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Anesthesia for Thoracoscopic Laser Ablation of Bullous Emphysema

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Background: We describe the anesthetic management for a new surgical procedure: laser ablation of emphysematous bullae *via* thoracoscope. Although thoracoscopy is not new, this is the first description of a series of patients with bilateral, chronic lung disease who underwent long periods of one-lung ventilation (OLV) during thoracoscopic therapy.

Methods: Twenty-six laser ablation procedures were performed in 22 patients. The patients were elderly (mean age 63 yr) with a large incidence of coexisting cardiovascular disease. Most required chronic home oxygen therapy. Patients were monitored invasively, and hemodynamic data were recorded every 5 min. Arterial blood gas analyses were performed every 15 min. Comparisons were made between three intraoperative periods: two-lung ventilation (TLV) before thoracoscopy, OLV during thoracoscopy, and TLV after thoracoscopy.

Results: All patients survived the operation despite a mean OLV duration of 170 min, but several experienced serious intraoperative problems, such as hypoxemia or hypotension. Hypoxemia was treated with nondependent lung continuous positive airway pressure and dependent lung positive end-expiratory pressure. In all patients the lungs were adequately ventilated, but bronchopleural fistulae occurred upon return to TLV in every case. The resulting air leaks, often 50% of inspired tidal volume, required the use of a pressure-cycled ventilator to maintain oxygenation. Postoperative air leaks greater than 50% of inspired tidal volume usually required subsequent surgical correction, while smaller leaks resolved spontaneously. Mechanical ventilation was required for an average of 5 days. Eighty-four percent have survived at least 6 months, and nearly all survivors report symptomatic improvement.

Conclusions: Ablation of bullae appears to provide symptomatic improvement, and thoracoscopy might be better tol-

erated than thoracotomy, especially in patients with severe bullous emphysema. (Key words: Emphysema. One-lung ventilation. Laser. Thoracic anesthesia. Thoracoscopy.)

THORACOSCOPY was originally described in 1915 by Jacobaeus¹ and was widely used prior to the introduction of anti-tuberculous chemotherapy. The procedure was traditionally carried out under local anesthesia, in part because of concern over poor pulmonary function. In recent years there has been renewed interest in the technique, as both a diagnostic and a therapeutic tool.^{2,3} Several authors have reported on the success of thoracoscopic ablation of lung bullae in patients with spontaneous pneumothorax, initially using electrocautery^{3,4} and more recently using the Nd:YAG and CO₂ lasers.^{5,6} These therapeutic thoracoscopies are of much longer duration than the diagnostic procedures. Moreover, the procedure is facilitated greatly by collapse of the operative lung, and previous authors have drawn attention to the benefit of one-lung ventilation (OLV) during thoracoscopy.^{2,3} In patients with spontaneous pneumothorax, OLV usually does not pose a major challenge as the underlying lung function is good.

We report the results of a prospective anesthetic case study of thoracoscopic laser ablation of bullae in patients with bilateral bullous emphysema. The patients selected for this procedure had such profound pulmonary impairment that they were not considered candidates for open thoracotomy. They are of anesthetic interest because their underlying lung disease is severe, and the thoracoscopic procedure requires a prolonged period of OLV. Hypoxia has long been recognized as a risk of OLV and has been shown to occur more frequently in patients not undergoing pulmonary resection.⁷ This report summarizes the anesthetic course and management of the first 26 procedures at our institution. The surgical and postoperative course of a similar group of patients undergoing this operation has been described by Wakabayashi *et al.*⁸

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Materials and Methods

University human subjects review board approval was obtained for the study. Twenty-two patients participated and underwent a total of 26 procedures. Four patients had operations on both lungs, 3–6 weeks apart. All patients were informed of the experimental nature of the operation and of the possibility of open thoracotomy in the event of complications. Preoperative assessment included forced vital capacity, forced expiratory volume in one second (FEV_1), and arterial blood gas analysis and testing of exercise tolerance when possible. An exercise tolerance of more than 5 min walking was considered a relative contraindication to the procedure, because such patients were not severely compromised enough to benefit from bullectomy. Allen's test for collateral circulation was performed on all patients. No premedication was given, as most of these patients experienced dyspnea at rest.

