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Supplemental Oxygen Reduces the Incidence of Postoperative Nausea and Vomiting

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Background: **Despite new anesthetic drugs and antiemetics, particularly 5-hydroxytryptamines, the incidence of postoperative nausea or vomiting remains between 20% and 70%. The authors tested the hypothesis that supplemental perioperative oxygen administration reduces the incidence of postoperative nausea or vomiting.**

Methods: **Patients undergoing colon resection were anesthetized with fentanyl and isoflurane. During and for 2 h after surgery they were randomly assigned to (1) 30% oxygen, balance nitrogen (n = 119); or (2) 80% oxygen, balance nitrogen (n = 112). The incidence of nausea or vomiting during the first 24 postoperative hours was evaluated by nurses blinded to group assignment and oxygen concentration. Data were analyzed with unpaired *t* or Mann–Whitney U tests. Results are presented as means ± SD; $P < 0.05$ was considered significant.**

Results: **Factors known to influence nausea and vomiting were comparable in the two groups. Perioperative oxygen saturation was well within normal limits in each treatment group; saturations the first postoperative morning were comparable in each group. Supplemental oxygen reduced the incidence of postoperative nausea or vomiting from 30% in the patients given 30% oxygen to 17% in those given 80% oxygen ($P = 0.027$).**

Conclusions: **Supplemental oxygen reduced the incidence of postoperative nausea or vomiting nearly twofold after colorectal surgery. The mechanism by which oxygen administration reduces the incidence of these postoperative sequelae remains unknown but may be related to subtle intestinal ischemia. Because oxygen is inexpensive and essentially risk-free, supplemental oxygen appears to be an effective method of reducing postoperative nausea and vomiting. (Key words: Anesthesia; antiemetics; surgery.)**

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THE incidence of postoperative nausea or vomiting remains between 20% and 70%, despite introduction of new antiemetic medications and short-acting opioids and anesthetics.[1–4] Not only are nausea and vomiting unpleasant for patients, but this "minor" complication may increase the risk of aspiration pneumonia because recovery of normal airway reflexes is often delayed in postoperative patients. Furthermore, nausea and vomiting are costly, being the leading cause of unexpected admission following planned day surgery.[5]

The incidence of postoperative nausea or vomiting depends on numerous nonanesthetic factors including the operative procedure, duration of surgery, age, gender, obesity, anxiety, gastroparesis, and history of motion sickness or previous postoperative nausea.[6,7] Important anesthetic-related factors include the type and dose of preanesthetic medication, ventilation techniques that promote gastric distension, postoperative pain, oral intake, and the amount of opioid required to treat postoperative pain.[1,4,8,9] General anesthesia is associated with a higher incidence of postoperative nausea or vomiting than regional anesthesia.[3,10] Inhaled anesthetics, in turn, cause more nausea than intravenous drugs.[1,6–8,11,12]

There is little doubt that nitrous oxide *per se* is eme-

togenic.[13] For example, virtually all nonsurgical volunteers given more than 60% nitrous oxide rapidly become nauseated and vomit.[14] Furthermore, recent large meta-analyses implicate nitrous oxide as a major cause of postoperative nausea and vomiting.[15,16] These results, though, were questioned in a letter to the editor by Overdyk and Roy,[17] who pointed out that the data equally well supported the theory that oxygen *per se* was antiemetic.

There is no established mechanism by which variation in perioperative inspired oxygen concentration would alter postoperative nausea and vomiting, which have a peak incidence many hours after surgery. However, we recently participated in a multicenter trial evaluating the effects of supplemental perioperative oxygen on the incidence of surgical wound infection (unpublished data). We therefore took this opportunity to test the hypothesis that the incidence of postoperative nausea and vomiting is less in patients given 80% than 30% perioperative oxygen. As secondary outcomes we investigated severity of early and late nausea and the overall incidence of nausea in 24 h.

