Use of Desflurane for Outpatient Anesthesia

A Comparison with Propofol and Nitrous Oxide

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Desflurane's induction and recovery characteristics were compared to those of propofol-nitrous oxide in outpatients undergoing laparoscopic procedures. Ninety-two healthy patients were randomized to receive either: 1) propofol induction and propofol-nitrous oxide maintenance (control), 2) propofol induction and desfluranenitrous oxide maintenance, 3) desflurane-nitrous oxide, or 4) desflurane alone for induction and maintenance of anesthesia. Inhalational induction with desflurane-nitrous oxide was faster than with desflurane alone (100 \pm 35 vs. 124 \pm 43 s). Inhalation inductions were associated with a high incidence of apnea (17 and 26%), breathholding (26 and 39%), and coughing (30 and 22%) in groups 3 and 4, respectively. The emergence time after discontinuation of desflurane in oxygen (4.5 \pm 2.1 min.) was significantly less than that after propofol-nitrous oxide (7.3 \pm 3.9 min.). However, times from arrival in the recovery room until the patients were judged fit for discharge were similar for all four treatment groups. Digit-symbol substitution test results and sedation visual analogue scores also were similar during the first 2 h in the recovery room. A lower incidence of moderate-to-severe nausea was reported in group 1 (15% vs. 52, 52, and 59% in groups 2, 3, and 4, respectively). In conclusion, induction of anesthesia with desflurane was rapid but is associated with a high incidence of airway irritation. Emergence and recovery profiles after maintenance of anesthesia with desflurane compared favorably to a propofol-nitrous oxide combination. However, propofol was associated with a lower incidence of nausea than was desflurane after outpatient anesthesia for laparoscopic surgery. (Key words: Anesthesia: outpatient. Anesthetic technique: inhalational; intravenous. Anesthetics, intravenous: propofol. Anesthetics, volatile: desflurane.)

DESFLURANE is a new volatile anesthetic possessing a low blood–gas partition coefficient^{1,2} that should provide rapid emergence from anesthesia. In comparison to isoflurane, desflurane has been reported to decrease awakening times and to result in less impairment of postoperative cognitive function.³ Such properties appear to make desflurane particularly attractive for outpatient anesthesia, for which there may be benefit with more rapid emergence from anesthesia.⁴

Propofol is widely used for both induction and maintenance of outpatient anesthesia because of its favorable

recovery characteristics. The use of a variable-rate infusion of propofol for maintenance of anesthesia during short surgical procedures was associated with fewer undesirable side effects and resulted in earlier ambulation and discharge when compared to an isoflurane-based anesthetic technique.⁵ Although induction of anesthesia in outpatients usually is achieved using intravenous (iv) drugs, the physical properties of desflurane suggest that it may be capable of achieving rapid induction of anesthesia.^{1,2} If induction with desflurane were rapid and smooth, it would be a useful alternative to the commonly used iv induction agents.

The purpose of this study was to compare the induction and recovery profiles after general anesthesia with desflurane with or without nitrous oxide or with propofol for induction, to those of a standard propofol–nitrous oxide technique in outpatients undergoing laparoscopic procedures.

Materials and Methods

Ninety-two nonpregnant gynecologic patients, 19-46 yr of age, ASA physical status 1 or 2, scheduled for outpatient laparoscopic surgery were studied according to a protocol approved by the Washington University Human Studies Committee. After obtaining written informed consent, patients were assigned randomly to one of four treatment groups using an open (nonblinded) study design. In the preoperative holding area, patients completed a digit–symbol substitution test (DSST) and 100-mm visual analogue scales (VAS; 0 = none to 100 = maximum) for pain and sedation .