A radial arterial cannula was inserted preoperatively, and baseline arterial blood gases were measured. In nine instances, these baseline values were determined while the patients breathed supplemental oxygen. Standard monitors included a five-lead electrocardiograph with continuous ST-segment analysis (Marquette series 7000, Milwaukee, WI), pulse oximeter, capnograph, and transcutaneous oxygen electrode (Novamatrix 811, Wallingford, CT). In 22 procedures, a pulmonary artery (PA) catheter was inserted *via* the right internal jugular vein. Ten of these were mixed-venous oximetry catheters (Opticath, Abbott, Chicago, IL). Induction of anesthesia employed 20–30 $\mu\text{g}/\text{kg}$ iv fentanyl and 0.1 mg/kg vecuronium. Anesthesia was maintained using halothane or isoflurane in 100% oxygen and additional fentanyl as required. When adequate relaxation was achieved, a left-sided double-lumen endotracheal tube (ETT; Bronchocath, Mallinckrodt, St. Louis, MO) was inserted *via* direct laryngoscopy. Proper position of the tube was confirmed using a fiberoptic bronchoscope inserted through the tracheal lumen, both before and after turning the patient to the lateral decubitus. Muscle relaxation was maintained with iv vecuronium, and continuous positive pressure ventilation was used throughout the procedure. The lungs of the first five patients were ventilated using the Dräger AV ventilator on a Narcomed 2B Anesthesia Machine. This proved inadequate during the latter part of the procedure, when large air leaks (more than 50% of inspired volume) were common. The lungs of subsequent patients were ventilated using the Sie-

mens Servo 900C ventilator (Siemens Life Support Systems, Solna, Sweden), which provides pressure-controlled ventilation with high inspiratory flows. Pressure-controlled ventilation provides nearly constant distension of the lungs even in the presence of varying airway leaks. An inspired oxygen fraction ($F_{I_{O_2}}$) of 100% was used throughout the procedure.

The following data were recorded at 5-min intervals: heart rate, systemic and pulmonary arterial blood pressure, pulse oximeter arterial hemoglobin saturation, mixed venous oxygen saturation, inspiratory and expiratory tidal volumes, respiratory rate, and end-tidal carbon dioxide. Arterial and mixed venous blood gases were determined from anaerobically collected specimens at 15–20-min intervals. Blood gas analysis was performed by the Nova-Stat 3 Analyzer (Nova, Waltham, MA) and oxygen saturation was measured *in vitro* by the OSM-3 Co-oximeter (Radiometer, Copenhagen). Cardiac output was determined every 10–15 min by thermodilution using the mean of three satisfactory curves. Alveolar oxygen tension was determined at the time of each blood-gas analysis using the alveolar gas equation.

The surgical procedure employed two to four small intercostal thoracoscopy incisions. The nondependent, operative lung was collapsed prior to insertion of the thoracoscope. The surgeons used a CO_2 laser *via* fiberoptics through the thoracoscope to shrink and cauterize the lung bullae. Hypoxemia occurring during OLV, defined here as any Pa_{O_2} value less than 70 mmHg, was treated initially by the application of 5 cmH_2O continuous positive airway pressure (CPAP) to the nondependent lung. If the hypoxemia persisted, a positive end-expiratory pressure (PEEP) of 5 cmH_2O was applied to the dependent lung. Continued hypoxemia during OLV was treated by an alternating PEEP/CPAP search using 5- cmH_2O increments, as recommended by Benumof.⁹ Rarely, brief periods of two-lung ventilation (TLV) were necessary to restore adequate oxygenation. Malpositioning of the ETT was excluded in these instances by direct observation using the fiberoptic bronchoscope.

Following laser ablation of the bullae, the operative lung was suctioned and re-expanded. A large air leak, often more than 50% of inspired tidal volume, was universal at this stage. In two patients in whom this leak could not be controlled by further laser cautery, a mini-thoracotomy was performed. All patients had underwater seal chest drains inserted. At the end of the surgical procedure, the double-lumen ETT was replaced

by a conventional single-lumen ETT and the patients were transferred to the recovery room.

Data were analyzed digitally using StatView 512+ (Brainpower, Calabasas, CA) on a Macintosh II CX computer. Mean and standard deviation values were determined for each of the measured variables during TLV before thoracoscopy, OLV during thoracoscopy, and TLV after thoracoscopy. Differences were compared for significance using the unpaired Student's *t* test with Bonferroni correction. A *P* value less than .05 was considered significant.