Methods

With approval of the Ethics Committee of the Donauspital-SMZO, Vienna, we studied 231 patients undergoing elective colon or rectum resection scheduled to last at least 2 h. The study was restricted to patients aged 18 to 80 yr who did not have a history of fever, infection, obesity, antiemetic drugs or emesis prior to surgery. All were given routine prophylactic antibiotics.

Patients were orally premedicated with lormetazepam 1 mg and ranitidine 150 mg approximately 2 h before surgery. Anesthesia was induced with sodium thiopental (3–5 mg/kg), vecuronium (0.1 mg/kg), and fentanyl (1–3 μg/kg). The patients' tracheas were intubated and their lungs mechanically ventilated at a tidal volume of 10 ml/kg to maintain end-tidal carbon dioxide partial pressure (P_{CO_2}) near 35 mmHg. Anesthesia was subsequently maintained with fentanyl, vecuronium, and isoflurane (approximately 0.9%) in a carrier gas. Blood pressure was maintained within 20% of preinduction values by adjusting isoflurane concentration or administration of fluids to avoid longer periods of hypotension.

The carrier gas was randomly assigned: (1) 30% oxygen, balance nitrogen (n = 119); or (2) 80% oxygen, balance nitrogen (n = 112). Thus, neither group was given nitrous oxide. Inspired gas during surgery was provided by a conventional anesthesia machine *via* a circle circuit. Fresh gas flows were adjusted as necessary to maintain the designated inspired oxygen concentration as determined by a Cicero EM monitor (Draeger, Luebeck, Germany).

Forced-air warming was used as necessary to keep distal esophageal temperature near 36°C. Nasogastric tubes were generally not used. Upon completion of surgery, the neuromuscular block was antagonized with 0.4 mg glycopyrrolate and 2.5 mg neostigmine. Participating patients were aggressively hydrated during and after surgery. Specifically, we administered 15 $ml \cdot kg^{-1} \cdot h^{-1}$ of crystalloid throughout surgery. Additionally, blood loss was replaced with crystalloid at a 4:1 ratio or colloid at a 2:1 ratio to maintain normal intravascular volume. Fluid was then given at a rate of 3.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for the first 24 postoperative hours; additional fluid was given as necessary to maintain urine output exceeding 1 $ml \cdot kg^{-1} \cdot h^{-1}$.

During the first 2 h of recovery, oxygen at the specified concentration was given at a rate of 10 l/min through an adhesive mask connected to a nonrebreathing system (AirCare, Apotheus Laboratories, Lubbock, TX). The gas supply for this valved system was provided by an oxygen blender; consequently, patients breathed exactly the specified oxygen concentration during recovery. However, additional oxygen was provided, as necessary, to maintain oxygen saturation of at least 95% in all patients. Arterial blood was sampled for gas analysis 1 h after introduction of anesthesia and 2 h after admission to the postanesthesia care unit. Oxygen saturation also was measured by a pulse oximeter on the first day after surgery.

The treatment randomization was based on computer-generated codes that were maintained in sealed, opaque envelopes until just after induction of anesthesia. The anesthesiologist was aware of the administered oxygen concentration. However, patients and surgeons and the nurses reporting postoperative nausea and vomiting were blinded to group assignments and actual inspired oxygen concentration. Intraoperative blinding was maintained by positioning cardboard shields over the flowmeters on the anesthesia machine and the inspired oxygen concentration monitor. The inspired oxygen concentration was similarly blinded postoperatively by shielding the oxygen blender.

Postoperative pain was treated with piritramid, a synthetic opioid with a potency roughly 0.8 times that of morphine. At the discretion of the attending surgeon, feeding was started on the first postoperative day with

clear liquid and solid food after bowel function was established. No routine antiemetic drugs were given. However, patients were given ondansetron, 4 mg intravenously, after 30 min of nausea or two episodes of vomiting, or upon request of the patient or attending physician. Other antiemetics were then permitted if nausea or vomiting continued.

