

Noninvasive Ventilation and Alveolar Recruitment Maneuver Improve Respiratory Function during and after Intubation of Morbidly Obese Patients

A Randomized Controlled Study

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ABSTRACT

Background: Morbid obesity predisposes patients to lung collapse and hypoxemia during induction of anesthesia. The aim of this prospective study was to determine whether noninvasive positive pressure ventilation (NPPV) improves arterial oxygenation and end-expiratory lung volume (EELV) compared with conventional preoxygenation, and whether NPPV followed by early recruitment maneuver (RM) after endotracheal intubation (ETI) further improves oxygenation and respiratory function compared with NPPV alone.

Methods: Sixty-six consecutive patients (body mass index, $46 \pm 6 \text{ kg/m}^2$) were randomized to receive 5 min of either

What We Already Know about This Topic

- Morbidly obese patients have a greater risk for developing hypoxemia because of decreased lung volume during anesthesia induction

What This Article Tells Us That Is New

- In 66 morbidly obese patients, a combination of preoxygenation with noninvasive positive pressure ventilation and a recruitment maneuver immediately after tracheal intubation more effectively maintains both lung volume and oxygenation during anesthesia induction than preoxygenation alone with either pure oxygen or noninvasive positive pressure ventilation

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conventional preoxygenation with spontaneous breathing of 100% O₂ (CON), NPPV (pressure support and positive end-expiratory pressure), or NPPV followed by RM (NPPV+RM). Gas exchange was measured in awake patients, at the end of preoxygenation, immediately after ETI, and 5 min after the onset of mechanical ventilation. EELV was measured immediately after ETI and 5 min after mechanical ventilation. The primary endpoint was arterial oxygenation 5 min after the onset of mechanical ventilation. Results are presented as mean \pm SD.

Results: At the end of preoxygenation, PaO₂ was higher in the NPPV and NPPV+RM groups ($382 \pm 87 \text{ mmHg}$ and $375 \pm 82 \text{ mmHg}$, respectively; both $P < 0.001$) compared with the CON group ($306 \pm 51 \text{ mmHg}$) and remained higher after ETI ($225 \pm 104 \text{ mmHg}$ and $221 \pm 110 \text{ mmHg}$, in the NPPV and NPPV+RM groups, respectively; both $P < 0.01$ compared with the CON group [$150 \pm 50 \text{ mmHg}$]). After the onset of mechanical ventilation, PaO₂ was $93 \pm 25 \text{ mmHg}$ in the CON group, $128 \pm 54 \text{ mmHg}$ in the NPPV group ($P = 0.035$ vs. CON group), and $234 \pm 73 \text{ mmHg}$ in the NPPV+RM group ($P < 0.0001$ vs. NPPV group). After ETI, EELV was higher in the NPPV group compared with the CON group ($P < 0.001$). Compared with NPPV alone, RM further improved gas exchange and EELV (all $P < 0.05$). A significant correlation was found between PaO₂ obtained 5 min after mechanical ventilation and EELV ($R^2 = 0.41$, $P < 0.001$).

Conclusion: NPPV improves oxygenation and EELV in morbidly obese patients compared with conventional preoxygenation. NPPV combined with early RM is more effective than NPPV alone at improving respiratory function after ETI.

IN morbidly obese patients, general anesthesia induces a greater reduction in lung volume and greater atelectasis formation,¹ with major alterations in oxygenation and respiratory mechanics.² Because obese patients may be at increased risk of difficult airway management,^{3–5} prevention or reversal of respiratory function alterations is particularly important in these patients.¹

The use of noninvasive positive pressure ventilation (NPPV) in the operating room for preoxygenation of morbidly obese patients has seldom been reported. Previous studies demonstrated that continuous positive airway pressure applied during induction of anesthesia reduces atelectasis formation and the duration of nonhypoxic apnea in nonobese^{6,7} and obese^{8,9} patients and improves lung function when applied after extubation.¹⁰ NPPV should increase the end-expiratory lung volume (EELV) by recruiting collapsed alveoli, thereby increasing the oxygen reserves held within the lungs.^{11,12} In a recent randomized study, no significant effect on arterial oxygenation after tracheal intubation has been shown, therefore suggesting that the beneficial effects on lung volume and alveolar recruitment might be inconstant and transient after discontinuation of NPPV.¹²

A recruitment maneuver (RM) consisting of a transient increase in inspiratory pressure¹³ was found to reduce anesthesia-induced atelectasis and to improve oxygenation when combined with positive end-expiratory pressure (PEEP) in obese patients.^{14–16} Although NPPV should recruit collapsed alveoli,¹¹ there is no clear evidence to date of an additional benefit of RM over NPPV when applied after tracheal intubation.