Results

Intraoperative Course

Twenty-two patients underwent 26 laser ablation procedures. The majority of the patients were elderly, the mean \pm SD age being 63.0 ± 7.4 yr. They had a high incidence of coexisting cardiovascular disease, and all patients were ASA physical status 3 or 4. Several patients had evidence of ischemic heart disease, which was evaluated preoperatively by an exercise test or dobutamine stress test. The underlying lung disease was severe: 13 of the 22 patients chronically breathed supplemental oxygen. Six were wheelchair-bound, and among the remainder the mean exercise tolerance was less than 200 yards' walking. In two patients, previous episodes of respiratory failure required mechanical ventilation; a further two patients had undergone tracheostomies for sputum clearance. One patient pre-

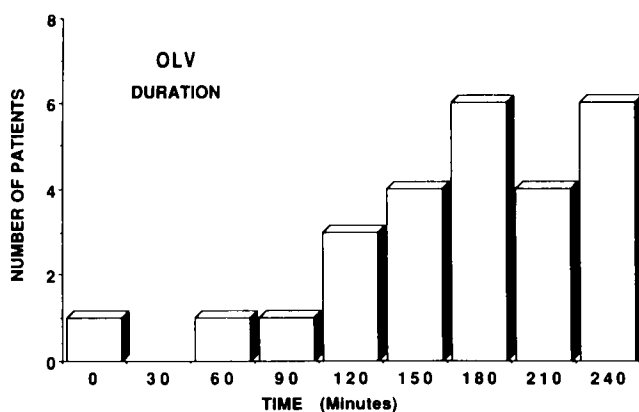


Fig. 1. Histogram showing the duration of one-lung ventilation (OLV) for 26 thoracoscopy procedures in 22 patients. For example, six procedures required OLV for 180 ± 15 min. An additional six procedures required 4 h of OLV.

Table 1. Cardiopulmonary Variables during Three Stages of Laser Thoracoscopy Procedures

	TLV _{pre}	OLV	TLV _{post}
PaO ₂ (mmHg)	392 \pm 123 (202-571)	190 \pm 99* (43-421)	366 \pm 122 (175-551)
PaO ₂ /PAO ₂ (mmHg)	0.64 \pm 0.18 (0.33-0.93)	0.33 \pm 0.18* (0.09-0.83)	0.58 \pm 0.20 (0.27-0.83)
PaCO ₂ (mmHg)	39.3 \pm 3.2 (33.4-43.8)	41.8 \pm 8.9 (21.8-60.4)	32.9 \pm 7.2† (24.6-51.8)
PETCO ₂ (mmHg)	33.7 \pm 7.1 (24-61)	34.1 \pm 7.5 (12-58)	31.9 \pm 5.2 (18-43)
CI	2.3 \pm 0.7 (1.2-3.4)	3.0 \pm 1.4 (1.0-6.6)	2.5 \pm 0.5 (1.7-3.2)
SvO ₂ (%)	82 \pm 6 (67-91)	75 \pm 12† (44-94)	70 \pm 11* (54-94)

TLV_{pre} = two-lung ventilation after anesthetic induction and intubation but before insertion of thoracoscope and deflation of operative lung; OLV = one-lung ventilation 10 min or longer after lung collapse; TLV_{post} = two-lung ventilation after reinflation of operative lung after completion of laser ablation.

* *P* < .01 versus TLV_{pre} value.

† *P* < .05 versus TLV_{pre} value.

sented with a recurrent pneumothorax following thoracoscopy in another institution.

The mean preoperative FEV₁ of all 22 patients was 0.75 ± 0.44 L; the mean vital capacity was 1.88 ± 0.74 L; and the mean PaO₂ was 67.6 ± 8.3 mmHg (range 52-79 mmHg). Nine patients were receiving supplemental oxygen at the time of blood-gas assessment, the FiO₂ ranging from 0.24 to 0.5. The mean preoperative ratio of arterial to alveolar oxygen tension (PaO₂/PAO₂) was 0.56 ± 0.12 (normal range 0.8-0.9). Although five patients had increased preoperative PaCO₂ values, the mean was only 45 ± 10 mmHg (range 34-75 mmHg).

