

# Noninvasive Ventilation Reduces Mortality in Acute Respiratory Failure following Lung Resection

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When treated with invasive endotracheal mechanical ventilation (ETMV), acute respiratory insufficiency after lung resection is fatal in up to 80% of cases. Noninvasive positive-pressure ventilation (NPPV) may reduce the need for ETMV, thereby improving survival. We conducted a randomized prospective trial to compare standard therapy with and without nasal-mask NPPV in patients with acute hypoxemic respiratory insufficiency after lung resection. The primary outcome variable was the need for ETMV and the secondary outcome variables were in-hospital and 120-d mortality rates, duration of stay in the intensive care unit, and duration of in-hospital stay. Twelve of the 24 patients (50%) randomly assigned to the no-NPPV group required ETMV, versus only five of the 24 patients (20.8%) in the NPPV group ( $p = 0.035$ ). Nine patients in the no-NPPV group died (37.5%), and three (12.5%) patients in the NPPV group died ( $p = 0.045$ ). The other secondary outcomes were similar in the two groups. NPPV is safe and effective in reducing the need for ETMV and improving survival after lung resection.

**Keywords:** noninvasive ventilation; acute hypoxemic respiratory insufficiency; endotracheal intubation; invasive mechanical ventilation; lung resection

Postoperative mortality and morbidity after lung resection are decreasing, but remain significant. A recent study found 30-d mortality and morbidity rates of 4% and 23.8%, respectively, after lobectomy, and of 11.5% and 25.7%, respectively, after pneumonectomy (1). Pulmonary complications remain the leading cause of death postoperatively, and acute respiratory insufficiency after lung resection is fatal in up to 60% to 80% of cases (2). This high mortality rate is largely related to complications of postoperative reintubation and mechanical ventilation (3). Invasive endotracheal mechanical ventilation (ETMV) increases the risk of bronchial stump disruption, bronchopleural fistula, persistent air leakage, and pulmonary infection (4, 5). Thus, a major objective in the conservative treatment of acute respiratory insufficiency after lung surgery is to avoid endotracheal intubation and postoperative mechanical ventilation.

Noninvasive positive-pressure ventilation (NPPV) is safe and as efficient as ETMV in improving gas exchange in patients with various patterns of acute respiratory failure (6–8). Moreover, in several controlled studies of acute exacerbations of chronic obstructive pulmonary disease (COPD), NPPV clearly reduced the need for ETMV and the risk of lower respiratory tract infection and pneumonia, thereby reducing in-hospital morbidity and mortality (9–11). Whether NPPV can reduce the need for ETMV in patients with acute hypoxemic respiratory insufficiency (AHRI) after lung resection had not been investigated before the present study.

We compared the efficacy of standard conservative treatment with and without NPPV in avoiding ETMV in patients with AHRI after lung resection. Our prospectively defined hypothesis was that NPPV would reduce the need for ETMV and improve survival.

## METHODS

Between May 1999 and July 2000, patients who were admitted for AHRI following lung resection were recruited prospectively. The study protocol was approved by the ethics committee of our institution, and all subjects gave their informed consent to participate.

Patients were enrolled if they met at least three of the following criteria: dyspnea at rest, defined as a respiratory rate (RR) of 25 breaths/min or more; active contraction of the accessory respiratory muscles; an arterial oxygen tension ( $\text{PaO}_2/\text{FiO}_2$  ratio  $< 200$ ); and chest-radiographic abnormalities (alveolar condensation, atelectasis, or interstitial pulmonary edema).

We excluded patients with upper airway obstruction, acute respiratory failure that required specific medical treatment (pulmonary embolism, status asthmaticus, pneumothorax), excessive secretions, respiratory arrest, need for emergency ETMV, obvious excessive agitation, airways that we could not protect, unstable cardiac conditions (ventricular dysrhythmia and myocardial ischemia or infarction), more than two new organ failures, or pregnancy. These exclusions were applied equally to both study groups. Patients' level of consciousness and pH cutoffs were not considered part of the exclusion criteria.

Patients were randomly assigned to standard treatment with or without NPPV. Bronchoscopy was performed at the outset of the study to diagnose surgical complications.

## Standard Treatment

All patients received oxygen supplementation to achieve an  $\text{Sao}_2$  above 90%, bronchodilators (aerosolized albuterol), patient controlled analgesia (PCA) (bolus dose = 1 mg morphine, lockout interval 7 min, maximum hourly dose = 7 mg), and chest physiotherapy.