No preanesthetic medication was administered. All patients breathed 100% oxygen for 2 min after receiving a preinduction dose of fentanyl 1.5 μ g · kg⁻¹ iv (to attenuate the hemodynamic response to tracheal intubation) and d-tubocurarine 3 mg iv (to minimize muscle fasciculations). In groups 1 and 2, anesthesia was induced with propofol 2.5 mg·kg⁻¹ iv administered over 2.5 min using a syringetype infusion pump (Bard InfusOR®, C.R. Bard, North Reading, MA). In groups 3 and 4, anesthesia was induced by inhalation of desflurane with nitrous oxide 60% in oxygen or with 100% oxygen, respectively. Desflurane was delivered using a modified Ohmeda anesthesia machine with a built-in, electrically heated, temperature-controlled vaporizer (DM5000®, Ohmeda, Madison, WI). The endtidal concentration of desflurane was continuously monitored using an infrared-absorption multigas analyzer

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Received from the Department of Anesthesiology, Washington University School of Medicine, St. Louis, Missouri. Accepted for publication April 11, 1991. Supported in part by a grant from Anaquest, Millburn, New Jersey.

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(Datex model 254°, Puritan Bennett, Tewksbury, MA). The gas analyzer was calibrated prior to each surgical procedure using gases analyzed to \pm 0.01% accuracy (Scott Medical Products, Plumsteadville, PA). Desflurane was delivered initially at an inspired concentration of 3%; the concentration subsequently was increased in 0.5% increments at 5–10-s intervals until loss of consciousness.

During the induction period, the time to loss of responsiveness was tested at 5–10-s intervals by asking the patients to open their eyes. When the patients did not respond to this command, the loss of the eyelash reflex was evaluated. Patients breathed spontaneously during induction of anesthesia; however, manual assistance of ventilation usually was necessary after loss of consciousness. The occurrence of respiratory complications (e.g., coughing, breath-holding, apnea, laryngospasm, or bronchospasm), excitatory phenomena (e.g., moving or myoclonus), pain on injection, and any other adverse effects during induction were recorded. After loss of consciousness, succinylcholine 1.5 mg·kg⁻¹ iv was administered to facilitate intubation.

In group 1, anesthesia was maintained with nitrous oxide 60% and a continuous infusion of propofol at an initial infusion rate of $160~\mu g \cdot kg^{-1} \cdot min^{-1}$, which subsequently was titrated within the range of $50-200~\mu g \cdot kg^{-1} \cdot min^{-1}$. In the remaining groups, anesthesia was maintained with desflurane 4–7% inspired concentration in combination with nitrous oxide 60% in groups 2 and 3 or with 100% oxygen in group 4. The maintenance infusion rate of propofol or the inspired desflurane concentration were adjusted to maintain an adequate depth of anesthesia, as judged by clinical signs and hemodynamic responses.

An additional dose of fentanyl 1.5 μ g · kg⁻¹ iv was administered to all patients 2 min prior to skin incision to attenuate the hemodynamic response to the surgical stimulus. Vecuronium 0.03-0.05 mg·kg⁻¹ was administered to facilitate ventilation and intraabdominal gas insufflation. Ventilation was controlled to produce an end-tidal carbon dioxide tension of 38 ± 5 mmHg, measured by capnography (Datex, Puritan Bennett), and body temperature was maintained at 36-37° C. Noninvasive blood pressure measurements (Dinamap®, Critikon, Tampa, FL), heart rate, hemoglobin oxygen saturation (Ohmeda Biox 3700®, Pleasanton, CA), end-tidal carbon dioxide tension, and desflurane concentration and propofol infusion rate were recorded every 2 min from induction until skin incision, every 1 min for the first 5 min after incision, and then every 5 min until the end of surgery. In addition, the time of discontinuation of the anesthetic agent, the total dose of propofol (in group 1), and the requirements for adjuvant therapy were recorded. At the end of the procedure, residual neuromuscular blockade was reversed with neostigmine $40-80 \mu g \cdot kg^{-1}$ and glycopyrrolate 8-16 μ g·kg⁻¹ iv.

