

Anesthesiology
69:980-982, 1988

Pharyngeal Insufflation of Oxygen Prevents Arterial Desaturation during Apnea

LYNN E. TELLER, M.D.,* CHRISTIAN M. ALEXANDER, M.D.,*
M. JACK FRUMIN, M.D.,† JEFFREY B. GROSS, M.D.‡

A significant problem during induction of anesthesia is the need to maintain adequate arterial oxygenation in apneic patients until airway control is obtained. Anesthesiologists frequently administer oxygen before inducing anesthesia; this "preoxygenation" fills the functional residual capacity (FRC) of the lungs with oxygen, increasing patients' oxygen reserves.¹ Various preoxygenation techniques have been advocated.²⁻⁴ For instance, 3 min of oxygen breathing significantly delays the onset of hypoxemia in healthy, apneic patients, although some of these individuals became hypoxemic after less than 7 min.⁴ Apnea or hypoventilation of this duration may occur when ventilation by mask is difficult (*e.g.*, poor mask fit) or contraindicated (*e.g.*, "full stomach"), and endotracheal intubation requires multiple attempts or is unsuccessful. The risk of hypoxia is compounded when the ratio of oxygen consumption to FRC is increased (*e.g.*, infancy, pregnancy), because oxygen reserves are exhausted more quickly.

In a typical apneic adult, approximately 250 ml/min of oxygen are transferred from the lungs into the bloodstream, while only 10-20 ml/min of CO₂ enter the lungs from the bloodstream;⁵ the remainder of the body's CO₂ production is buffered by red blood cells or dissolved in tissues. This alveolar gas deficit causes alveolar pressure to become slightly subatmospheric; if the airway is patent, there will be a net flow of gas from the pharynx into the alveoli.⁶ We reasoned that, if we kept the pharynx filled with oxygen, the onset of hypoxia would be delayed, since oxygen, rather than air, would be drawn into the lungs by this mechanism. We undertook this randomized, double-blind, crossover study to determine whether insuffla-

tion of oxygen into the pharynx of paralyzed, "preoxygenated" patients could prolong the period during which they remain adequately oxygenated.

MATERIALS AND METHODS

Twelve ASA physical status 1 or 2 patients without symptomatic pulmonary disease scheduled for surgery under general anesthesia consented in writing to participate in this study, which was approved by our Institutional Review Boards. Each patient received an iv infusion and routine monitoring including an Ohmeda 3700[®] pulse oximeter with ear probe, set to the fast-response mode (3-s averaging). A PPG SARA[®] mass spectrometer set to the "neuro" mode monitored inspired and end-tidal gas concentrations at approximately 30-s intervals. Patients breathed oxygen from the circle system (6 l/min inflow) for 3 min. We instructed them to hyperventilate until their end-tidal CO₂ tensions were approximately 25 mmHg; this ensured that after 10 min of apnea, arterial CO₂ tensions would not exceed 80 mmHg.⁷

After induction of anesthesia with thiopental 5 mg/kg, midazolam 2 mg, and fentanyl 100 µg, and paralysis with succinylcholine 100 mg iv, we inserted a 36 Fr Rusch nasal airway. To ensure that subjects' lungs could be ventilated in an emergency, we administered a single breath of oxygen before starting to time the duration of apnea. We then removed the facemask and maintained the subject's head in neutral position with gentle jaw support. After inserting an 8 Fr catheter 2 cm beyond the tip of the nasal airway, we insufflated either oxygen (3 l/min) or placebo (no insufflation) through the catheter in a randomized manner. The oxygen delivery tubing passed over the patient's left shoulder to an independent flowmeter out of view of the investigators, who were unaware of whether oxygen or placebo was being administered during any given trial. Succinylcholine infusion maintained paralysis, as demonstrated by the absence of response to an ulnar nerve stimulator, while supplemental doses of thiopental, midazolam, and fentanyl maintained an adequate level of anesthesia. Apnea continued until either: 1) SaO₂ dropped to 92%, or 2) 10 min had elapsed. At this time, we recorded the "duration of apnea," removed the oxygen cannula, and ventilated patients' lungs by mask with oxygen. We also recorded the minimum SaO₂ for each apneic trial; this often occurred a few seconds after the

* Assistant Professor of Anesthesia, University of Pennsylvania School of Medicine.

