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Comparison of desflurane and isoflurane in anaesthesia for dental surgery

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Summary

We studied 50 ASA I–II patients, aged 18–65 yr, undergoing elective orofacial surgery. Anaesthesia was induced with fentanyl and propofol, and maintained with 66 % nitrous oxide in oxygen and either desflurane or isoflurane to compare recovery characteristics and cardiovascular stability. Cardiovascular responses to induction, intubation and incision were similar with both agents, although the increase in heart rate in response to intubation was less marked in the desflurane group. Maximum end-tidal concentrations of desflurane required were 4.0–10.6 % (mean 6.8 %) compared with maximum isoflurane concentrations of 1.1–2.3 % (mean 1.6 %). Mean duration of anaesthesia was 46 (SD 17.9) min (range 25–89 min) in the desflurane group and 41 (11.5) (23–60) min in the isoflurane group. Times to extubation were 6.7 (2.1) (3–10) min and 11.3 (4.1) (5–23) min, to eye opening 6.8 (2.2) (3–11) min and 12.7 (6.9) (7–37) min, to stating date of birth 9.0 (2.3) (4–12) min and 15.0 (6.9) (8–39) and to discharge from the recovery room 45 (11.6) (22–80) min and 64 (20.9) (28–134) min, for the desflurane and isoflurane groups, respectively (all $P < 0.0001$). No serious complications occurred in any patient. (*Br. J. Anaesth*. 1995; **75**: 289–292)

Key words

Anaesthetics volatile, isoflurane. Anaesthetics volatile, desflurane. Recovery, postoperative. Surgery, dental.

Desflurane differs from isoflurane in being halogenated exclusively with fluorine. It has a boiling point close to room temperature (22.8 °C) and the lowest blood–gas partition coefficient (0.42) of the available inhalation agents. Its solubility in other tissues is also lower than that of other volatile agents [1, 2]. Studies in volunteers have shown rapid onset of effect and recovery [3, 4].

Previous studies in patients have demonstrated shorter recovery times with desflurane compared with isoflurane anaesthesia [5–8]. The aim of the present study was to evaluate its use in patients undergoing dental surgery.

Patients and methods

After regional Ethics Committee approval and written informed consent, we studied 50 adult patients, ASA I–II. All were undergoing elective dental surgery under general anaesthesia, expected to last 20–60 min, with a planned overnight stay in hospital (extraction of wisdom or other teeth, reduction of facial fractures). A venous blood sample was obtained before anaesthesia for measurement of serum concentrations of creatinine, bilirubin, alkaline phosphatase and hepatic transaminases.

Premedication comprised oral temazepam 10–20 mg. Minimum standard monitoring was commenced before anaesthesia was induced with fentanyl 1–2 μg kg^{-1} and propofol 1–3 mg kg^{-1}. Ventilation with 66 % nitrous oxide in oxygen was assisted manually as required. Twenty-five patients each received isoflurane (group I) or desflurane (group D) from recently calibrated dedicated Ohmeda Tec 4 and Tec 6 vaporizers, respectively. Allocation was randomized. End-expiratory concentrations of volatile agent and carbon dioxide were measured by an Ohmeda RGM 5250 monitor. The concentration of agent was adjusted to maintain adequate depth of anaesthesia (stable arterial pressure and heart rate), as in routine practice. Neuromuscular monitoring using a Datex Relaxograph was commenced before neuromuscular block was produced with mivacurium 0.15–0.2 mg kg^{-1}. Tracheal intubation, almost always by the nasal route, was performed after the onset of block (3–5 min after induction). Additional doses of blocker were administered if required but this was rarely necessary. Ventilation was adjusted to maintain normocapnia. Heart rate, systolic and diastolic arterial pressures (SAP and DAP), arterial oxygen saturation (Sp_{O_2}, end-tidal carbon dioxide concentration and end-tidal agent concentrations were recorded at 2-min intervals from the time of induction until 10 min after incision, and then at 5-min intervals.

The majority of patients ($n = 43$) had intraoperative nerve blocks or local infiltration with bupivacaine for postoperative analgesia.

Residual neuromuscular block was antagonized if required at the end of surgery using neostigmine with glycopyrronium. The anaesthetic agent and nitrous oxide were discontinued after recovery of the TOF ratio to at least 0.7, and the circuit flushed with oxygen 12 litre min^{-1} until the inspired concentration of the volatile agent was zero. Ventilation was assisted until it resumed spontaneously.