At the end of anesthesia and after return of the eyelash reflex, patients were asked at 5-10-s intervals to open their eyes. The times at which the patients opened their eyes and responded to specific verbal commands ("squeeze my fingers"; "what is your name?"; "what is your date of birth?") were recorded. At 30, 60, 90, and 120 min after surgery, the patients repeated the DSST and VAS for pain and sedation. In addition, the degree of sedation (asleep and difficult to arouse; asleep but easily arousable; awake and calm; or awake and anxious or agitated) was assessed 30 min after surgery by a research nurse who was blinded as to the anesthetic treatment. At the same time, the nurse asked the patients to rate their nausea as none, mild (nausea not requiring treatment), moderate (nausea and vomiting requiring antiemetic treatment), or severe (persistent nausea with repeated episodes of vomiting, requiring treatment). Patients who had received opioid analgesic medications prior to the assessment of nausea or sedation were excluded from the data analysis. Patients were encouraged to sit, stand, walk, and take oral fluids when they felt capable of doing so. The time at which these events occurred was recorded and also was used to determine when the patient was fit for discharge.

Data are expressed as mean values \pm standard deviations. Continuous variables were analyzed using analysis of variance (with Bonferroni multiple-comparison tests used to assess differences between the four groups), and chi-squared tests were used for descriptive (categorical) variables. Changes in continuous variables over time were analyzed using paired t tests. In all cases, P values < 0.05 were considered statistically significant.

Results

The four groups were comparable with respect to age, weight, ASA physical status, and types and duration of surgical procedures performed (table 1).

The time to loss of consciousness in the patients receiving propofol for induction of anesthesia (groups 1 and 2) was 75 ± 25 s. The time to loss of the eyelash reflex was significantly longer for patients receiving inhalational induction with desflurane alone (group 4), 124 ± 43 s, than with desflurane-nitrous oxide (group 3), 100 ± 35 s. Side effects and complications during induction of anesthesia are summarized in table 2. Of the patients receiving propofol, 41% complained of pain at the infusion site during induction. The incidence of excitatory phenomena (e.g., spontaneous movements) was similar in all four treatment groups. Breath-holding, apnea, and coughing were more frequent during inhalational induction of anesthesia with desflurane (groups 3 and 4). The incidence of these complications was not significantly altered by the presence of nitrous oxide. Laryngospasm occurred during induction with desflurane in one patient

TABLE 1. Demographic Characteristics of the Four Anesthetic Treatment Groups

	Group 1	Group 2	Group 3	Group 4
Number	23	23	23	23
Age (yr)	31 ± 6	28 ± 6	30 ± 5	31 ± 6
ASA physical status (1/2)	20/3	19/4	21/2	18/5
Weight (kg)	63 ± 9	65 ± 17	71 ± 14	64 ± 9
Procedures (n)				
Diagnostic	6	6	8	1 6
Laser treatment	3	4	2	4
Tubal ligation	14	13	13	13
Operating time (min)	38 ± 28	50 ± 37	44 ± 29	36 ± 26
Anesthesia time (min)	56 ± 30	68 ± 38	61 ± 31	55 ± 29
PACU fentanyl dose (μg)	70.5 ± 91	51.5 ± 65	27.3 ± 38	37.3 ± 50

Values are mean ± SD or numbers.

in each of the inhalation groups. Oral secretions also were troublesome in three patients induced with desflurane.

One patient in group 3 became extremely distressed during induction of anesthesia and forcefully removed the face mask; in this case the inhalation induction was abandoned and anesthesia was induced with propofol 2.5 mg·kg⁻¹ iv. In addition, one patient in group 4 had nitrous oxide added for the latter portion of the induction phase because of an excessive delay in achieving a loss of consciousness (>5 min). These two patients were not included in the analysis of our induction data.