## NPPV

Ventilation was provided via a cushion bridge nasal mask (Profil lite; Respironics, Inc., Murrysville, PA). NPPV was provided with the BiPAP S/T-D Ventilatory Support System (Bipap Vision; Respironics, Inc.). Pressure support was increased to achieve an exhaled tidal volume of 8 to 10 ml/kg and a respiratory rate of less than 25 breaths/min. The  $\text{FiO}_2$  was adjusted to obtain a percutaneous oxygen saturation above 90%. The duration of ventilation was standardized according to Wysocki and coworkers (10).

## Criteria for ETMV

ETMV was given if at least one major criterion or two minor criteria were present. Major criteria included respiratory arrest, respiratory pauses with loss of consciousness or gasping respiration, encephalopathy, and cardiovascular instability. Minor criteria included a 20% or more increase in breathing rate or in arterial carbon dioxide tension ( $\text{Paco}_2$ ), or a decrease in  $\text{PaO}_2$ , as compared with the respective values at the study outset (9, 10).

## Follow-Up

Heart rate (HR), RR, arterial blood pressure, arterial blood gas values and chest-radiographic appearance were recorded at inclusion in the study, 2 h after treatment began, and once a day. We scored the radiographic abnormalities according to the protocol of Wysocki and co-

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workers (10). The simplified acute physiology score (SAPS II) was determined (12).

### Statistical Analysis

The primary outcome variable was the need for ETMV. Secondary outcomes were in-hospital and 120-d mortality rates, durations of intensive care unit (ICU) and hospital stay, and need for fiberoptic bronchoscopy. Results are reported as mean  $\pm$  SD. All tests are two-tailed. Continuous variables were analyzed through analysis of variance (ANOVA) and qualitative variables were analyzed with the non-parametric Mann-Whitney U test. Mortality and ETMV rates were compared by using the chi-square test. Values of  $p < 0.05$  were considered statistically significant. A Kaplan-Meier curve of cumulative survival over a period of 120 d was constructed. A log-rank test (Mantel-Cox test) was used. All statistical tests were run on the Statview Microsoft package (SAS Institute Inc., Cary, NC).

### RESULTS

During the 16-mo study period, 1,800 patients underwent lung resection at our institution, and 492 of these were admitted to our ICU. Forty-eight patients who experienced AHRI were enrolled in the study. The interim analysis of data from these 48 patients showed a significant between-group difference in the rate of ETMV: consequently, the study was stopped. Twenty-four patients were randomly assigned to standard treatment (no-NPPV group) and 24 to standard treatment plus NPPV (NPPV group). The indication for lung resection was lung cancer in all 48 cases. All the patients were extubated in the operating room. Preoperative characteristics were similar in the two groups (Table 1).

Inclusion characteristics were similar in the two groups except for a higher mean  $P_{aCO_2}$  and lower mean arterial pH in the NPPV group; both variables were within the normal range. Two hours after initiation of treatment, both groups had significant improvements in  $P_{aO_2}$  and  $P_{aO_2}/F_{iO_2}$  ratio; a decrease in breathing rate was found in both groups but was larger in the NPPV group; and a decrease in HR occurred only in the NPPV group (Table 2).

TABLE 1. PREOPERATIVE CHARACTERISTICS OF PATIENTS

	No NPPV (n = 24)	NPPV (n = 24)	p Value*
	Mean $\pm$ SD	Mean $\pm$ SD	
Age, yr	63 $\pm$ 9	58.9 $\pm$ 10	0.09
SAPS II at ICU admission	16.8 $\pm$ 4.4	16.9 $\pm$ 5.4	0.74
Pulmonary function testing			
FVC, % pred	93.2 $\pm$ 17.8	101.5 $\pm$ 20.0	0.36
FEV <sub>1</sub> , % pred	80 $\pm$ 19.7	78.5 $\pm$ 18.6	0.71
TLC, % pred	100.3 $\pm$ 18.1	97.7 $\pm$ 16.1	0.42
FRC, % pred	117.1 $\pm$ 33.5	113.1 $\pm$ 30.7	0.80
Predicted postoperative FEV <sub>1</sub> (%)	61.6 $\pm$ 15.9	61.7 $\pm$ 13.5	0.90
Arterial blood gases	N = 19	N = 15	
pH	7.41 $\pm$ 0.03	7.42 $\pm$ 0.02	0.19
$P_{aO_2}$ , mmHg	78.7 $\pm$ 8.9	67.5 $\pm$ 26.8	0.19
$P_{aCO_2}$ , mmHg	39.4 $\pm$ 4.6	34.2 $\pm$ 13.3	0.44
Type of ICU admission			
Emergency admission	7	8	0.54
Days in ICU before inclusion	1.7 $\pm$ 1.5	2.1 $\pm$ 1.9	0.57
Extent of resection			
Lobectomy	14	16	0.53
Bilobectomy	2	0	0.14
Pneumonectomy	7	7	

