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Optimal Oxygen Concentration during Induction of General Anesthesia

Lennart Edmark, M.D., D.E.A.A., Kamelia Kostova-Aherdan, M.D.,† Mats Enlund, M.D., Ph.D.,‡ Göran Hedenstierna, M.D., Ph.D.§*

Background: **The use of 100% oxygen during induction of anesthesia may produce atelectasis. The authors investigated how different oxygen concentrations affect the formation of atelectasis and the fall in arterial oxygen saturation during apnea.**

Methods: **Thirty-six healthy, nonsmoking women were randomized to breathe 100, 80, or 60% oxygen for 5 min during the induction of general anesthesia. Ventilation was then withheld until the oxygen saturation, assessed by pulse oximetry, decreased to 90%. Atelectasis formation was studied with computed tomography.**

Results: **Atelectasis in a transverse scan near the diaphragm after induction of anesthesia and apnea was 9.8 ± 5.2 cm^2 (5.6 ± 3.4% of the total lung area; mean ± SD), 1.3 ± 1.2 cm^2 (0.6 ± 0.7%), and 0.3 ± 0.3 cm^2 (0.2 ± 0.2%) in the groups breathing 100, 80, and 60% oxygen, respectively ($P < 0.01$). The corresponding times to reach 90% oxygen saturation were 411 ± 84, 303 ± 59, and 213 ± 69 s, respectively ($P < 0.01$).**

Conclusion: **During routine induction of general anesthesia, 80% oxygen for oxygenation caused minimal atelectasis, but the time margin before unacceptable desaturation occurred was significantly shortened compared with 100% oxygen.**

THE use of 100% oxygen during induction of general anesthesia has become a standard in many institutions, although preoxygenation was initially proposed as an optional precaution.[1] There are strong logical merits for this standard regarding maximal safety during the transition from an awake to an anesthetic state.[2] Lately, evidence has appeared that 100% oxygen is a major cause of atelectasis during the induction and maintenance of anesthesia.[3] Thirty percent oxygen during induction of anesthesia was not associated with atelectasis. This was true even in patients with a high body mass index.[4]

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* Chief of Staff, Department of Anesthesiology and Intensive Care, Central Hospital, Västerås, and Research Associate, Department of Medical Sciences, Clinical Physiology, University Hospital, Uppsala, Sweden. † Staff Radiologist, Department of Radiology, ‡ Senior Consultant/Lecturer, Department of Anesthesiology and Intensive Care, Central Hospital, Västerås, Sweden. § Professor in Clinical Physiology, Department of Medical Sciences, Clinical Physiology, University Hospital, Uppsala, Sweden.

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Address reprint requests to Dr. Edmark: Department of Anesthesiology and Intensive Care, Central Hospital, S-721 41 Västerås, Sweden. Address electronic mail to: lennart.edmark@ltvastmanland.se. Individual article reprints may be purchased through the Journal Web site, www.anesthesiology.org.

The current study was designed to see whether 80 or 60% oxygen would produce less atelectasis than 100% oxygen during anesthesia induction and to what extent lower oxygen concentrations during induction shorten the time to hypoxia, defined as a saturation of 90% or less, as measured by pulse oximetry. We aimed at finding a compromise between acceptable safety (apnea) time and minimum atelectasis formation.

Materials and Methods

Patients

The study was approved by the Regional Ethics Committee (Uppsala, Sweden), and all patients gave their written informed consent to participate. Thirty-six healthy, nonsmoking females with American Society of Anesthesiologists physical status class I or II and with a body mass index (weight in kilograms divided by the square of the height in meters) of 31 or less, scheduled for elective hysterectomy due to benign disease, were enrolled in the study. Preoperative evaluation was carried out the day before surgery. All patients had normal electrocardiograms. Their arterial oxygen and carbon dioxide tensions were normal (ABL 505; Radiometer, Copenhagen, Denmark), and their spirometry and lung volumes (by helium dilution technique), measured in the supine position, were also normal. Patients' oxygen uptake and carbon dioxide output at rest were measured, and major differences in metabolic rate between groups were excluded (integrative cardiopulmonary testing; Jaeger, Wurzburg, Germany). Patients were randomized to one of three oxygenation groups: 100, 80, or 60% oxygen (in nitrogen).