Measurements

Appropriate morphometric and demographic characteristics of each treatment group were tabulated. Preoperative laboratory values and historical factors likely to influence postoperative nausea and vomiting were recorded. These factors included history of previous postoperative nausea or vomiting, smoking history, preoperative hemoglobin, coexisting systemic diseases, and substantial alcohol use (more than two drinks per day).

Hemodynamic responses, oxygen saturation, end-tidal P_{CO_2}, anesthetic concentrations, inspired oxygen concentration, and esophageal temperature were measured during anesthesia every 15 min. In the postanesthesia care unit, hemodynamic responses and oxygen saturation were recorded at 30-min intervals. In addition to the routine use of a pulse oximeter during anesthesia, we did blood-gas analyses 1 h after introduction of anesthesia and 2 h after admission to the postanesthesia care unit. Oxygen saturation was also measured with a pulse oximeter on the first postoperative morning.

As recommended,[1,18,19] nausea and vomiting incidence and severity were determined at intervals over a 24-h period starting upon arrival in the postoperative care unit. Nausea and vomiting were evaluated while patients were awake and over the intervals from 0 through 6 h and 6 through 24 h. At the end of each query period, the number of vomiting episodes was determined from the nursing records. Vomiting within each period was scored as *none* if there was none, *mild* if there was one episode, *moderate* if there were two or three episodes, and *severe* if vomiting occurred more than three times. Patients were asked to rate nausea on a three-point scale as *none*, *mild*, or *severe*.

Data Analysis

Potentially confounding factors in the two treatment groups were compared with unpaired, two-tailed *t* tests. End-tidal isoflurane level and intraoperative mean arterial pressure were first averaged over the anesthetic period in each patient on each study day. Subsequently, these values were averaged among the patients in each group.

For our primary outcome, the combined nausea and vomiting analysis, any score for nausea or vomiting exceeding *none* was considered positive. Combined nausea and vomiting was compared with a chi-square analysis. We separately evaluated the nausea and vomiting scores for the 0 through 6 h and 6 through 24 h periods. As is usual in studies such as this one, the highest vomiting severity score and the highest severity nausea score was used for analysis. The maximum severity scores for nausea and for vomiting were analyzed using a Mann–Whitney U test. Results are presented as means ± SDs; $P < 0.05$ was considered statistically significant.

Additionally, we used multiple regression to assess the fractional contribution of potentially related factors, including morphometric and demographic characteristics, history of smoking, history of alcohol abuse, history of previous postoperative nausea and vomiting, and duration of surgery. This generalized mixed-effects model allowed us to determine the relative contributions of continuous, ordinal, and nominal factors. Perioperative oxygen concentration and factors having a univariate *P* value < 0.25 were entered into a stepwise regression; factors contributing with a *P* value < 0.10 were retained in the analysis.

Results

Morphometric and demographic factors, hemodynamic responses, and anesthetic management were comparable in the patients given 30 and 80% oxygen (table 1). Isoflurane concentration did not differ significantly between patients suffering postoperative nausea or vomiting and those who did not (0.8 ± 0.2 *vs.* 0.9 ± 0.2%, $P = 0.15$); mean arterial pressure was also similar in patients with and without postoperative nausea or vomiting (94 ± 11 *vs.* 95 ± 10 mmHg, $P = 0.90$). Potential confounding factors and oxygen saturation on the first postoperative morning were comparable in the two treatment groups. There were no differences in body core temperature and in administered fluid during anesthesia and in the postanesthesia care unit between the two study groups. The amount of opioid used for postoperative pain relief was comparable in the two groups (table 2).