Definition of abbreviations: ICU = intensive care unit; NPPV = noninvasive positive pressure ventilation;  $P_{aCO_2}$  = arterial carbon dioxide tension;  $P_{aO_2}$  = arterial oxygen tension; SAPS = simplified acute physiology score.

\* p values refer to between-group comparisons.

The predicted postoperative FEV<sub>1</sub> was calculated as the proportion of remaining functional lung volume multiplied by the preoperative FEV<sub>1</sub>. The proportion of remaining functional lung volume was estimated on the basis of the extent of resection and on the lung perfusion distribution calculated from the preoperative lung perfusion scan.

Duration of NPPV was  $2.1 \pm 2.4$  (mean  $\pm$  SD) d, with  $14.3 \pm 2.8$  h of NPPV per day. The average inspiratory pressure support level was  $8.5 \pm 1.9$  cm H<sub>2</sub>O and the average expiratory pressure level was  $4 \pm 0.1$  cm H<sub>2</sub>O. Two patients were switched to a full face mask because of substantial air leakage at the mouth. One patient was dropped from the study. This patient underwent 7 d of NPPV (IPAP 20 cm H<sub>2</sub>O, EPAP 4 cm inspiratory and expiratory airway pressures (cm H<sub>2</sub>O), but chose to stop the NPPV. He underwent ETMV and tracheostomy, and survived after hospital discharge.

Twelve of the 24 patients (50%) in the no-NPPV group required ETMV, versus only five of the 24 patients (20.8%) in the NPPV group ( $p = 0.035$ ) (Table 3, Figure 1). Nine patients in the no-NPPV group (37.5%) died, versus only three (12.5%) in the NPPV group ( $p = 0.045$ ). The durations of ICU stay and of hospital stay were similar in the two groups. In neither group did any patients die after hospital discharge, so that in both groups, in-hospital mortality was equal to 120-d mortality (Table 3, Figure 2).

Of the 17 patients who required ETMV, nine in the no-NPPV group and one in the NPPV group were started on ETMV during the first 48 h after lung resection (Figure 1). The reasons for ETMV in the no-NPPV group (12 patients) were severe hypoxia with polypnea in all patients (average  $P_{aO_2}/F_{iO_2}$  ratio of  $111.6 \pm 54.3$ ), which was associated with encephalopathy in six patients and with hemodynamic instability in one patient. Among the patients who underwent ETMV in the no-NPPV group, only two had a  $P_{aO_2}/F_{iO_2}$  ratio above 200: one had a  $P_{aO_2}/F_{iO_2}$  ratio of 223.33 (the criterion for ETMV was encephalopathy), and another had a  $P_{aO_2}/F_{iO_2}$  ratio of 201.47 (the criterion for ETMV was cardiovascular instability). With the exception of these two patients, the average  $P_{aO_2}/F_{iO_2}$  ratio was  $86.36 \pm 11.56$ . Similarly, the reasons for ETMV in the NPPV group (five patients) were encephalopathy ( $p < 0.05$ ), which was associated with dyspnea in one patient, with hypoxia in three patients, and with polypnea and hypoxia in one patient. The average  $P_{aO_2}/F_{iO_2}$  ratio was  $124.37 \pm 50.2$  (Table 4).

None of the preoperative or study inclusion variables were predictive of mortality (Table 5). Of the 12 patients who died, all had received ETMV. The causes of death were end-stage multiple organ failure, accompanied in five cases by bronchopleural fistula and pyothorax. One patient died in the surgical ward 2 wk after ICU discharge. Overall 120-d survival was similar to in-hospital survival in the two study groups. During the study period we did not notice any complication directly attributable to eTMV or NPPV.