Anesthesia and Monitoring

Premedication consisted of acetaminophen 1 g orally, and five patients were also given 10 mg diazepam orally. Anesthesia was induced and measurements were performed at the Radiology Department before transport to surgery. The patients were hydrated with 200 ml Ringer's acetate and 300 ml of the colloid dextran before induction to reduce the risk of postinduction hypotension. The electrocardiogram and peripheral oxygen saturation (Spo_2) were continuously assessed during the procedure. End-tidal carbon dioxide ($FETCO_2$) and end-tidal oxygen concentrations ($FETO_2$) were continuously monitored. Blood pressure was measured intermittently. The monitor for all measurements was a CS 3 (Datex-Ohmeda, Helsinki, Finland).

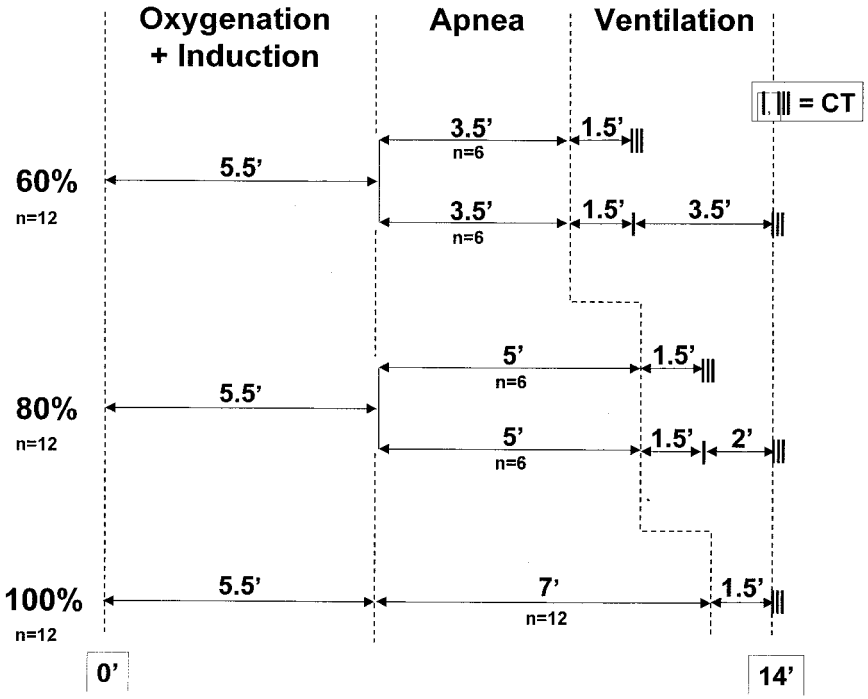

Fig. 1. Schematic presentation of study layout. The time points of computed tomography (CT) scanning are indicated with I or III, depending on the number of scans performed.

Data collection started when the patients began to breathe the randomly chosen oxygen concentration through a tightly fitted mask. After breathing spontaneously for 1 min, the patients were given bolus doses of fentanyl, 0.2 mg, and alfentanil, 1 mg. A target-controlled propofol infusion (Diprifusor; AstraZeneca, Macclesfield, Cheshire, United Kingdom) was started 2 min after the beginning of preoxygenation, aiming for an initial target blood concentration of 8 μg/ml, which was reduced to 6 μg/ml after tracheal intubation. Rocuronium, 30 mg, was given approximately 30 s after starting the propofol infusion to facilitate tracheal intubation. All patients lost consciousness and stopped breathing 30–60 s after the start of the propofol infusion.

The experimental procedure during anesthesia in the different groups is outlined in figure 1.