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The times from stopping all anaesthesia until eye opening, extubation of the trachea and ability to state the date of birth were recorded in every case. Nausea, vomiting, or any other adverse events, were recorded in the postoperative recovery ward, together with other routine observations. All patients were prescribed an oral analgesic and encouraged to take it in the recovery room if possible. Whether or not the patient was capable of taking oral analgesia was judged by the recovery room nurse, who was unaware of the treatment group. This nurse also judged the patient's fitness for discharge (based on adequate verbal communication, absence or control of pain and nausea, and satisfactory cardiovascular observations and oxygen saturation on room air).

A second sample of venous blood was obtained after operation for measurement of serum concentrations of creatinine, bilirubin and hepatic enzymes. Adverse events before discharge were recorded and patients were contacted by telephone after 14–21 days for further enquiry about adverse events.

Data for heart rate and arterial pressure within each group were analysed using repeated measures analysis of variance followed by the Tukey–Kramer test for multiple comparisons. Fisher's exact test or the Mann–Whitney test were used for comparing the incidence of side effects and recovery times, respectively.

Results

The groups were comparable in weight, height, age and sex distribution (table 1). Mean doses of fentanyl and propofol were also similar (fentanyl 1.9 (SD 0.2) $\mu g\ kg^{-1}$ in both groups; propofol 2.3 (0.4) and 2.2 (0.3) $mg\ kg^{-1}$ in the desflurane and isoflurane groups, respectively). In all cases, the highest end-tidal concentration of volatile agent was required during the first 10 min after induction of anaesthesia, and ranged from 4 % to 10.6 % (mean 6.8 %) for desflurane and from 1.1 % to 2.3 % (mean 1.7 %) for isoflurane. By the end of surgery, the end-tidal volatile anaesthetic concentrations had been reduced, and were 2.8–6.1 % (mean 3.9 %) for desflurane and 0.3–1.4 % (mean 0.9 %) for isoflurane. These doses were greater for isoflurane than for desflurane in terms of relative MAC values in nitrous oxide (desflurane 4 % and 2.8 %, isoflurane 0.56 % and 0.5 % for adults younger than and older than 31 yr, respectively [9, 10]).

Table 1 Patient characteristics (mean (SD) [range] or number). No significant differences

	Age (yr)	Height (cm)	Weight (kg)	Sex (M/F)
Desflurane	29 [20–58]	168 (10) [150–187]	69 (13.8) [48–98]	11/14
Isoflurane	26 [18–51]	166 (9.8) [152–185]	65 (12.9) [46–95]	17/8

There were only small changes in heart rate after induction of anaesthesia in both groups. There was an increase in heart rate after intubation in both groups, which was greater and statistically significant at 6 and 8 min compared with preinduction values in the isoflurane group. Heart rate in this group was significantly higher than in the desflurane group at 8 and 10 min after induction (91 *vs* 81 beat min^{-1} at 8 min and 85 *vs* 76 beat min^{-1} at 10 min). No dysrhythmias were observed in any patient.

SAP was significantly higher in the desflurane group before induction and tended to remain higher throughout anaesthesia, although only significantly so at 4 min and at the end of anaesthesia. In both groups SAP decreased significantly after induction and increased transiently after intubation. Changes in DAP followed a similar pattern, but differences between groups were not significant at any time.

Arterial oxygen saturation decreased slightly in both groups after induction of anaesthesia and during intubation. Mean and median oxygen saturations were greater in the isoflurane group, before and after induction, the difference being statistically significant at 4 and 6 min after induction. The differences between groups at equivalent time points were always less than 1 %, and were never less than 95 % in any patient at any time.

Mean duration of anaesthesia was 46 (SD 17.9) (median 40; range 25–89) min and 41 (11.5) (40, 23–60) min, respectively, in the desflurane and isoflurane groups.

All patients had attained a TOF ratio greater than 0.7 before assessment of recovery. Recovery times to all end points in the desflurane group were significantly shorter, as was time of fitness for discharge from the recovery room (table 2; $P < 0.0001$). Twenty-four patients who received desflurane were judged ready to take oral analgesia in the recovery room compared with 19 who received isoflurane ($P = 0.1$).

Two patients suffered nausea and one vomited in the isoflurane group, and one patient had nausea and vomiting i n the desflurane group in the recovery ward.

