scleral rigidity, orbicularis oculi muscle tension and external pressure. Many studies have examined the effects of anaesthetics and related drugs on IOP. Inhalation anaesthetics such as halothane and isoflurane decrease IOP by lowering the formation rate of aqueous humour and increasing the trabecular outflow facility. The i.v. hypnotic agents propofol and thiopental also decrease IOP and have been demonstrated to attenuate the increase in IOP in response to laryngoscopy and intubation.

Desflurane is a volatile anaesthetic with a low blood-gas solubility coefficient. Recovery from anaesthesia with desflurane is more rapid than with other potent inhalation anaesthetic agents and comparable with propofol. Because of its irritant properties on the airway, desflurane is not suitable for inhalation induction of anaesthesia in ophthalmic patients, but it may be an ideal maintenance anaesthetic. In dogs, desflurane (7.0% expired) was associated with a baseline IOP of 11.3 (SD 3.8) mm Hg; normal IOP is approximately 15 mm Hg, with a “normal” range of approximately 10–20 mm Hg.

There have been no studies on the effects of desflurane on IOP in humans. We performed a randomized, prospective, single-blind study to compare the effects of desflurane, isoflurane and propofol on IOP in patients undergoing elective non-ophthalmic surgery.

Methods and results

After obtaining approval from the Ethics Committee of Vienna General Hospital, we studied 48 patients undergoing elective non-ophthalmic surgery. Inclusion criteria were age 16–60 yr, ASA I or II, no known allergies or adverse reactions to any of the anaesthetic agents used in the study, and no pre-existing ophthalmic disease. Before surgery, patients were allocated randomly to one of three groups: propofol (n = 16), isoflurane (n = 16) or desflurane (n = 16). All patients were premedicated with midazolam 7.5 mg, 1 h before surgery. Anaesthesia was induced with fentanyl 2–4 µg kg⁻¹, thiopental 3–5 mg kg⁻¹ and vecuronium 0.1 mg kg⁻¹. Ventilation was adjusted to maintain end-tidal carbon dioxide partial pressure at 4.3–4.6 kPa. Anaesthesia was maintained with continuous infusion of propofol 4–8 mg kg⁻¹ h⁻¹ (n = 16) or 1 MAC of isoflurane or desflurane. The lungs were ventilated with 70% nitrous oxide in oxygen. Arterial pressure, electrocardiography, heart rate, oxygen saturation, end-tidal carbon dioxide concentration and nasopharyngeal temperature were measured throughout anaesthesia. IOP was obtained by a hand-held applanation tonometer (Perkins). Patients were positioned supine for examination. All measurements were made by one ophthalmologist blinded to the
anaesthetic technique. IOP was measured before induction of anaesthesia, immediately before intubation, 1, 3, 5 and 10 min after intubation, every 15 min during anaesthesia and at least 1 min after extubation. A final measurement was performed in the recovery room.

One-way ANOVA with Scheffé’s post hoc test was used to compare baseline data. ANOVA for repeated measures and paired t tests were used to assess differences within groups. Correlations were performed using Pearson’s correlation coefficient. All data are presented as mean (SD). P < 0.05 was considered significant.

All patients were evaluated and were comparable in age, weight, sex and duration of operation. Baseline values of mean arterial pressure (MAP), heart rate and IOP were similar between groups. After induction of anaesthesia, IOP decreased significantly 1 min after the end of induction (propofol group, P < 0.05; isoflurane and desflurane groups, P < 0.01). During maintenance of anaesthesia, there were no significant changes in IOP over time in any group (fig. 1). In the recovery room, IOP returned to baseline in the desflurane and isoflurane groups, but remained significantly lower in the propofol group (P < 0.01). MAP and heart rate decreased significantly from baseline (both P < 0.01) during anaesthesia but increased towards baseline in the recovery room. There were no significant differences in MAP or heart rate between groups. No correlation was found between MAP, heart rate and IOP values.

Comment

The results of this study demonstrated that induction of anaesthesia significantly decreased IOP. Maintenance of anaesthesia with desflurane sustained a low IOP at a level equivalent to that of isoflurane and propofol.

Many factors (in addition to glaucoma) influence IOP, such as genetic status, age, refractive error and race. In addition, IOP is dependent on PEEP ventilation, PaO2, and Paco2. Any acute increase in intra-abdominal or intrathoracic pressure, not infrequently observed during extubation, may increase IOP. Particular attention must be paid to induction of and emergence from anaesthesia as both mechanical and pharmacological stresses have an amplifying effect on IOP. Most anaesthetic and hypnotic agents, including volatile anaesthetics, barbiturates, opioids, neuroleptics and benzodiazepines decrease IOP in proportion to the depth of anaesthesia. Jantzen emphasized the importance of anaesthetic drugs for general anaesthesia in ophthalmic surgery.6 He reported that i.v. anaesthetics and volatile agents reduce IOP, with the possible exception of ketamine.

There have been no studies assessing the influence of desflurane on IOP in humans. Artru demonstrated the influence of desflurane and halothane anaesthesia on IOP in dogs.5 Both halothane and desflurane decreased IOP by approximately 7 mm Hg from baseline values in unanaesthetized dogs, with no difference between groups.

The results of our study indicate that desflurane, in common with isoflurane and propofol, maintained low IOP values during anaesthesia in patients undergoing non-ophthalmic surgery. The decrease in IOP after induction of anaesthesia was sustained if maintenance was provided by desflurane, isoflurane or propofol. In all groups, IOP increased towards baseline values after emergence from anaesthesia. Interestingly, IOP values remained significantly lower compared with baseline in the propofol group. We conclude that desflurane was as safe as isoflurane and propofol for maintenance of anaesthesia. The sustained low IOP after propofol anaesthesia may be beneficial in patients at risk and remains to be evaluated.

References