Ventilation

The strict preoxygenation period with normal spontaneous ventilation was 1 min. This period was followed by an induction phase with decreased spontaneous ventilation. An oropharyngeal airway was used to facilitate positive-pressure mask ventilation when spontaneous breathing ceased. The oxygenation continued until the patient was fully asleep and a steady state in end-tidal oxygen concentration was achieved. The end-tidal values of oxygen and carbon dioxide at steady state were noted before ventilation was stopped. The mask was removed, and the patient's trachea was immediately intubated. Five and a half minutes elapsed from the start of preoxygenation to the start of the intubation maneuver. No fresh gas was connected to the endotracheal tube during the period of apnea. The apnea time was defined as the period after stopping the ventilation until the saturation, as measured by pulse oximetry, had fallen to 90%.[5] The oxygen saturation was continuously registered. The fresh gas was connected to the patient, and manual ventilation with 40% oxygen in nitrogen was started in all three groups at the time when Spo_2 had decreased to 90%. The lowest oxygen saturation and the time when it occurred were registered, as was the time to return to a saturation of 96%. The patients breathed and were ventilated through the Mentell system (Anmedic, Vallentuna, Sweden), a hybrid low-flow anesthetic circuit.[6] This circuit has a built-in positive end-expiratory pressure (PEEP) of 3 cm H_2O. No other level of PEEP was used. A fresh gas flow of 6 l/min was used to prevent rebreathing during the period of preoxygenation and during the period of anesthesia induction. As spontaneous breathing ceased during induction, manual ventilation on mask began with the aim to keep end-tidal Pco_2 normal and fresh gas flow unaltered. This was achieved with tidal volumes of 7–10 ml/kg body weight and respiratory rates less than 10 breaths/min. After the period of apnea, the patient was connected to the respirator (Anmedic), and a standard ventilatory setting was used, consisting of a tidal volume of 10 ml/kg body weight and a respiratory rate of 12 breaths/min. A fresh gas flow of 6 l/min with 40% oxygen in nitrogen was used when ventilation was started after the apnea period. The fresh gas flow was lowered to approximately 1 l/min, still with 40% oxygen, to avoid hyperventilation, as soon as Spo_2 was raised to 96% and end-tidal carbon dioxide was normalized. Airway pressure did not exceed 25 cm H_2O in any patient.

Computed Tomography

The lungs were studied with computed tomography (CT; Siemens Somatom HiQ; Siemens, Erlangen, Germany). The patients were placed in the supine position, with the arms parallel to the body, horizontally on the CT table. All CT scans were carried out in apnea at the end of expiration. Three scans were made: a basal scan 10 mm above the dome of the right diaphragm, a scan at the hilus, and one at the apex of the lungs. A control CT was taken in the conscious patient to exclude any atelectasis before anesthesia. During anesthesia, the three scans were performed as soon as ventilation was started after apnea. In the 100% group, this occurred approximately 14 min from the start of preoxygenation. As the three groups were apneic for different time periods until the saturation had fallen to 90%, depending on their initial inspiratory oxygen concentration, the 60 and 80% groups were split into two subgroups each. One subgroup (first six patients) had their three scans performed immediately after controlled ventilation was started, as was the situation in the 100% group. The other subgroup (last six patients) had only a basal scan immediately after apnea, and then three scans were undertaken approximately 14 min after start of preoxygenation. Thereby, scans were taken at the same time point in the 60 and 80% subgroups as in the 100% group. Performing the scanning procedure including positioning took 1.5 min.

The radiologist was unaware of the group affiliation of the individual patient when making the examination of the CT scans. Calculations were made using the region of interest presented in the Somatom computer program. For tracing the atelectasis, the window level and width were set at −500 and ± 1,500 Hounsfield units, respectively. Atelectasis was defined as areas with attenuation values between −100 and +100 Hounsfield units and was expressed in square centimeters and in percent of the total lung area.[7] The dorsal border was traced manually between the atelectasis and the pleura. The ventral border was drawn a few centimeters above the dense area, and the atelectasis was calculated by the region of interest program. The total lung area was calculated as the inner thorax area, excluding the heart and great vessels. Each region of interest was redrawn two times, and the mean value was used.