None of the patients required rapid-sequence induction, and none was given epidural analgesia. Pulmonary function was not formally evaluated in this study. However, no pulmonary complications (*i.e.*, clinically important atelectasis) were detected that could be attributed

Table 1. Demographic and Morphometric Factors and Anesthetic Management

	30% Oxygen	80% Oxygen	*P*
Number of women/total	57/119	41/112	0.11
Age (yr)	60 ± 13	59 ± 14	0.77
Weight (kg)	75 ± 15	75 ± 15	0.94
Height (cm)	169 ± 9	171 ± 9	0.09
Preoperative hemoglobin (mg/dl)	13.3 ± 2.3	12.9 ± 2.4	0.37
Intraoperative fentanyl (μg/kg)	11.4 ± 6.6	10.2 ± 4.7	0.24
Average end-tidal [isoflurane] (%)	0.9 ± 0.2	0.9 ± 0.2	0.45
Average intraoperative mean arterial pressure (mmHg)	93 ± 10	96 ± 10	0.14
Intraoperative SaO_2 (%)	98 ± 1	100 ± 1	0.96
Duration of surgery (h)	3.0 ± 1.3	2.7 ± 1.0	0.29
Postoperative Sa_{O_2} (%)	96 ± 2	98 ± 2	0.90

Data are mean ± SD. There were no statistically significant differences between the two study groups.

Sa_{O_2} = arterial oxygen saturation.

Table 2. Potential Confounding Factors

	30% Oxygen	80% Oxygen	*P*
Alcohol intake (%)	2%	5%	0.43
Current smokers (%)	38%	43%	0.73
History of previous postoperative nausea and vomiting (%)	6%	5%	0.85
Postoperative gastric tube (%)	17%	14%	0.73
Sp_{O_2} first postoperative morning (%)	94 ± 3	94 ± 3	0.99
Piritramid 0–6 h (mg)	18 ± 11	19 ± 10	0.41
Piritramid 6–24 h (mg)	25 ± 16	25 ± 15	0.84
Piritramid in first 24 h (mg)	46 ± 24	47 ± 29	0.60

Data are mean ± SDs. There were no statistically significant differences between the two study groups. Alcohol intake was defined by ≥2 drinks/day.

Sp_{O_2} = oxygen saturation as measured by pulse oximetry.

to supplemental oxygen. No patients assigned to 80% oxygen required supplemental oxygen to maintain oxygen saturation ≥ 95%; in contrast, 21 patients (17.6%) in the 30% group required additional oxygen during the immediate postoperative period. Six of them developed postoperative nausea or vomiting (five experienced nausea and one vomited). The fraction of patients in whom nasogastric tubes were inserted and the amounts of postoperative opioid piritramid given for treatment of pain were comparable in each group.

The overall incidence of postoperative nausea or vomiting in the first 24 postoperative hours was 30% in the patients assigned to 30% oxygen, but only 17% in those given 80% oxygen ($P = 0.027$). The incidence was thus nearly halved by supplemental oxygen. As a consequence, patients given 80% oxygen were less likely to rescue antiemetic medication (22% in the 30% oxygen group *vs.* 16% in the 80% group); however, this difference was not statistically significant. More than a third of the patients who developed postoperative nausea or vomiting were given antiemetic drugs (36% in the 30% group and 37% in the 80% group, $P = 0.96$) Only two patients in the 80% oxygen group vomited, whereas seven did in the 30% group ($P = 0.11$). The overall incidence of postoperative nausea or vomiting is shown in table 3, along with the values for each observation period.

The presence of a gastric tube, history of postoperative nausea or vomiting, alcohol use, gender, and intraoperative fentanyl dose showed univariate predictive probabilities < 0.25. These factors and oxygen randomization were thus entered into the stepwise regression. Oxygen concentration showed a probability of 0.03 in the multivariate regression; previous history of postoperative nausea or vomiting and gender each had $P < 0.01$. The P value for fentanyl was 0.05. The other factors contributed at a P value > 0.10 and were dropped from the analysis (table 4).

Discussion

Numerous factors are known to influence the incidence of postoperative nausea or vomiting.[1,7,9,12] No previous study, though, has specifically evaluated the effect of inspired oxygen concentration on the incidence of postoperative gastrointestinal complications. Our data indicate that supplemental perioperative oxygen produces a statistically significant and clinically important reduction in the incidence of postoperative nausea or vomiting.