One patient in the isoflurane group developed postoperative laryngospasm and two others experienced faintness and postural hypotension with no obvious cause, several hours after operation. Another two patients in this group complained of headache after operation. One patient in the desflurane group

Table 2 Recovery times (min) from stopping anaesthesia to extubation, eye opening, stating date of birth (DOB) and fitness for discharge (mean (SD) [median; range]). $P < 0.0001$ between groups for all variables

	Duration of anaesthesia	Extubation	Eyes Open	DOB	Fit for discharge
Desflurane	46 (17.9) [40; 25–89]	6.7 (2.1) [7; 3–10]	6.8 (2.2) [7; 3–11]	9 (2.3) [9; 4–12]	45 (11.6) [44; 22–80]
Isoflurane	41 (11.5) [40; 23–60]	11.3 (4.1) [11; 5–23]	12.7 (6.9) [10; 7–37]	15 (6.9) [12; 8–39]	64 (20.9) [56; 28–134]

Table 3 Serum concentrations of creatinine, bilirubin and hepatic transaminases before (Preop.) and after anaesthesia (Postop.) (mean (SD)); ($n = 16$ for the isoflurane group and $n = 25$ for the desflurane group) *$P < 0.05$ compared with respective preoperative value

	Desflurane		Isoflurane	
	Preop.	Postop.	Preop.	Postop.
Creatinine (μmol litre^{-1})	78 (11.6)	76 (14.9)	73 (16.5)	72 (15.1)
Bilirubin (μmol litre^{-1})	16.6 (9.2)	18.2 (12.1)	14.5 (4.0)	15.6 (2.7)
Alkaline phosphatase (u. litre^{-1})	72 (20.1)	70 (17.7)	69 (18.4)	68 (19.5)
Aspartate transaminase (u. litre^{-1})	32 (11.3)	28 (7.3)*	31 (9.8)	27 (8.3)*
Alanine transaminase (u. litre^{-1})	30 (16.0)	28 (13.3)	28 (9.6)	24 (7.2)*

developed a short-lasting bacteraemia 4 h after excision of a mandibular cyst (rigors, pyrexia, tachycardia and hypotension). This responded to i.v. fluids and antibiotics. Although there were some significant changes in liver transaminases in both groups, these were minor and of no clinical significance (table 3).

Six patients (two in the desflurane group) could not be contacted by telephone after operation. All other patients had no anaesthesia-related problems in the 14–21-day follow-up period.

Discussion

Our main finding in this study was a reduction in recovery time with desflurane. Previous studies comparing desflurane with isoflurane have reported similar reductions in early recovery times [5, 8]. Significant reductions in time to fitness for discharge from the recovery room have been described previously in only one study of elderly patients [7].

Real differences in times to fitness for discharge may be masked by administrative details, such as the availability of anaesthetic staff to pronounce fitness, or nursing and portering staff to transfer patients. There may also be policies, actual or implied, dictating minimum times to be spent in the recovery ward. By recording the time taken to fitness for discharge instead of the time to leave the recovery area, we have attempted to measure a potential for improvement, rather than reflect administrative practices. Another possible reason for the difference between this and previous studies may be the use of local anaesthetics to provide postoperative analgesia. This could have reduced the need for postoperative opioids which may cause sedation and delay discharge from the recovery area.

One reason for the shorter recovery times in the desflurane group may have been the use of lower concentrations (in terms of MAC values) than in the isoflurane group. This was not deliberate, as concentrations were adjusted according to apparent clinical needs. The adequacy of anaesthesia is supported further by the fact that patients in the desflurane group had a lower increase in heart rate in response to intubation, suggesting that these patients may have been more deeply anaesthetized at that time.

A statistically significant reduction in time to arousal and extubation, although useful, may not represent a significantly important advantage. However, earlier discharge from the recovery ward, particularly in the setting of the present study, is a definite advantage as the turnover of patients can be increased. This would be advantageous in situations where the recovery area has limited space. In any case, early recovery reduces the need for intensive nursing. Although our study was not carried out in patients undergoing day-case surgery, the results would indicate an advantage of desflurane in outpatient dental anaesthesia.

The criteria used for discharge from the recovery room were relatively subjective in the present study and were based on the routine used in this area of the hospital. Similar results for recovery have been reported by others also using semi-objective criteria [7]. We believe that the use of the same method of assessment in all patients by experienced nursing staff who were unaware of the anaesthetic agent used should have removed any bias.

In the present study there was little evidence of sympathetic stimulation which has been reported with desflurane [11–14]. The cardiovascular changes around the time of induction were clinically acceptable, and similar to those observed with isoflurane. Increases in arterial pressure and heart rate after induction coincided with intubation, and the less marked increase in heart rate after intubation may have reflected more rapid onset of deep anaesthesia with desflurane. Any sympathetic stimulation may also have been attenuated by premedication with temazepam and induction with propofol and fentanyl.

The absence of effect on serum creatinine, bilirubin, alkaline phosphatase and hepatic transaminases was in keeping with previous studies, which have found that desflurane undergoes little biotransformation and produces minimal metabolites, with elimination mainly via the lungs [15, 16].

The use of desflurane may be associated with coughing, breath-holding or laryngospasm when used for induction of anaesthesia [17, 18]. This was not observed in the present study as anaesthesia was induced using fentanyl and propofol.

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