Statistical Analysis

The sample size for this study was based on the knowledge from earlier studies[8,9] and the assumption that a reduction of atelectasis of 50% or more would be of clinical importance. We did not assume the main variables in the study, *i.e.*, the amount of atelectasis and the apnea time, to have a normal distribution. Therefore, and since three groups were involved, the Kruskal-Wallis nonparametric one-way analysis of variance was applied, and a P value of 0.01 or less was considered significant.

Results

The three study groups were similar in demographic and physiologic data collected the day before the investigation (table 1).

No patient presented with atelectasis in the awake state before anesthesia induction. During anesthesia, immediately after the period of apnea, the mean atelectasis area in the basal scan was close to 10 cm^2 (5.6%) in the 100% group compared to 1.3 cm^2 (0.6%) and 0.3 cm^2 (0.2%) in the 80 and 60% groups, respectively ($P < 0.001$; table 2 and fig. 2). The atelectatic area in the hilus scans was much smaller, although the relation between the three groups was the same as at the basal cut. The apex scans indicated less than 0.1 cm^2 atelectasis in any group.

The time from start of preoxygenation until apnea commenced, corresponding to the time of oxygenation with either 100, 80, or 60% oxygen, was approximately 5.5 min, with no difference between the groups (table 2).

All patients had an Spo_2 of 99% at the beginning of the apnea period. The mean time for Spo_2 to decrease to 90% during the apnea was close to 7 min in the 100% group, 5 min in the 80% group, and 3.5 min in the 60% group ($P < 0.01$; table 2 and fig. 3). The pattern in which Spo_2 decreased during the period of apnea is shown in figure 4. Immediately after apnea, ventilation was commenced, and then CT scanning was performed and completed in 1.5 min. Thus, the total time from the initiation of preoxygenation to the completion of CT scanning was 14 min in the 100% oxygen group and 12 and 10.5 min in the 80 and 60% oxygen groups, respectively.

There was no statistically significant change in the atelectasis formation in the 80 and 60% oxygen groups

Table 1. Demographic Characteristics and Preoperative Values in Patients Given 100%, 80%, or 60% Oxygen during Anesthesia Induction

	F_{IO_2} (%)		
	100	80	60
Patients (n)	12	12	12
Age (yr)	44 ± 9	48 ± 7	47 ± 6
Height (cm)	166 ± 6	164 ± 5	166 ± 6
Weight (kg)	67 ± 7	69 ± 10	70 ± 11
Body mass index	24.3 ± 2.4	25.4 ± 3.4	25.3 ± 3.3
Hemoglobin (g/l)	121 ± 14	132 ± 15	124 ± 9
Pao_2 (mmHg)	92.3 ± 5.3	87.8 ± 9	90 ± 9
$Paco_2$ (mmHg)	38.3 ± 2.3	36.8 ± 3	38.3 ± 3
FEV_1 (l)	3.2 ± 0.4	3.0 ± 0.8	3.1 ± 0.5
VC (supine, l)	3.9 ± 0.5	3.6 ± 0.9	3.8 ± 0.4
FRC (supine, l)	1.8 ± 0.3	1.9 ± 0.3	1.9 ± 0.3
RV (supine, l)	1.7 ± 0.3	1.6 ± 0.3	1.8 ± 0.3
$\dot{V}o_2$ (ml/min)	250 ± 26	263 ± 47	241 ± 37
$\dot{V}co_2$ (ml/min)	215 ± 42	236 ± 52	213 ± 40

Data are presented as mean ± SD.

FEV_1 = forced expired volume in 1 s; F_{IO_2} = fractional inspired oxygen tension; FRC = functional residual capacity; $Paco_2$ = arterial carbon dioxide tension; Pao_2 = arterial oxygen tension; RV = residual volume; VC = vital capacity; $\dot{V}co_2$ = carbon dioxide output; $\dot{V}o_2$ = oxygen uptake.