The mechanism by which supplemental perioperative oxygen might reduce the incidence of postoperative nausea or vomiting remains unknown. A substantial effect of inspired oxygen concentration is particularly interesting, because supplemental oxygen administration was restricted to the intraoperative period and the first 2 h of recovery. In contrast, the peak incidence of nausea or vomiting was roughly 6 h after surgery—a time that is consistent with previous observations.[20]

It is well established that supplemental oxygen increases arterial and tissue oxygen partial pressures.[21] Oxygen partial pressures thus surely differed significantly during and immediately after surgery, although oxyhemoglobin saturation remained well within normal limits in both groups throughout anesthesia and the

Table 3. Nausea and Vomiting Incidence and Severity during the First 24 Postoperative Hours

		30% Oxygen	80% Oxygen	*P**
0–6 h after surgery	PONV (number/%)	18/15.1	9/8	0.141
	Nausea (number/%)	18/15.1	9/8	0.077
	None/mild/severe	101/10/8	103/8/1	
	Vomiting (number/%)	2/1.7	0	0.169
	None/mild/moderate/severe	117/0/0/2	112/0/0/0	
6–24 h after surgery	PONV (number/%)	24/22.2	11/9.8	0.045†
	Nausea (number/%)	21/17.6	10/8.9	0.066
	None/mild/severe	98/12/9	102/3/7	
	Vomiting (number/%)	7/5.9	2/1.8	0.108
	None/mild/moderate/severe	112/1/4/2	110/1/0/1	
Entire 24-h period	PONV (number/%)	36/30.3	19/17	0.027†
	Nausea (number/%)	33/27.7	18/16	0.034†
	None/mild/severe	86/20/13	94/11/7	
	Vomiting (number/%)	7/5.9	2/1.8	0.108
	None/mild/moderate/severe	112/1/4/2	110/1/0/1	

PONV = postoperative nausea and/or vomiting.

* *P* value for PONV calculated by chi-square test; severity of nausea or vomiting calculated by Mann-Whitney test.

† Statistically significant differences between the two study groups.

immediate postoperative period. Humans have no important reservoir for oxygen. We can thus assume that oxygenation was comparable in both patient groups after supplemental oxygen administration was discontinued. The observed identical oxygen saturations in each group on the first postoperative morning is consistent with this theory. Blood and tissue oxygenation must, therefore, have been comparable in the two groups during the period of maximal nausea or vomiting.

The three major pathophysiologic pathways triggering postoperative nausea or vomiting are a vestibulocochlear pathway, the central chemoreceptor trigger zone, and a local gastrointestinal pathway. Patients participating in this study presumably experienced little vestibulocochlear stimulation, and there is no reason to assume that oxygen administration altered this region. Global cerebral oxygenation was surely elevated in the patients given 80% oxygen. However, there is no known mechanism by which supranormal oxygen partial pressures directly influence the central chemoreceptor trigger zone.

Both nitrogen and oxygen diffuse slowly across the bowel wall, with oxygen passing at roughly twice the rate of nitrogen. Oxygen, though, is consumed by tissues and gut bacteria. Total bowel inflation might thus be greater in patients given 70% nitrogen than in those given 20%. This might influence postoperative nausea or vomiting because bowel distention releases 5-hydroxytryptamine, a well-known mediator of nausea and vomiting.[6,7,9]

An alternative mechanism is that—despite adequate arterial saturation—there were physiologically important differences in tissue oxygenation during and immediately after surgery in the two treatment groups. We speculate that relatively low partial pressures in the patients given 30% oxygen either (1) directly triggered release of mediators that facilitated subsequent nausea or vomiting or (2) injured tissue that subsequently released emetogenic factors. We have no basis for choosing one scenario over the other. However, the latter seems somewhat more likely.