Hemodynamic stability was satisfactorily maintained in all four groups (fig. 1). Systolic blood pressure decreased after induction of anesthesia; the minimum value was recorded immediately prior to skin incision. After skin incision, the systolic pressure in the desflurane-nitrous oxide group initially was significantly lower than in the control group; however, the systolic pressure gradually returned to awake levels in all four groups over the remainder of the case, and there were no other differences between groups. After induction of anesthesia, the heart rate surpassed the awake rate; the increase was significantly greater in the propofol-desflurane (group 2) patients than in the control patients. Heart rate decreased just before skin incision and remained stable and similar among the four groups for the remainder of the maintenance period.

In group 1, the mean propofol maintenance infusion rate was $111 \pm 46 \ \mu g \cdot kg^{-1} \cdot min^{-1}$. The end-tidal concentration of desflurane required to maintain a stable blood pressure and heart rate is shown in figure 2. This was significantly greater in patients not receiving nitrous oxide (group 4). The end-tidal desflurane concentration at the end of anesthesia was also significantly higher in the absence of nitrous oxide; mean values were 3.6 ± 1.6 , 3.4 ± 1.5 , and $4.6 \pm 0.9\%$, for groups 2, 3, and 4, respectively. The mean propofol infusion rate at the end of anesthesia in group 1 was $72 \pm 34 \ \mu g \cdot kg^{-1} \cdot min^{-1}$.

The duration of preoperative fasting, as well as the

amount of iv fluid administered pre- and postoperatively, were similar in all four groups. None of the patients required any supplemental analgesic, antihypertensive, β -blocking, anticholinergic, or inotropic drugs to maintain hemodynamic stability during the intraoperative period.

Emergence and recovery data are summarized in table 3. Compared to the desflurane-alone group (group 4), the emergence times from the end of administration of the anesthetic to spontaneous eye-opening, response to simple commands, and ability to state their name and date of birth were significantly longer in the patients receiving propofol-nitrous oxide for maintenance of anesthesia. However, the times from arrival in the recovery room to sitting, standing, walking, and tolerating oral fluids and to being judged fit for discharge were similar in all four treatment groups. Patients in all four groups had significantly lower DSST scores (10-20% below baseline values) at 30 and 60 min after surgery; the scores returned to baseline during the remainder of the evaluation period. There were no significant differences in the DSST scores among the four groups during the 120-min evaluation period. Patients also rated their sedation (using VAS) as significantly greater than preoperative levels (45-60 mm

TABLE 2. Incidence of Complications Associated with Induction of Anesthesia in the Four Treatment Groups

	Propofol		Desflurane	
	Group 1	Group 2	Group 3	Group 4
Pain on injection Apnea	30 (7) 0	52 (12) 4 (1)	0* 17 (4)	0* 26 (6)*
Breathholding	ŏ	0	26 (6)*	39 (9)*
Laryngospasm	0	0 (9)	4(1)	4(1)
Coughing Excess secretions Spontaneous movements	0 17 (4)	9 (2) 0 26 (6)	30 (7)* 4 (1) 30 (7)	22 (5)* 9 (2) 48 (11)

Values are percentage of occurrences; number of occurrences is in parentheses.

^{*} Significantly different from control (group 1), P < 0.05.

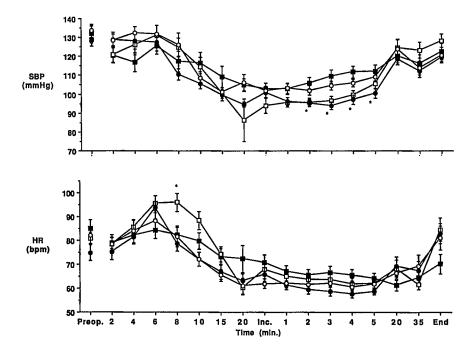


FIG. 1. Systolic blood pressure (SBP) and heart rate (HR) from immediately prior to induction (Preop.) to 20 min after induction of anesthesia; from skin incision (Inc.) to 35 min after skin incision; and at the end of anesthesia (End). Values are means \pm SEM. Symbols represent propofol control (solid squares), propofol/desflurane–N₂O (open squares), desflurane–N₂O (solid circles), and desflurane–O₂ (open circle). *P < 0.05, from propofol control (group 1).

vs. 10-15 mm) in all four groups after 30 min of recovery. Sedation scores were stable at 20-40 mm for the remainder of the evaluation period. Again, there were no significant differences between the four groups at any time point. However, 30 min after emergence from anesthesia, patients who received desflurane in oxygen (group 4) were judged by the blinded research nurse to be less sedated (fig. 3).