Table 2. Oxygenation and Apnea Times, End-tidal Oxygen Concentration, and Atelectasis in Patients Given 100%, 80%, or 60% Oxygen

	F_{IO_2} (%)		
	100	80	60
Oxygenation (s)*	328 ± 42	323 ± 30	324 ± 42
ET_{O_2}, just before apnea (%)	93 ± 1.4	75 ± 1.9	53 ± 1.3
Apnea time, time to reach 90% saturation (s)	411 ± 84†	303 ± 59	213 ± 69
Range	239–528	171–380	128–360
n	11‡	12	12
Atelectasis, cm^2 basal scan after apnea	9.8 ± 5.2†	1.3 ± 1.2	0.3 ± 0.3
Range	0.8–19.3	0.1–3.2	0.0–0.9
n	12	11‡	12
Atelectasis, hilus scan after apnea (cm^2)	2.4 ± 2.0	0.4 ± 0.4	0.2 ± 0.4
n	12	6	6
Atelectasis, basal scan (cm^2)			
After apnea	NA	0.6 ± 0.5	0.2 ± 0.2
After 14 min		0.4 ± 0.3	0.3 ± 0.3
n		6	6

Data are presented as mean ± SD (and range).

*Includes 1 min of preoxygenation.

†$P < 0.01$ comparing the three groups regarding apnea time, and the amount of atelectasis at the basal scan immediately after apnea (Kruskal–Wallis test).

‡Missing value due to technical reasons.

ET_{O_2} = end-tidal oxygen concentration; F_{IO_2} = fractional inspired oxygen tension.

immediately after apnea compared with when CT scans were repeated at 14 min (corresponding to the time of CT in the 100% oxygen group; table 2).

The lowest saturation, the time at which Sp_{O_2} started to increase, the time to recover to normal saturation, and the difference between end-tidal P_{CO_2} before and immediately after apnea are summarized in table 3.

Discussion

A major finding in the current study was that breathing 100% oxygen during preoxygenation and anesthesia induction caused significantly more atelectasis formation than did breathing 80 or 60% oxygen. This atelectasis causes intrapulmonary shunting,[10] and the clinical impression is that it contributes to postoperative pulmonary complications.[11]

The anesthetic procedure was uneventful in all but one patient. This patient showed signs of heavy mucus production during the operation, and suction of the endotracheal tube was carried out several times. It must be observed that this patient had more atelectasis (19.3 cm^2) at the basal scan directly after apnea than did any other patient. However, even after the exclusion of this patient, the atelectatic area (now 9.0 ± 4.5 cm^2) remained significantly larger than the areas in the 80 or 60% oxygen groups ($P < 0.01$). This patient also had the shortest apnea tolerance (239 s) in comparison with the group average of 411 s. Excluding this patient from the results in the 100% group increased the mean apnea tolerance to 428 s in the 100% group. We chose not to exclude her, as she most certainly demonstrates a major mechanism in the development of a larger than normal amount of atelectasis during a routine induction of anesthesia.

An expected finding was that the safe time of apnea, defined as keeping peripheral oxygen saturation more

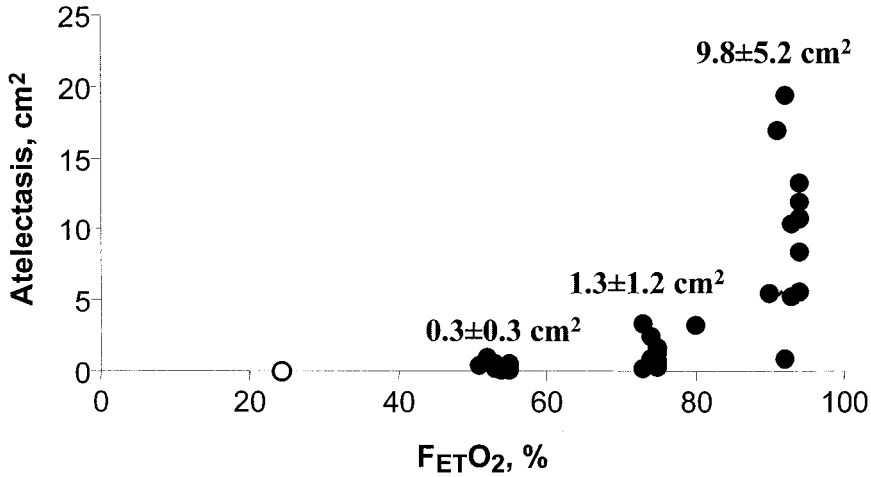

Fig. 2. Atelectasis 1 cm above the diaphragm in individual patients (filled circles) after induction of anesthesia and a period of apnea in relation to their end-tidal oxygen concentration (F_{ETO_2}) just before the period of apnea. The results are compared with data (open circle) taken from Rothen *et al.*[3] in which subjects were ventilated with 30% oxygen in nitrogen. Mean ± SD is presented above each group.