The aim of this study was to determine whether NPPV improves oxygenation and EELV more efficiently than conventional methods at induction of anesthesia, and whether RM applied after NPPV further improves respiratory function after initiation of mechanical ventilation.

Materials and Methods

Study Population

Seventy consecutive adult patients with American Society of Anesthesiologists Physical Status Classification scores of II–III and a body mass index greater than or equal to 40 kg/m² and who were scheduled for laparoscopic sleeve gastrectomy or Roux-en-Y gastric bypass were included in this prospective, single-blind, randomized study. The study was approved by the institutional review board (Clermont-Ferrand, France), and written informed consent was obtained from all patients (Trial Registration: Clinicaltrials.gov Identifier: NCT00852384).

Exclusion criteria were: consent refusal; age younger than 18 yr; pregnancy; severe cardiac failure, defined as New York Heart Association classification greater than III; and chronic obstructive pulmonary disease, defined as forced expiratory volume in 1 s less than 80% of expected value.

Study Design and Intervention

Patients fasted for 6 h and received oral premedication with hydroxyzine and cimetidine in sodium citrate before anesthesia. The study was conducted in the operating room with the patient in the beach chair position, as described previously,¹⁷ after placement of a five-lead electrocardiograph, a pulse oximeter (S/5 monitor; Datex-Engstrom, Helsinki, Finland), and intravenous crystalloid infusion. The radial artery was cannulated (Leader-Cath®; Vygon, Ecouen, France) during local anesthesia for continuous monitoring of arterial blood pressure and arterial blood gas measurements and continuous pulse pressure variation monitoring. In each patient, additional boluses of colloid (HES 130/0.4/6%; Voluven, Fresenius Kabi, Bad Hamburg, Germany) were given to maintain pulse pressure variation less than 13%.¹⁸ Anesthetists with sufficient experience in the management of morbidly obese patients performed all anesthesia procedures.

The study design is summarized in figure 1. Patients were randomly assigned to one of the three intervention groups using a concealed allocation approach (computer-generated codes) with sealed envelopes: (1) conventional preoxygenation: spontaneous breathing *via* a facemask (CON group); (2) NPPV (NPPV group); and (3) NPPV + RM (NPPV+RM group): NPPV followed by RM applied after tracheal intubation. In all patients, preoxygenation consisted of a single 5-min trial. In the CON group, spontaneous breathing at an inspired oxygen concentration (FiO₂) of 100% was performed using the main circuit of the ventilator (Cato, Dräger™; Lübeck, Germany) with 15 l/min fresh gas flow and the adjustable pressure-limiting valve fully opened. Patients were asked to breathe at their usual tidal volume. In the NPPV and NPPV+RM groups, pressure support ventilation (PSV) was adjusted to attain an expiratory tidal volume (VT_e) of 8 ml/kg predicted body weight, a PEEP level of 6–8 cm H₂O, and an airway pressure (pressure support level + PEEP) of no more than 18 cm H₂O (Engström CareStation; Datex-Ohmeda, General Electric, Helsinki, Finland). The inspiratory trigger sensitivity was set at its minimal value to detect minimal inspiratory effort while avoiding autotriggering. FiO₂ was set at 100%. In all patients, preoxygenation was performed using a facemask surrounding the nose and mouth (Vmask™; Hans Rudolph Inc., Shawnee, KS), fitted tightly to the face to prevent air leaks.

At the end of the 5-min administration of oxygen, general anesthesia was induced using propofol (2 mg/kg), remifentanyl (0.25 µg · kg⁻¹ · m⁻¹), and succinylcholine. The mask was maintained until the onset of apnea, defined as the loss of expiratory flow on the monitor, and no manual ventilation was attempted before intubation (cuffed tube 7–7.5; Portex,