† Lecturer in Anesthesia, Stanford University School of Medicine.

‡ Associate Professor of Anesthesia, University of Pennsylvania School of Medicine.

Received from the Departments of Anesthesia, University of Pennsylvania and Philadelphia Veterans Administration Medical Center, Philadelphia, Pennsylvania. Accepted for publication July 18, 1988. Presented in part at the Annual Meeting of the American Society of Anesthesiologists, San Francisco, California, October, 1988.

Address reprint requests to Dr. Gross: Department of Anesthesia (112), Philadelphia VA Medical Center, University and Woodland Avenues, Philadelphia, Pennsylvania 19104.

Key words: Anesthetic techniques; induction. Apnea. Oxygen. Oxygenation; apneic.

resumption of ventilation because of the time delay in pulse oximeter measurements.⁸

After completing the first arm of the crossover study as described above, we began the second phase by manually hyperventilating the patients' lungs with oxygen and isoflurane for 3 min; the resulting SaO₂ and end-tidal respiratory gas concentrations were approximately the same as those which prevailed before the first apneic trial (see below). The isoflurane reduced the requirement for intravenous anesthetic agents during the second trial. We then repeated the apneic study sequence, insufflating placebo (*i.e.*, no insufflation) or oxygen (3 l/min), whichever was not given during the first trial, through the nasal catheter. Criteria for ending apnea were the same as those used during the first trial. After recording the "duration of apnea" and the minimum observed SaO₂, we manually ventilated the patients' lungs and proceeded with their anesthetics.

Paired *t* tests compared each patient's duration of apnea and minimum SaO₂ during oxygen insufflation with those observed in the absence of oxygen insufflation. To independently assess the effect of oxygen insufflation during the first and second trials, we used unpaired *t* tests with a Bonferroni correction. Two-way analysis of variance determined if the method of preoxygenation (spontaneous ventilation with oxygen *versus* controlled ventilation with oxygen/isoflurane) significantly affected any of the variables. *P* < 0.05 indicated significance.

RESULTS

Patients were between 35 and 65 yr of age (52 ± 4, \bar{x} ± SE) and had significant histories of tobacco use (20 ± 6 pack-years). SaO₂ while breathing room air was 97 ± 1%. During pharyngeal oxygen insufflation, SaO₂ never fell below 97% during the entire 10 min of apnea in any subject; the mean minimum saturation achieved was 98 ± 1%. Conversely, in the absence of oxygen insufflation, the duration of apnea was 6.8 ± 0.6 min (*P* < 0.001), and the minimum saturation observed was 91 ± 1% (*P* < 0.001). These results, as summarized in table 1, were independent of whether preoxygenation was by spontaneous or controlled ventilation.

Prior to apnea, SaO₂, F_{ET} O₂, and P_{ET} CO₂ did not differ appreciably between oxygen and placebo (*i.e.*, no oxygen insufflation) trials; these variables were also similar between the first (*i.e.*, spontaneous ventilation) and second (*i.e.*, controlled ventilation) trials (table 1). All patients' vital signs remained within 25% of their baseline values during the apneic periods, and no patient had any sequelae from participation in the study.