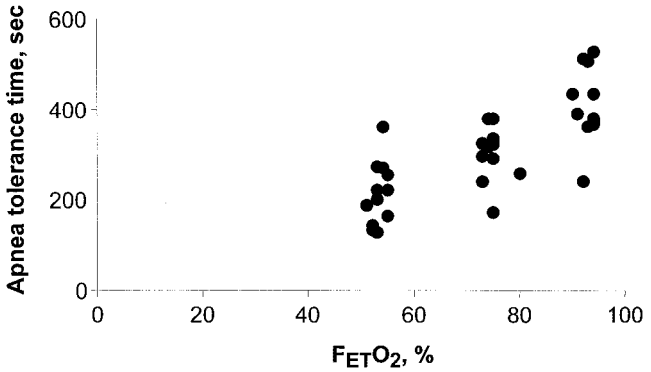

Fig. 3. Apnea tolerance time in individual patients (filled circles) in relation to their end-tidal oxygen concentrations (F_{ETO_2}) just before the apnea period. For definition of apnea tolerance, see text.

than 90%, was significantly longer when breathing 100% oxygen than 80 or 60% oxygen. In a patient with signs of a difficult airway or with any other reason to have maximal safety during induction, 100% oxygen is always preferable in order to have an extra minute or two for securing the airway. A "cannot ventilate, cannot intubate" situation may unpredictably occur during induction of anesthesia and supports the use of 100% oxygen at all times during preoxygenation. However, if morbidity or mortality from atelectasis formation can be proven, this has to be considered against the probability of an unpredictable "cannot ventilate, cannot intubate" situation. The risk–benefit ratio for the 60% group *versus* the 80% group seemed unfavorable. There was a small and probably clinically unimportant difference in atelectasis formation between the groups, while there was 1.5 min less time to decrease to 90% saturation.

Did the prolonged apnea *per se* increase the size of the atelectasis? The design of the study excluded the possibility of addressing this question, as we judged the information regarding apnea tolerance in the 80 and 60% oxygen groups to be of paramount interest. This limitation warrants a study without prolonged apnea to be properly investigated. For the time being, we can only compare with earlier studies of atelectasis formation during anesthesia with a short apnea for the intubation of the airway, in which the amount of atelectasis has been as large as in the present patients on 100% oxygen.[4,12,13]

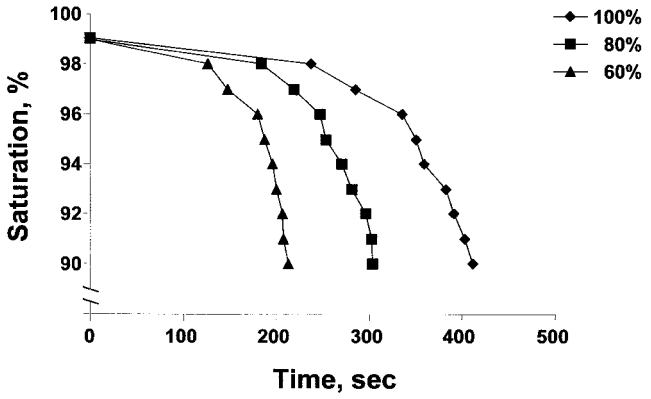

Fig. 4. The pattern of Spo_2 decrease during apnea in the groups ventilated with 100, 80, and 60% oxygen during induction, respectively. Means ± SDs at the end of apnea (90% saturation) were 411 ± 84, 303 ± 59, and 213 ± 69 s in the 100, 80, and 60% groups, respectively.