The laser thoroscopic procedure required an unusually long period of OLV, as shown in figure 1. The mean OLV duration was 170 ± 53 min, and six patients required roughly 4 h of OLV. Table 1 shows intraoperative values (mean \pm SD and range) of several cardiopulmonary variables. The first column (TLV_{pre}) shows values measured after anesthetic induction and intubation, before insertion of the thoracoscope and deflation of the operative lung. The second column (OLV) represents an average of data measured during OLV, 10 min or more after operative lung deflation. The third column (TLV_{post}) shows average data measured following the resumption of TLV after thoracoscopy. Figure 2 is a scattergram plot of the mean PaO₂ during OLV for each patient *versus* the corresponding

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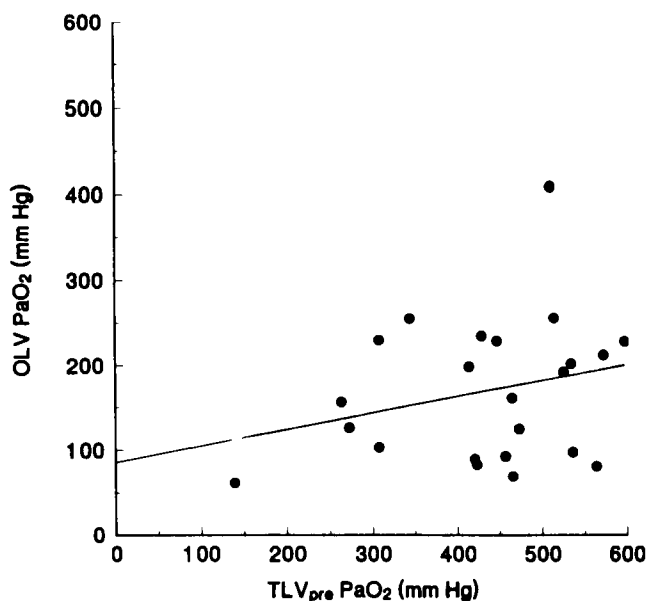


Fig. 2. Scattergram plot of the mean Pa_{O_2} value during OLV for each patient versus the corresponding $\text{TLV}_{\text{pre}} \text{Pa}_{\text{O}_2}$ value. Linear regression: $y = 0.19x + 86.1$. Correlation coefficient: $r = 0.26$. The value of Pa_{O_2} during the TLV_{pre} period is poorly predictive of the Pa_{O_2} during OLV.

$\text{TLV}_{\text{pre}} \text{Pa}_{\text{O}_2}$ value. The Pa_{O_2} (or $\text{Pa}_{\text{O}_2}/\text{PA}_{\text{O}_2}$) values during the TLV_{pre} period were poorly predictive of the values during the OLV period. The correlation coefficient between Pa_{O_2} values for the two periods was $r = 0.26$. The linear regression (fig. 2) has a slope of 0.19 ± 0.15 and a y-intercept of 86 mmHg. The standard error of the y-estimate is 83 mmHg. The preoperative Pa_{O_2} value also was not predictive of Pa_{O_2} during OLV: $r = 0.11$. There was no significant relationship between preoperative use of supplemental oxygen and intraoperative hypoxemia.

Moderate hypoxemia ($\text{Pa}_{\text{O}_2} < 70$ mmHg) required the use of nondependent lung CPAP in seven cases (27% of total). Six of these seven patients also required dependent lung PEEP (5 cmH_2O) to maintain Pa_{O_2} values greater than 70 mmHg. Hypoxemia was more likely to occur as the duration of OLV increased. Dividing the OLV period for each patient into three equal segments, the mean Pa_{O_2} during the first segment was 263 ± 103 mmHg, while during the last segment it was 161 ± 87 mmHg ($P < .01$). In four patients the mean Pa_{O_2} values were less than 75 mmHg during the last segment of OLV, yet they were greater than 200 mmHg during the first segment. Eighty percent of all Pa_{O_2} values less than 75 mmHg occurred during the last one-

third of OLV. One patient did not tolerate even short periods of OLV, exhibiting both hemoglobin desaturation and simultaneous hypotension on each attempt to isolate the nondependent lung. Incorrect placement of the double-lumen ETT was excluded by fiberoptic bronchoscopy and the procedure was completed, with difficulty, using a single-lumen tube. This patient also required perioperative inotropic support (dopamine, 10 $\mu\text{g}/\text{kg}/\text{min}$ iv infusion).