The groups in which anesthesia was induced and maintained with propofol reported a significantly lower incidence of moderate or severe nausea to the independent blinded observer than did any of the other three groups (fig. 4). The desflurane–nitrous oxide group (group 3) experienced significantly more episodes of postoperative vomiting and also had a significantly greater requirement for antiemetic therapy in the postoperative period than did the control patients (group 1). Opioid analgesic requirements, pain VAS scores, and hemodynamic values during recovery, were similar for all four anesthetic groups.

Discussion

Induction of anesthesia by inhalation of desflurane was rapid. This agent appeared to possess airway irritant properties; problems with breath-holding, apnea, and laryngospasm, however, were easily overcome by using positive pressure and by gently assisting ventilation. In contrast with the absence of any respiratory irritation found in a study of volunteers breathing desflurane at inspired concentrations of 1.8 and 5.4%, the occurrence of airway irritation in the current study is consistent with the findings of a previous study in patients in whom a

similar induction technique was used⁸ and with the problems reported when desflurane was used for inhalation induction of anesthesia in children.⁹ Similarly, a vital-capacity rapid-inhalation induction technique (the "singlebreath" technique) using higher inspired concentrations of desflurane also was associated with airway irritation.^{10,11} Had the inspired concentration of desflurane been increased more slowly, we might not have observed as many respiratory problems. However, a modified technique would likely have resulted in a slower induction of anesthesia. Since our study was not blinded, observer bias also might have altered the reporting of induction complications.

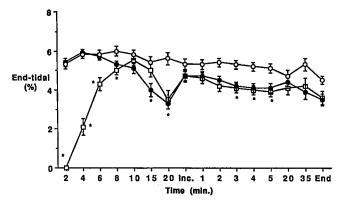


FIG. 2. End-tidal desflurane concentration from 2 to 20 min after induction of anesthesia; at skin incision (Inc.) to 35 min after incision; and at the discontinuation of desflurane administration (End). Values are means \pm SEM. Symbols represent propofol/desflurane–N₂O (open squares), desflurane–N₂O (solid circles), and desflurane–O₂ (open circles). *P < 0.05, from desflurane/O₂ (group 4).

TABLE 3. Emergence and Recovery Times (minutes) in the Four Treatment Groups

	Group 1	Group 2	Group 3	Group 4
Open eyes	7.3 ± 3.9	5.1 ± 1.9	5.9 ± 2.7	4.5 ± 2.1*
Squeeze hand	8.3 ± 3.9	6.4 ± 2.4	7.0 ± 2.7	5.1 ± 2.3*
Give name	9.8 ± 4.2	8.0 ± 2.8	8.7 ± 3.2	6.7 ± 2.8*
Give date of birth	10.2 ± 4.7	8.3 ± 2.9	9.0 ± 3.5	6.7 ± 2.9*
Sit up in chair	89 ± 30	101 ± 37	94 ± 41	89 ± 43
Stand up alone	98 ± 40	115 ± 65	103 ± 43	93 ± 42
Tolerate fluids	108 ± 54	142 ± 73	129 ± 65	93 ± 33
Walk to bathroom	155 ± 57	187 ± 75	145 ± 47	158 ± 67
Fit for discharge	199 ± 75	204 ± 82	196 ± 51	215 ± 57

Values are mean ± SD.