### DISCUSSION

This study shows that use of NPPV in patients with AHRI after lung resection can reduce the need for ETMV and reduce in-hospital and 3-mo mortality rates. The main objective of the treatment of AHRI after lung surgery is to avoid ETMV. A recent retrospective study found a 76% postoperative mortality rate in patients who required ETMV after lung resection (13). Similarly, a retrospective evaluation of 600 lung resection patients (F. Parquin, personal communication) and the present prospective study found mortality rates of 80% and 67%, respectively, in patients who required ETMV. By contrast, all of our patients with postoperative AHRI, who did not require ETMV survived. Causes of death related to ETMV include bronchial stump disruption, bronchopleural fistula, persistent air leakage, and pulmonary infection (4, 5).

Although the use of NPPV is well established in AHRF of nonsurgical origin (6, 10), studies of NPPV after chest surgery are lacking. The present prospective randomized controlled

TABLE 2. CHARACTERISTICS OF PATIENTS AT INCLUSION AND 2 h AFTER TREATMENT INITIATION\*

	No-NPPV		NPPV		p Value*
Inclusion criteria					
Respiratory rate > 25, n	22		21		0.55
Accessory muscle recruitment, n	17		19		0.47
PaO <sub>2</sub> /FiO <sub>2</sub> < 200, n	23		23		
Chest radiographic alterations, n	23		23		
Patient characteristics					
SAPS II at inclusion	17.2 ± 4.8		15.3 ± 5.1		0.23
Chest radiograph score	4.5 ± 2.6		5.8 ± 2.5		0.11
Arterial lactate level	1.5 ± 0.5		1.5 ± 1.0		0.27
Diagnosis at inclusion					
Interstitial pulmonary edema	18		20		0.43
Atelectasis	20		20		
Documented pneumonia <sup>‡</sup>	7		6		0.74
	Inclusion	2 h	Inclusion	2 h	
Systolic pressure, mmHg	136.0 ± 25.2	123.9 ± 16.2 <sup>†</sup>	127.86 ± 24.75	124.2 ± 15.6	
Heart rate, beats/min	117.1 ± 32.0	114.1 ± 28.0	112.9 ± 21.5	98.52 ± 13.80 <sup>‡§</sup>	
Respiratory rate, breaths/min	28.8 ± 4.2	24 ± 5.8 <sup>†</sup>	27.1 ± 7.2	20.78 ± 3.25 <sup>‡§</sup>	
pH	7.42 ± 0.06	7.43 ± 0.06	7.38 ± 0.08 <sup>‡</sup>	7.38 ± 0.08 <sup>§</sup>	
PaO <sub>2</sub> , mmHg	68.9 ± 13.6	93 ± 36.8 <sup>†</sup>	71.7 ± 23.4	122.5 ± 61.3 <sup>‡§</sup>	
PaCO <sub>2</sub> , mmHg	38.9 ± 5.1	39.6 ± 5.2	44.3 ± 8.6 <sup>‡</sup>	45.0 ± 11.8	
PaO <sub>2</sub> /FiO <sub>2</sub>	127.1 ± 42.5	155.6 ± 53.7 <sup>†</sup>	126.8 ± 42.1	199.1 ± 8 <sup>‡§</sup>	

Definition of abbreviations: NPPV = noninvasive positive pressure ventilation; PaCO<sub>2</sub> = arterial carbon dioxide tension; PaO<sub>2</sub> = arterial oxygen tension; SAPS = simplified acute physiology score.

\* p values refer to the between-group comparisons at inclusion.

<sup>†</sup> p < 0.05 for within-group comparisons of data at study inclusion and data 2 h after treatment initiation.

<sup>‡</sup> p < 0.05 for the between-group comparison at study inclusion.

<sup>§</sup> p < 0.05 for the between-group comparison two hours after treatment initiation.

<sup>‡</sup> Bacterial cultures revealed the following organisms: group 1, 2 Haemophilus influenzae, 1 Streptococcus pneumoniae, 1 Pseudomonas aeruginosa, 2 Escherichia coli; group 2, 5 Haemophilus influenzae, 1 Branhamella catarrhalis, 1 Enterobacter freundii.

study provides the first evidence that NPPV may be effective in AHRI complicating lung resection surgery. At the interim analysis, we found that NPPV significantly decreased the rate of ETMV use, from 50% to 20.8%. Consequently, we discontinued the study. As compared with standard treatment alone, NPPV reduced in-hospital mortality from 37.5% to 12.5%, most likely by avoiding ETMV-related complications such as tracheobronchial bacterial contamination, bronchopleural fistula, and pyothorax (14, 15).