DISCUSSION

Pharyngeal insufflation of oxygen to delay the onset of hypoxemia in preoxygenated patients has numerous

TABLE 1. Duration of Apnea (*i.e.*, Time from Cessation of Ventilation Until Either (1) SaO₂ fell to 92%, or (2) 10 Min had Elapsed) and Minimum Observed SaO₂ With and Without Pharyngeal Oxygen Insufflation. Values are Means ± SE

	O ₂ Insufflation	No O ₂ Insufflation
First trial		
Duration of apnea (min)	10.0 ± 0	7.1 ± 0.6*
Minimum SaO ₂ (%)	98 ± 1	92 ± 1*
Pre-apnea SaO ₂ (%)	99 ± 1	99 ± 1
Pre-apnea F _{ET} O ₂ (%)	87 ± 1	88 ± 2
Pre-apnea P _{ET} CO ₂ (mmHg)	26 ± 2	22 ± 2
N	6	6
Second trial		
Duration of apnea (min)	10.0 ± 0	6.6 ± 0.9*
Minimum SaO ₂ (%)	99 ± 1	91 ± 1*
Pre-apnea SaO ₂ (%)	99 ± 1	99 ± 1
Pre-apnea F _{ET} O ₂ (%)	90 ± 1	92 ± 1
Pre-apnea P _{ET} CO ₂ (mmHg)	27 ± 1	28 ± 2
N	6	6
Combined		
Duration of apnea (min)	10.0 ± 0	6.8 ± 0.6†
Minimum SaO ₂ (%)	98 ± 1	91 ± 1†
Pre-apnea SaO ₂ (%)	99 ± 1	99 ± 1
Pre-apnea F _{ET} O ₂ (%)	88 ± 1	90 ± 1
Pre-apnea P _{ET} CO ₂ (mmHg)	27 ± 1	25 ± 1
N	12	12

* *P* < 0.01 compared with oxygen insufflation (same trial).
† *P* < 0.001 compared with oxygen insufflation.

practical applications. For instance, despite the presence of a patent airway, mask ventilation may be difficult in patients who are edentulous or who have craniofacial abnormalities or trauma; pharyngeal oxygen insufflation may allow additional time for laryngoscopy should intubation prove difficult. Also, with this technique, otolaryngologists can safely be provided with a full 10 min to visualize airway structures, unimpeded by the presence of an endotracheal tube or by patients' respiratory movements. Similarly, when laryngoscopy is taught, pharyngeal insufflation of oxygen allows the student additional time to visualize the larynx and intubate the trachea before ventilation *via* mask must be resumed. Finally, this technique may provide crucial extra minutes of safe laryngoscopy time during the rapid-sequence induction of anesthesia in patients with "full stomachs," in whom mask ventilation is contraindicated.

Previous descriptions of pharyngeal oxygen insufflation through catheters or modified laryngoscopes ("oxyscopes") have dealt with patients who are breathing spontaneously.^{9,10} The present study is the first to document that insufflation of oxygen into the pharynx of apneic, paralyzed patients significantly delays arterial desaturation. To minimize any potential risk to subjects, we studied only ASA physical status 1 and 2 patients; therefore, the quantitative data of table 1 are only applicable to such

patients. However, the fact that oxygen insufflation always delayed the development of hypoxemia suggests that this qualitative finding may be applicable to most preoxygenated patients whose airways are open between the pharynx and alveoli. This was dramatically illustrated during induction of anesthesia in a 150-kg patient who was not a subject in the study: Despite preoxygenation and adequate ventilation *via* mask with oxygen, numerous attempts at endotracheal intubation were aborted because hypoxia developed rapidly during laryngoscopy. Insufflation of oxygen into his pharynx after ventilation *via* mask provided us with the crucial extra minutes we needed to intubate his trachea.

We found that, in the absence of oxygen insufflation, SaO_2 remained above 92% for only 6.8 ± 0.6 min ($\bar{x} \pm \text{SE}$); this is slightly shorter than the 8.6 ± 1.0 min found by Gambee *et al.* in PS I patients ranging in age from 27–34 yr.⁴ Two factors may account for this difference. First, our patients had significant smoking histories and were older than Gambee's patients, who did not smoke. Second, we placed the pulse oximeter on the ear rather than the finger. During changes in oxygenation, readings from pulse oximeter probes on the finger are delayed by about 30 s when compared with readings from pulse oximeter probes on the earlobe or with radial arterial blood samples analyzed by co-oximetry.⁸ Therefore, the finger probes used by Gambee *et al.* did not detect arterial desaturation as quickly as did our ear probes.