Induction of anesthesia was faster when desflurane was administered with 60% nitrous oxide. The induction times in the current study were consistent with other investigations using a similar induction technique in adults and children. However, the use of nitrous oxide in our study did not appear to influence the incidence of side effects during induction.

The frequency of pain on injection during induction of anesthesia with propofol (over 40% of the patients in groups 1 and 2) was greater than previously reported.⁵ In previous investigations, the frequency of pain on injection appeared to be related to the site as well as the size of the vein into which the propofol was injected.^{5,12} However, the incidence of pain on injection can be decreased by the co-administration of lidocaine.¹³

The end-tidal concentration of desflurane required to maintain blood pressure and heart rate within 15–20% of baseline values was significantly higher without nitrous oxide. This is consistent with the previously reported reduction of the desflurane anesthetic requirement by nitrous oxide. The average maintenance propofol infusion rate (111 \pm 46 $\mu g \cdot k g^{-1} \ min^{-1}$) was consistent with propofol requirements reported previously by our group, as well as by other investigators who administered it as part of a "balanced" anesthetic technique. 5,14,15

Only minor differences were observed in intraoperative hemodynamic parameters among the four treatment groups. Heart rate and blood pressure values were easily maintained within 15–20% of baseline values by regulation of the administered concentration of the anesthetic agent. The higher heart rate values in group 2 during the early postinduction period occurred at a time when the expired desflurane concentration was still rising (fig. 2) and may reflect a period of "light" anesthesia as the effects of the induction dose of propofol were wearing off (as a result of redistribution and elimination). No supplemental opioid analgesics or vasoactive medications were required for the maintenance of hemodynamic stability.

As would be predicted based on its low solubility,^{1,2} recovery from desflurane anesthesia was rapid. In fact,

emergence from anesthesia maintained with desflurane alone was significantly faster than was that after a propofol-nitrous oxide technique. The emergence times in the current study were similar to previously reported times after propofol-nitrous oxide and desflurane-nitrous oxide anesthesia. ^{5,7,16,17,18} There was no significant difference among the four groups with respect to the times required to reach later recovery milestones. It is possible that the intraoperative administration of fentanyl may have masked potential differences among the groups.

During the early recovery period, sedation (as subjectively evaluated by a blinded observer) appeared to be greater in patients receiving propofol for maintenance of anesthesia. However, the patients' own assessment of their level of sedation (using VAS scores) as well as the results of the DSST were similar among the four treatment groups. The reason for these differences in subjective (observer) and objective (patient) evaluations of sedation is not entirely clear. Although VAS have been demonstrated to be reliable in evaluating subjective feelings,⁶ it appears that the differences among the treatment groups were subtle and were probably of little, if any, clinical significance.

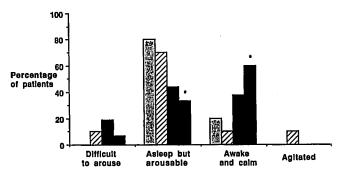


FIG. 3. Level of sedation as rated by an observer who was blinded as to the anesthetic treatment, 30 min after emergence from anesthesia. Symbols represent propofol control (bars with light shading), propofol/desflurane- N_2O (hatched bars), desflurane- N_2O (bars with dark shading), and desflurane- O_2 (solid bars). *P < 0.05, from control (group 1).

^{*} Significantly different from control (group 1), P < 0.05.

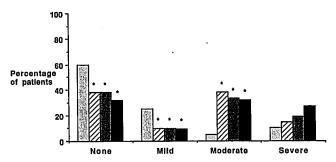


FIG. 4. Percentage of patients with no nausea or with mild, moderate, or severe nausea 30 min after emergence from anesthesia. Symbols represent propofol control (bars with light shading), propofol/desflurane- N_2O (hatched bars), desflurane- N_2O (bars with dark shading), and desflurane- O_2 (solid bars). *P < 0.05, from control (group 1).