Carbon dioxide elimination was not a serious problem during OLV, as shown in Table 1. The mean OLV Pa_{CO_2} was 41.8 ± 8.9 mmHg, compared with a mean $\text{TLV}_{\text{pre}} \text{Pa}_{\text{CO}_2}$ of 39.3 ± 3.2 mmHg. One patient, who had chronic hypercarbia, experienced a mildly increased Pa_{CO_2} throughout the TLV_{pre} period (41–52 mmHg) and the subsequent OLV (50–54 mmHg). This patient received 3 mg midazolam intravenously during insertion of monitoring catheters, and his Pa_{CO_2} immediately before induction of anesthesia was 85 mmHg. The mean Pa_{CO_2} for all patients decreased during the TLV_{post} stage of the procedure (table 1), whereas end-tidal carbon dioxide was almost unchanged.

Table 1 also shows cardiac index and Sv_{O_2} data obtained from the PA catheter. No statistically significant change in cardiac index occurred during OLV. The gradual decrease in Sv_{O_2} from 82 (TLV_{pre}) to 75 (OLV) to 70 (TLV_{post}) was significant ($P < .01$). Since neither cardiac output nor arterial oxygen content changed significantly during the procedure, this decrease in Sv_{O_2} is the result of an increase in oxygen consumption. Although PA pressures did not change significantly during the three stages of the procedure, they were consistently moderately elevated. The mean intraoperative PA diastolic pressure was 22 ± 6.0 mmHg, and the mean PA wedge pressure was 19 ± 7.6 mmHg.

Postoperative Course

Pressure-controlled ventilation was continued in the recovery room, and chest tubes were connected to water seal only (no suction). Chest tube suction and PEEP were avoided during the early postoperative period, because they greatly increase flow through bronchopleural fistulae. In the first four patients, rapid separation from mechanical ventilation and early (same day) extubation of the trachea were attempted. This led to a high incidence of reintubation, with one patient (a nonsurvivor) being reintubated four times. Subsequent patients were allowed a longer period of assisted ventilation. $\text{Pa}_{\text{O}_2}/\text{PA}_{\text{O}_2}$ values often worsened during the

first 1–3 postoperative days, while chest x-rays demonstrated a bilateral congested pattern. The mean $\text{Pa}_{\text{O}_2}/\text{PA}_{\text{O}_2}$ ratio decreased from 0.57 ± 0.44 on postoperative day zero to 0.27 ± 0.10 on postoperative day 3 ($P < .01$). However, no patients experienced hypoxemia ($\text{Pa}_{\text{O}_2} < 70$ mmHg) during this period.

The mean duration of postoperative mechanical ventilation was 5.3 ± 5.4 days; the longest period was 45 days in a patient whose course was complicated by a pulmonary abscess, sepsis, and myocardial ischemia. Two patients required urgent reoperation for large bronchopleural air leak within the first 48 h. Another two patients underwent mini-thoracotomy during the initial surgery for the same reason. One patient died of myocardial infarction after 13 days of assisted ventilation. Other postoperative deaths resulted from renal failure, pneumonia, and stroke. The survival rate in the present study group is 84% 6 months after surgery.

Nearly all survivors report symptomatic improvement in the form of better exercise tolerance and a reduction in dyspnea. Most exhibit improvements in postoperative pulmonary function tests. Pulmonary function data obtained more than 1 month postoperatively are available for 11 of the 22 patients. For these 11 patients, the mean vital capacity improved from 2.13 ± 0.79 l preoperatively to 2.66 ± 0.68 l postoperatively ($P < .05$). The mean FEV_1 for this group improved from 0.83 ± 0.53 l to 1.05 ± 0.67 l. The mean percentage improvement in pulmonary function, defined as $\text{mean}[100 \times (\text{preoperative value} - \text{postoperative value})/\text{preoperative value}]$, was 32% for vital capacity and 30% for FEV_1 ($P < .01$).