The benefits observed in the NPPV group were not ascribable to greater severity of illness at inclusion in the no-NPPV group. The patients in both groups had similar physiologic characteristics and severity scores before surgery and at study inclusion; in particular, the extent of lung resection was similar in the two groups. Because cardiogenic lung edema responds

positively to NPPV, such an effect could have been implicated in our patients with postoperative lung edema. However, none had evidence of heart disease on preoperative echocardiography. Moreover, several clinical and experimental studies have demonstrated that lung edema after lung resection is non-cardiogenic (16–18). The NPPV group showed a significant reduction in RR and HR, as well as a significant improvement in the PaO<sub>2</sub>/FiO<sub>2</sub> ratio as compared with the control group. This indicates that NPPV relieves hypoxemic respiratory failure. The lack of improvement in mean PaCO<sub>2</sub> with NPPV was due to the inclusion of two hypercapnic patients. These two patients' PaCO<sub>2</sub> values worsened with NPPV, from 69 and 64 mm hg, respectively, at the study outset to 75 and 82 mm hg after 2 h, leading to intubation. In the rest of the group, NPPV improved PaCO<sub>2</sub> and pH values. We are therefore confident that NPPV was effective in improving ventilation and thus in decreasing the need for ETMV. This observation also suggests that severe hypoxia associated with acute postoperative hypercapnia may constitute a relative contraindication to NPPV.

In most cases, target blood gas levels were achieved with 8 cm H<sub>2</sub>O of inspiratory pressure and 4 cm H<sub>2</sub>O of expiratory pressure, as described in the literature (8, 19, 20, 25). Higher inspiratory pressures were required in COPD (21) or after upper abdominal surgery (22). This suggests that the magnitude of the appropriate pressure boost may vary with the stage and type of the respiratory disease, as well as with respiratory compliance. Failure of NPPV occurred in five of our patients, and was associated with an increase in PaCO<sub>2</sub>, development of encephalopathy, patient-ventilator asynchrony, air leaks, and increasing patient discomfort despite an increase in inspiratory pressure support.

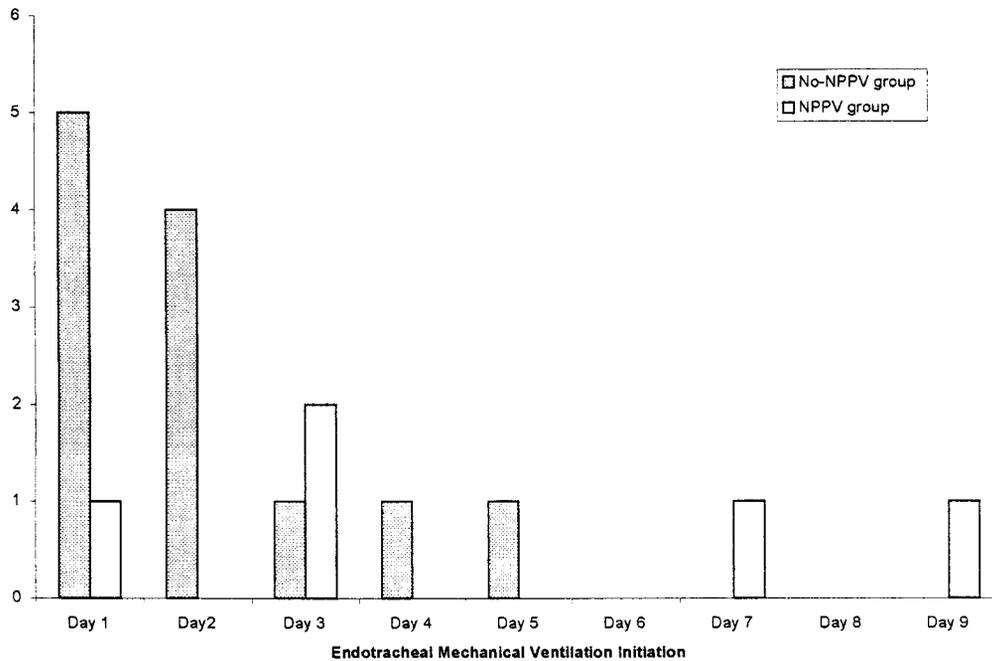
TABLE 3. ENDOTRACHEAL MECHANICAL VENTILATION, MORTALITY, AND LENGTH OF INTENSIVE CARE UNIT AND HOSPITAL STAYS