For reasons of safety, we resumed ventilation when SaO_2 fell to 92%, regardless of whether or not 10 min had elapsed. Undoubtedly, in the absence of oxygen insufflation, a greater decrease in SaO_2 would have occurred had apnea continued for the full 10 min. Despite this, minimum oxygen saturations in the absence of oxygen insufflation were significantly less than those observed during oxygen insufflation. Because we resumed ventilation after 10 min, the present data are insufficient to predict the maximum safe duration of apnea using our technique. However, Frumin *et al.* found that, when oxygen was connected, apneic patients whose tracheas were intubated remained well oxygenated for as long as 55 min.⁶

Although we attempted to provide identical conditions prior to the first and second apneic trials, there were several unavoidable differences. For instance, it was necessary to use controlled ventilation to "preoxygenate" patients prior to the second apneic trial, because they were already fully paralyzed; however, end-tidal gas tensions did not differ significantly between the first and second trials. The single positive-pressure breath prior to the first apneic

trial did not prolong the recorded duration of apnea, because we did not start the timer until after the breath was given. Finally, we added low concentrations of isoflurane prior to the second apneic trial to decrease the need for intravenous agents.¹¹ Despite these differences, our results indicate that the duration of apnea and minimum observed oxygen saturation depended only upon the insufflation of oxygen, and that the method of ventilation during "preoxygenation" did not significantly affect the results.

In conclusion, preoxygenation followed by insufflation of oxygen *via* nasopharyngeal cannula provides at least 10 min of adequate oxygenation in healthy apneic patients whose airways are unobstructed and in whom the trachea is not intubated. Unlike "insufflating laryngoscopes," no special equipment is required; oxygen is provided without interruption during airway manipulations, thus reducing the likelihood that room air will enter the pharynx and lungs. By significantly delaying the onset of hypoxia, this technique may be beneficial in situations when extra minutes are needed to gain control of the airway.

REFERENCES

1. Dillon JB, Darsi ML: Oxygen for acute respiratory depression due to the administration of thiopental sodium. *JAMA* 159:1114–1116, 1955
2. Drummond GB, Park GR: Arterial oxygen saturation before intubation of the trachea. *Br J Anaesth* 56:987–993, 1984
3. Gold MI, Muravchick S: Arterial oxygenation during laryngoscopy and intubation. *Anesth Analg* 60:316–318, 1981
4. Gambee AM, Hertzka RE, Fisher DM: Preoxygenation techniques: Comparison of three minutes and four breaths. *Anesth Analg* 66:468–470, 1987
5. Severinghaus JW, Eger EI: The rate of rise of PA_{CO_2} in the apneic anesthetized patient. *ANESTHESIOLOGY* 22:419–425, 1961
6. Frumin MJ, Epstein RM, Cohen G: Apneic oxygenation in man. *ANESTHESIOLOGY* 20:789–798, 1959
7. Stock MC, Schisler JQ, McSwenney TD: The CO_2 rate of rise during apnea in anesthetized humans with airway obstruction. *Anesth Analg* 67:S222, 1988
8. Kagle DM, Alexander CM, Berko RS, Guiffre M, Gross JB: Evaluation of the Ohmeda 3700 pulse oximeter: Steady-state and transient response characteristics. *ANESTHESIOLOGY* 66:376–380, 1987
9. Wung JT, Stark RI, Indyk L, Driscoll JM: Oxygen supplementation during endotracheal intubation of the infant. *Pediatrics* 59:1046–1047, 1977
10. Todres ID, Crone RK: Experience with a modified laryngoscope in sick infants. *Crit Care Med* 9:544–545, 1981
11. Benumof JL, Augustine SD, Gibbons JA: Halothane and isoflurane only slightly impair arterial oxygenation during one lung ventilation in patients undergoing thoracotomy. *ANESTHESIOLOGY* 67:910–915, 1987