Discussion

This group of patients is of particular interest to anesthesiologists because they have severe bilateral lung disease with chronic hypoxemia and are undergoing a procedure requiring prolonged periods of OLV (average 170 min). The dependent ventilated lung is often as seriously diseased as the nonventilated operative lung. Several patients (four in the study group) subsequently have undergone laser ablation of bullae in the second lung. Although these patients were not considered good candidates for open thoracotomy,^{7,8} the duration of OLV in our series is much longer than that normally required for lobectomy or pneumonectomy. Nevertheless, these patients tolerated OLV better than

we had expected, albeit with a significant incidence of hypoxemia (27% of patients having $\text{Pa}_{\text{O}_2} < 70$ mmHg). The Pa_{O_2} values during the TLV_{pre} period were poorly predictive of Pa_{O_2} during OLV: $r = 0.26$. Furthermore, neither the preoperative Pa_{O_2} value nor the use of supplemental oxygen were predictive of Pa_{O_2} during OLV. The explanation for this may lie in the variable relationship between the function of the two lungs in these patients. All candidates for this procedure therefore should be considered at risk for intraoperative hypoxemia.

Ventilation, *i.e.*, the removal of carbon dioxide, was not a serious problem during these procedures. In fact, the decrease in the arterial to end-tidal carbon dioxide tension gradient that occurred at the end of the operation (table 1) suggests a decrease in alveolar dead space ventilation. This reduction in dead space could result from the elimination of emphysematous lung bullae during the procedure.

Malposition of the double-lumen ETT is a common cause of hypoxemia during OLV, and confirmation of correct tube position using the fiberoptic bronchoscope is essential in this population. Turning the patient to the lateral decubitus or manipulation of the operative lung with the thoracoscope may cause the ETT to move. The ETT position therefore should be reconfirmed by bronchoscopy following any unexpected decrease in Pa_{O_2} . Episodes of hypoxemia not corrected by ETT repositioning were treated using a CPAP/PEEP search protocol as suggested by Benumof.⁹ Continuous positive airway pressure applied to the nondependent lung usually was limited to 5 cmH₂O to prevent distention and possible interference with the surgery. We subsequently found that by allowing the lung to distend slightly near the end of the procedure we could assist the surgeon in the identification of small air leaks.

Hypoxemia occurred more often as the duration of OLV increased, with most of the Pa_{O_2} values below 75 mmHg occurring during the last one-third of OLV. This may have been due in part to the development of absorption atelectasis in the dependent lung from the use of high Fi_{O_2} values. High inspired oxygen concentrations can lead to arteriovenous shunting in areas of airway closure and further cause "absent expired ventilation" in lung units with low ventilation-perfusion ratios.¹⁰ Another possible mechanism of hypoxemia in these cases is progressive fluid transudation in the dependent lung, again exaggerated by the prolonged surgery and the relatively large volumes of crystalloid

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transfused.¹¹ A previous clinical study showed the calculated transpulmonary shunt to increase with duration of OLV.⁷ The authors postulated a progressive uptake of residual oxygen from the nondependent lung. This last factor should be improved by the application of nondependent lung CPAP.

Regional hypoxia also may lead to dependent lung hypoxic pulmonary vasoconstriction (HPV). The potential for dependent lung HPV is particularly important in these patients, as it will cause shunting of blood to the nondependent, nonventilated lung. This phenomenon is less important in surgery for pulmonary resection because the nondependent lung vasculature is ligated early in the surgery. Further, it has been suggested that a moderate degree of surgical stimulation could increase blood flow to the nonventilated lung, possibly by means of a local prostaglandin release.^{#12}

On the other hand, there is evidence that HPV may not be an important protective mechanism in this patient population. Patients with severe chronic obstructive pulmonary disease (COPD) have increased PA pressures and a reduced pulmonary vascular bed.¹³ In one study, the transpulmonary shunt in five COPD patients did not increase during sodium nitroprusside infusion, in contrast to the control group without COPD, in whom there was a significant increase.¹⁴ This finding suggests an impaired HPV reflex in COPD patients.¹⁵

The safety of ventilation with 100% oxygen during these laser procedures must be considered carefully. Many of these patients cannot maintain reasonable levels of oxygenation during OLV with any $F_{I_{O_2}}$ value less than 100%. In 2 yr of experience with these procedures, we have seen no evidence of a combustion hazard while using 100% oxygen. The operative lung is not ventilated during the laser ablation, and the chest cavity on the operative side is filled with room air. As an additional safety measure, we now are using air rather than oxygen for nondependent lung CPAP. The relative effectiveness of air-CPAP *versus* oxygen CPAP will be determined in further studies.