	No-NPPV (n = 24) Mean ± SD	NPPV (n = 24) Mean ± SD	p Value*
ETMV, n (%)	12 (50%)	5 (20.8%)	0.035
In-hospital deaths, n (%)	9 (37.5%)	3 (12.5%)	0.045
Length of ICU stay, d	14 ± 11.1	16.65 ± 23.6	0.52
Length of hospital stay, d	22.8 ± 10.7	27.1 ± 19.5	0.61
120 - d mortality, n (%)	9 (37.5%)	3 (12.5%)	0.045

Definition of abbreviations: ETMV = endotracheal mechanical ventilation; ICU = intensive care unit; NPPV = noninvasive positive pressure ventilation

\* p values are for the between-group comparisons for each variable.

Number of patients



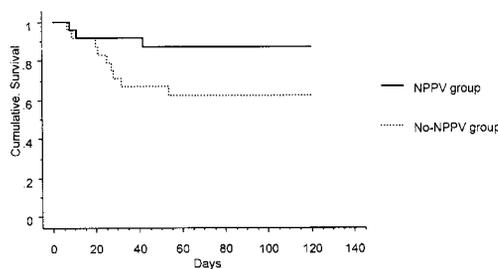
**Figure 1.** Number of patients who required ETMV on each study day. On the 17 patients who required ETMV, nine in the no-NPPV group and one in the NPPV group were started on ETMV within 48 h of inclusion. Physiologic characteristics at the time of initiation of ETMV were similar in the two groups.

In contrast to previous studies of NPPV in nonsurgical patients (9, 10), our study found no evidence that NPPV reduced the length of stay in the ICU or hospital, even when survivors alone were considered. This is probably attributable to the long duration of our weaning protocol. Further studies are needed to evaluate weaning protocols for NPPV, as previously done for ETMV (23). The mean duration of NPPV per day was about 14 h in our patients. Except for the study by Kramer and colleagues (20) in which the average duration of was over 20 h for the first day and 14 h during the second day), the mean duration of NPPV in our study was two times longer than in other studies (9, 21, 24). Despite this longer duration, NPPV through a nasal mask was well tolerated, with only two patients requiring a full face mask and only one patient deciding to stop NPPV.

Our study had several limitations. It is difficult to eliminate a bias when a study cannot be blinded. Furthermore, the use of severe hypoxia with clinical intolerance as a criterion for intubation may have led to a higher rate of intubation in the no-NPPV group because of the salutary effect of NPPV on oxygenation. The ability of such patients to fully understand the

information provided to them can be questioned. All patients and their families received a written informed consent form that was available at their bedside, and NPPV could be discontinued at the patient's or the family's request.

In conclusion, NPPV is a safe and effective means of reducing the need for ETMV and improving survival in patients with AHRI after chest surgery. This suggests that NPPV should be added to the standard conservative therapy of AHRF complicating lung resection.



**Figure 2.** Kaplan-Meier 120-d cumulative survival. Nine patients died in the no-NPPV group (37.5%), versus only three in the NPPV group (12.5%) ( $p < 0.005$ ). The 120-d mortality rate was equal to the in-hospital mortality rate. The log-rank test value for Kaplan-Meier survival was 0.05.