The incidence and degree of nausea was significantly greater in all three desflurane treatment groups than in the propofol-nitrous oxide group. Furthermore, the combination of desflurane and nitrous oxide was associated with a significantly greater incidence of vomiting and a greater requirement for antiemetic medication. The incidence of nausea in the desflurane-treated patients was consistent with the incidence reported after laparoscopic procedures in studies involving volatile agents. 19 It has been noted previously that the incidence of postoperative nausea and emesis with propofol is low, 5,20 a characteristic that may be particularly beneficial for outpatient procedures associated with a high risk of postoperative nausea and vomiting. It is possible, however, that the incidence of postoperative nausea and vomiting after propofol and desflurane anesthesia would be equivalent after nonlaparoscopic procedures. Intraoperative administration of fentanyl also may have influenced the incidence of postoperative nausea and vomiting in our study; however, this would have affected all four groups to an equal degree. Patients who required postoperative opioid analgesic medications in the early recovery period were excluded from the assessment of nausea and vomiting.

This study can be criticized in that the investigators were not blinded as to the anesthetic technique being used. Unfortunately, blinding the study would have been virtually impossible because of the comparisons being made between iv and inhalational techniques. It might have been possible to blind the use of nitrous oxide, although the differences in desflurane requirements with and without nitrous oxide usually would have indicated whether it was being used or not. Although care was taken to use objective end-points and to use automated devices for data collection, the possibility of observer bias cannot be ruled out in this study. All the postoperative evaluations were performed by a blinded observer.

The study could be further criticized in that the inspired concentration of desflurane and the maintenance

propofol infusion rate were adjusted in response to hemodynamic variables and clinical signs of inadequate anesthesia (e.g., diaphoresis or lacrimation). As a consequence of alterations in the amount of desflurane delivered during the induction and maintenance periods, it is difficult to relate measurements of end-expired concentrations to the minimum alveolar concentration (MAC) because of inadequate equilibration. In the absence of an objective monitor of the "depth of anesthesia," our study was designed to mimic the clinical situation by allowing the anesthesiologist to titrate the anesthetic agent using clinical signs and to maintain hemodynamic stability. In an open study, this titration methodology allows for potential observer bias.

Finally, the use of intraoperative fentanyl to blunt the hemodynamic responses to tracheal intubation and skin incision may have influenced the emergence profiles. However, we wanted to minimize pain on emergence from anesthesia because of concerns for patient comfort and because the administration of potent opioid analgesics in the early postoperative period would have interfered with our assessment of cognitive function.

It has been reported that recovery after anesthesia with propofol and nitrous oxide compares favorably with that after thiopental—isoflurane—nitrous oxide in patients undergoing superficial surgical procedures.⁵ In addition, we have also reported that maintenance of anesthesia with desflurane—nitrous oxide caused less residual impairment of cognitive function than did isoflurane—nitrous oxide after outpatient anesthesia.³ Desflurane appears to offer advantages over isoflurane when used for maintenance of anesthesia during outpatient surgical procedures. Surprisingly, emergence times after desflurane compare favorably to propofol when used during ambulatory surgery.

In conclusion, induction of anesthesia by inhalation of desflurane is rapid, but the incidence of respiratory irritation may limit widespread acceptance of this induction technique. Early recovery from outpatient anesthesia with desflurane appears to be more rapid than with propofolnitrous oxide. However, maintenance of anesthesia with propofol was associated with a lower incidence of postoperative nausea in patients undergoing laparoscopic procedures. Overall discharge times after outpatient anesthesia with desflurane and propofol were similar in this patient population.

The authors would like to thank the residents and staff, and in particular Dr. S. Mark Poler and Dr. Mathew Bodner, of the Department of Anesthesiology for their cooperation during this study. In addition, the support of Dr. Michael Damask and Tom Genna of Anaquest is appreciated. Finally, the authors would like to thank our research nurse, Linda Kratz, for performing the patient evaluations and Michael Shapiro for his assistance with our data analysis.

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