Bronchopleural air leak was a major problem during the latter part of the laser bullectomy procedure and contributed to the prolonged periods of OLV seen in the study. In two patients, air leaks could not be closed

satisfactorily through the thoracoscope and a mini-thoracotomy was performed. Pressure-controlled ventilation using a ventilator capable of delivering high gas flows at low peak pressures is essential in the period after resuming TLV. The Siemens Servo 900C ventilator in the pressure-control mode can adequately ventilate the lungs when air leaks amount to more than 50% of inspired tidal volume. Any pressure-controlled ventilator could be used, but it must be able to deliver high inspired gas flows to maintain a preset peak airway pressure in the presence of rapidly varying airway leaks or large changes in compliance. A volume-controlled ventilator with a pressure-limiting option does not accomplish this and would be inadequate. Volume-controlled ventilators deliver a preset inspired tidal volume, regardless of what fraction of that volume reaches the patient's lungs. In the presence of large bronchopleural fistulae, most of the inspired volume may not reach the lungs, the peak inspired airway pressure will decrease, and the lungs will not be distended adequately.

In conclusion, laser ablation by thoracoscopy appears to be a promising new treatment for severe bullous emphysema. This procedure is less invasive than open thoracotomy and should be better tolerated by patients with severe pulmonary impairment.⁸ Furthermore, laser ablation is practical in the treatment of diffuse bullous disease, which does not lend itself to direct surgical excision. Most of our patients demonstrated significant postoperative improvements in pulmonary function tests, and nearly all survivors were symptomatically improved. Despite the many risk factors of this population, most of them tolerated long periods of OLV. It is hoped that the anesthetic guidelines developed in this study will contribute to the safety of this procedure.

References

1. Jacobsen HC: The practical importance of thoracoscopy in surgery of the chest. *Surg Gynecol Obstet* 32:493, 1921
2. Page RD, Jeffery RR, Donnelly RJ: Thoracoscopy: A review of 121 consecutive surgical procedures. *Ann Thorac Surg* 48:66-68, 1989
3. Wakabayashi A: Thoracoscopic ablation of blebs in the treatment of recurrent or persistent spontaneous pneumothorax. *Ann Thorac Surg* 48:651-653, 1989
4. Wakabayashi A: Thoracoscopic ablation of blebs (letter). *Ann Thorac Surg* 49:851, 1990
5. Torre M, Belloni P: Nd:YAG Laser pleurodesis through thoracoscopy: New curative therapy in spontaneous pneumothorax. *Ann Thorac Surg* 47:887-889, 1989

Anderson HW, Benumof JL: Intrapulmonary shunting during one-lung ventilation and surgical manipulation (abstract). *ANESTHESIOLOGY* 55:A377, 1981.

6. Boutin C: The laser in thoracoscopy. *Pneumologie* 43:96-97, 1989
7. Kerr JH, Crampton Smith A, Prys-Roberts C, Meloche R, Foex P: Observations during endobronchial anaesthesia: II. Oxygenation. *Br J Anaesth* 46:84-92, 1974
8. Wakabayashi A, Brenner M, Kayaleh R, Berns MW, Barker SJ, Rice SJ, Tadir Y, Della Bella L, Wilson AF, *et al.*: Thoracoscopic carbon dioxide laser treatment of bullous emphysema. *Lancet* 337:881-883, 1991
9. Benumof JL: Anesthesia for thoracic surgery. Philadelphia, WB Saunders, 1987, pp 284-285
10. Dantzker DR, Wagner PD, West JB: Instability of lung units with low V_A/Q ratios during oxygen breathing. *J Appl Physiol* 38: 886-895, 1975
11. Ray JF, Yost L, Moallem S, Sanoudos GM, Villamena P, Parades RM, Clauss RH: Immobility, hypoxemia and pulmonary arteriovenous shunting. *Arch Surg* 109:537-541, 1974
12. Piper P, Vane J: The release of prostaglandins from lung and other tissues. *Ann N Y Acad Sci* 180:363-385, 1971
13. Matthay RA, Niederman MS, Weideman HP: Cardiovascular-pulmonary interaction in chronic obstructive pulmonary disease with special reference to the pathogenesis and management of cor pulmonale. *Med Clin North Am* 74:571-618, 1990
14. Casthely PA, Lear S, Cottrell JE, Lear E: Intrapulmonary shunting during induced hypotension. *Anesth Analg* 61:231-235, 1985
15. Benumof JL: Hypoxic pulmonary vasoconstriction and infusion of sodium nitroprusside (editorial). *ANESTHESIOLOGY* 50:481-483, 1979