The tissue at greatest risk of intraoperative injury in our patients may be the intestine. Intestinal tissue is highly metabolically active and has a notoriously poor tolerance for even brief periods of hypoxia or ischemia.[22] There are at least three major factors that augmented the chances of relative intestinal ischemia in our

Table 4. Predictors of Postoperative Nausea and Vomiting from a Generalized, Mixed-effects Model

Factor	Odds Ratio	95% Confidence Interval
Group assignment	0.45	0.23–0.90
Gender	0.40	0.20–0.80
PONV history	11.3	2.62–48.9
Fentanyl	0.999	0.998–1.000

Note: Morphometric and demographic characteristics, history of smoking, history of alcohol abuse, history of previous postoperative nausea and vomiting, and duration of surgery were entered into a generalized mixed-effects model. Perioperative oxygen concentration and factors having a univariate $P < 0.25$ were entered into the stepwise regression; factors contributing with a $P < 0.10$ were retained in the analysis.

patients. The first is that surgical stress, including that from laparotomy, decreases gastrointestinal blood flow.[23,24] Elevations of intraabdominal pressure to more than 20 mmHg also markedly reduce mesenteric and mucosal blood flow[25] and may be associated with gut ischemia.[26] The second factor is that anesthesia inhibits tonic thermoregulatory vasoconstriction,[27,28] thus increasing blood flow to peripheral tissues.[29,30] This arteriovenous shunt dilation is accompanied by compensatory splanchnic vasoconstriction.[31,32] The third factor is that our patients were undergoing intestinal resections: Retractor positioning, intestinal mobilization, and surgery *per se* probably all cause inadequate tissue perfusion in at least some regions.

A consequence of ischemia is release of 5-hydroxytryptamine (serotonin) from the intestine, where it acts as a local vasodilator.[33,34] Serotonin is among the most potent known triggers of nausea and vomiting. Consistent with this theory, excessive physical exertion transiently reduces splanchnic blood flow[35] and may cause relative gut ischemia. Nausea and vomiting under this condition are common, even if no defect in small intestine mucosal integrity has been observed.[36] Substantial regional tissue hypoxia is, of course, less likely in patients given 80% oxygen than in those given just 30%. A potential mechanism explaining our results is thus that supplemental oxygen administration minimizes regional intestinal hypoxia.

In the pulmonary circulation, hyperoxia causes vasodilatation, whereas the systemic circulation constricts.[37] Furthermore, induced high concentrations of oxygen produce a vagally mediated bradycardia that is sustained for about 30 min after the inspired oxygen concentration returns to normal.[38] It is also likely that vagal release of acetylcholine provokes intestinal vasodilatation.[39] Increased splanchnic perfusion would, of course, improve intestinal oxygenation and reduce release of 5-hydroxytryptamine.

Very high inspired oxygen concentrations (*i.e.*, 100%) directly injure pulmonary tissues over a period of a day or more.[40,41] High oxygen concentrations are also associated with absorption atelectasis,[42–46] although there is little evidence that even 100% perioperative oxygen causes clinically important pulmonary dysfunction.[47] Our data do not support clinically important complications related to 80% perioperative oxygen, because no pulmonary complications were observed and oxygen saturations on the first postoperative morning were comparable in the two groups.

The incidence of postoperative nausea or vomiting clearly depends on numerous morphometric and demographic factors, anesthetic management, and the site and duration of surgery. It is likely that the extent to which supplemental oxygen reduces postoperative nausea and vomiting is similarly influenced by these factors. However, inspired oxygen concentration remained a statistically significant predictor for postoperative nausea and vomiting even when likely confounding factors were entered into a generalized, mixed-effects model. Prophylactic administration of effective antiemetic drugs reduces the incidence of gastrointestinal complications and might, thus, reduce the relative effect of supplemental oxygen. However, antiemetic therapy is costly[48] and associated with a 3% incidence of complications.[49]

In summary, supplemental oxygen given intraoperatively and for 2 postoperative hours reduced the incidence of postoperative nausea or vomiting after colorectal surgery by 43% ($P = 0.027$). The mechanism by which oxygen administration reduces the incidence of these postoperative sequelae remains unknown but may be related to subtle intestinal ischemia. Because oxygen is inexpensive and essentially risk-free, supplemental oxygen appears to be an effective method of reducing postoperative nausea and vomiting.

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