**TABLE 4. CHARACTERISTICS AT THE TIME INVASIVE MECHANICAL VENTILATION WAS BEGUN**

	No-NPPV (n = 12) Mean ± SD	NPPV (n = 5) Mean ± SD	p Value*
Reason for ETMV			
Encephalopathy	6	5	0.01
> 20% rise in respiratory rate versus baseline	12	2	0.01
> 20% fall in Pao <sub>2</sub> versus baseline	12	4	0.11
Hemodynamic or cardiovascular instability	1	0	0.50
Physiologic characteristics			
Heart rate, beats/min	114.2 ± 15.6	119 ± 14.9	0.52
Systolic blood pressure, mm Hg	131 ± 30.1	107 ± 36.6	0.17
Respiratory rate, breath/min	29.5 ± 6.9	26.25 ± 13.2	0.77
Arterial lactate level	1.6 ± 0.4	1.3 ± 0.5	0.30
Arterial pH	7.41 ± 0.07	7.32 ± 0.102	0.15
Pa <sub>O<sub>2</sub></sub> , mmHg	65.9 ± 19.9	74.6 ± 10.9	0.15
Pa <sub>CO<sub>2</sub></sub> , mmHg	43.4 ± 9.3	63.9 ± 20.5	0.07
Pa <sub>O<sub>2</sub></sub> /F <sub>I<sub>O<sub>2</sub></sub></sub>	111.6 ± 54.3	124.375 ± 50.2	0.67

*Definition of abbreviations:* ETMV = endotracheal mechanical ventilation; NPPV = noninvasive positive pressure ventilation; Paco<sub>2</sub> = arterial carbon dioxide tension; Pao<sub>2</sub> = arterial oxygen tension.

\* p values are for between-group comparisons at the time of initiation of invasive mechanical ventilation (IMV)

**TABLE 5. CHARACTERISTICS IN THE PATIENTS WHO DIED AND IN THOSE WHO SURVIVED**

	Patients Who Survived (n = 36) Mean ± SD	Patients Who Died (n = 12) Mean ± SD	p Value
Age, yr	60 ± 10.0	63 ± 9.5	0.37
Weight/height ratio	0.4 ± 0.08	0.4 ± 0.05	0.79
Pulmonary function testing			
FVC, % pred	99.1 ± 20.1	91.3 ± 14.6	0.303
FEV <sub>1</sub> , % pred	79.1 ± 19.1	78.1 ± 19.1	0.84
TLC, % pred	98.3 ± 16.8	101.1 ± 18.1	0.84
FRC, % pred	115.1 ± 32.1	114.9 ± 31.2	0.94
Preoperative arterial blood gases			
pH	7.42 ± 0.03	7.41 ± 0.02	0.31
Pa <sub>O<sub>2</sub></sub> , mm Hg	75.3 ± 17.1	66.9 ± 28.7	0.39
Pa <sub>CO<sub>2</sub></sub> , mm Hg	36.92 ± 8.9	37.05 ± 15.2	0.08
Characteristics at study inclusion			
Postoperative FEV <sub>1</sub> , % pred	61.4 ± 14.6	62.8 ± 14.0	0.65
SAPS II at ICU admission	16.8 ± 5.3	17.3 ± 3.2	0.80
SAPS II at inclusion	15.7 ± 5.02	18.3 ± 4.6	0.11
Heart rate, beats/min	115.9 ± 28.4	111.4 ± 20.6	0.93
Systolic blood pressure, mm Hg	132.6 ± 25.7	128.7 ± 23.3	0.85
Respiratory rate, breaths/min	27.6 ± 6.2	29 ± 4.7	0.75
Chest radiograph score	5.1 ± 2.9	5.2 ± 1.6	0.62
Arterial lactate level	1.5 ± 0.8	1.7 ± 0.7	0.25
Arterial pH	7.39 ± 0.07	7.42 ± 0.1	0.51
Pa <sub>O<sub>2</sub></sub> , mm Hg	71.6 ± 20.3	64.4 ± 11.8	0.37
Pa <sub>CO<sub>2</sub></sub> , mm Hg	42.7 ± 7.6	37.7 ± 5.7	0.08
Pa <sub>O<sub>2</sub></sub> /F <sub>O<sub>2</sub></sub>	126.9 ± 41.5	127.2 ± 45.3	0.92

Definition of abbreviations: ICU = intensive care unit; Pa<sub>CO<sub>2</sub></sub> = arterial carbon dioxide tension; Pa<sub>O<sub>2</sub></sub> = arterial oxygen tension; SAPS = simplified acute physiology score.

\* p values are for between-group comparisons of preoperative data.

The predicted postoperative FEV<sub>1</sub> was calculated as the proportion of remaining functional lung volume multiplied by the preoperative FEV<sub>1</sub>. The proportion of remaining functional lung volume was estimated on the basis of the extent of resection and on the lung perfusion distribution calculated from the preoperative lung perfusion scan.

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