Table 3. Oxygen Saturation Data and Increase in End-tidal Carbon Dioxide Tension per Minute during Apnea in Patients Given 100%, 80%, or 60% Oxygen

	Fio_2 (%)		
	100	80	60
Lowest saturation (%)	88 ± 1	87 ± 2	88 ± 2
Time when saturation starts to increase (s)	27 ± 9	26 ± 5	32 ± 15
Time to 96% Spo_2 (s)	36 ± 9	37 ± 7	47 ± 23
ΔPCO_2 (mmHg/min)	1.5 ± 0.8	1.5 ± 0.8	2.3 ± 1.5

Data are presented as mean ± SD.

Fio_2 = fractional inspired oxygen tension; ΔPCO_2 = increase in end-tidal carbon dioxide tension; Spo_2 = oxygen saturation measured by pulse oximetry.

Another key question was whether the prolonged apnea increased the size of the atelectasis more so in the 100% oxygen group than in the others, since the apnea period was longer in this group. We tested the impact of this time difference by performing CT scans in a subgroup of patients breathing 80 or 60% oxygen at the same time point as we did in the 100% oxygen group. There was no difference in atelectasis in the 60 or 80% groups when comparing values directly after apnea (10.5 or 12 min after start of preoxygenation) with values at the time point when the 100% oxygen group was studied (14 min). It must be observed, however, that the patients in the 60 and 80% groups were ventilated with tidal volumes of 10 ml/kg some minutes longer before the new CT scans at 14 min, in contrast to the patients in the 100% group, who were ventilated only during the 1.5 min it took to complete the scans. It may be argued that this longer period of positive-pressure ventilation could have prevented or reversed atelectasis formation in these subgroups. However, it has been shown that reopening of collapsed lung tissue requires inflation of the lungs up to 40 cm H_2O, corresponding to a vital capacity maneuver.[14] It can also be proposed that complete airway closure with gas trapping and gas absorption is one likely mechanism of atelectasis during anesthesia, as hypothesized in clinical studies[15] and shown in mathematical modeling.[16] Whether the lungs are ventilated need not affect gas absorption, provided that airways remain closed over the respiratory cycle.

Application of PEEP can open up closed airways and may reduce the formation of atelectasis.[10] The level of PEEP in this study was only 3 cm H_2O. It might have reduced atelectasis formation, but equally so in the three groups since all groups had the same PEEP. PEEP is

effective only as long as it is applied, and during the period of apnea, there was apparently no PEEP.

The effect on postoperative atelectasis of using 80% oxygen during anesthesia was recently investigated by Akça *et al.*[17] In their study, 80% oxygen was compared with 30% given during colon resection and for 2 h after the procedure. No difference in incidence and severity of atelectasis was seen the first postoperative day. In the light of our results, the amount of atelectasis in the study by Akça *et al.*[17] may have originated from the use of 100% oxygen during induction of anesthesia. The use of 80% oxygen might be of borderline importance to produce atelectasis. If so, no difference might be expected between the 30 and 80% groups.

Although there were no statistical differences in the current study among patients regarding physiologic data, some details deserve a comment. In the 80% group, the mean hemoglobin value was 8–11 g higher per liter than in the other two groups. This might have enhanced the apnea tolerance in the 80% group. On the other hand, patients in the 100% group were 4 yr younger than in the 80% group, and the body mass index was slightly higher in the 80% group, as was the preoperative oxygen consumption awake at rest. These three factors may decrease apnea tolerance. However, this study was too small to discriminate between the significance of factors other than oxygen level in influencing the formation of atelectasis during anesthesia induction. Clearly, those other factors vary in impact in different patients, as illustrated by the large variation in atelectasis formation in the 100% group.

We may conclude that preoxygenation with 80% oxygen seemed to be beneficial in reducing atelectasis formation during anesthesia induction, compared with 100% oxygen, but with a reduction in apnea tolerance. This reduction in apnea tolerance can make a difference in unpredictable, complicated situations during anesthesia induction. Until a large clinical trial can prove significant morbidity from atelectasis during or after anesthesia, the standard of using 100% oxygen for preoxygenation should continue.

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