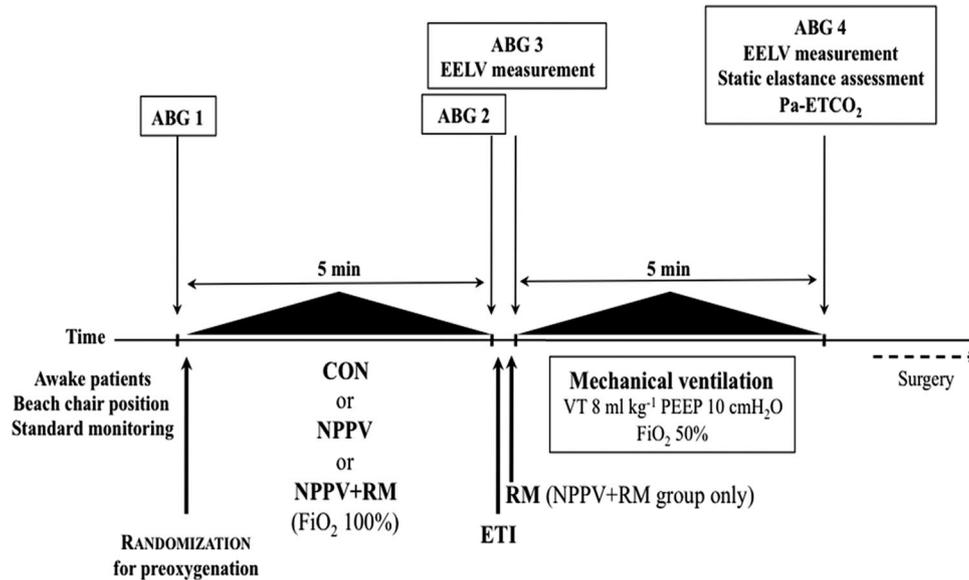


Fig. 1. Schema of the procedure. Standard clinical parameters were recorded, and arterial blood gases (ABG 1) were sampled just before preoxygenation. In all patients, preoxygenation consisted in a single 5-min trial. Additional ABG measurements were performed at the end of preoxygenation (ABG 2), immediately after ETI (ABG 3), and 5 min after the onset of mechanical ventilation (ABG 4). In all patients, EELV measurements were performed the day before the experiment (in awake patients), after tracheal intubation, and after the 5-min period of mechanical ventilation. CON = conventional preoxygenation; EELV = end-expiratory lung volume; ETI = endotracheal intubation; NPPV = noninvasive positive pressure ventilation; Pa-ETCO₂ = arterial to end-tidal partial pressure of carbon dioxide; PEEP = positive end-expiratory pressure; RM = recruitment maneuver; VT = tidal volume; FiO₂ = inspired fraction of oxygen.

London, England). Tracheal intubation was performed with patients in the beach chair position. Anesthesia was maintained by continuous infusion of propofol and remifentanyl to target a bispectral index between 40 and 50. No patient required fiberoptic intubation. The duration of anesthesia induction (defined as the time between the end of preoxygenation and intubation) and episodes of desaturation (defined as a drop in oxygen saturation measured by pulse oximetry [SpO₂] less than 92% before intubation) were recorded for all patients. Immediately after intubation, patients were ventilated in the volume-controlled mode with a VTE of 8 ml/kg predicted body weight, a respiratory rate adjusted to maintain an arterial carbon dioxide tension of 35–42 mmHg, an inspiratory/expiratory ratio of 0.5, FiO₂ of 0.5, and PEEP of 10 cm H₂O.

In the NPPV+RM group, the RM consisted of applying a continuous positive airway pressure of 40 cm H₂O for 40 s. With a decrease in systolic arterial pressure of 20% or greater, the RM would have been discontinued.

Physiologic Measurements

Arterial blood samples were taken 1–2 min before and immediately after the 5-min preoxygenation trial, immediately after intubation, and 5 min after the onset of mechanical ventilation at a PEEP of 10 cm H₂O (IL Synthesis, Instrumentation Laboratory®, Lexington, MA). The end-tidal oxygen concentration (ET_{O₂}, expressed as a percentage of atmospheric pressure), for which a value of more than 90% defined adequate denitrogenation,¹⁹ and expired concentration

of carbon dioxide (ET_{CO₂}) were measured breath-by-breath with a calibrated gas analyzer located in the ventilator with a sample line connected to the filter placed between the Y-piece and the mask. Arterial to end-tidal partial pressure of carbon dioxide (Pa-ETCO₂), a useful indicator of lung collapse and re-opening that has been found to correlate closely with atelectatic lung area on computed tomography (CT),²⁰ was recorded after the 5-min period of mechanical ventilation.

In all patients, a preoperative lung function test was performed 1–2 days before surgery, and EELV measurements were obtained using the helium dilution method (Spirodyne®; Dyn'R, Muret, France) in a 30° head-up position at end expiration to determine reference EELV values when awake. Subsequent EELV measurements (fig. 1) were performed using an automated procedure available on the ventilator (COVX module; General Electric Healthcare). This requires a FiO₂ step change of 0.1, without interruption of mechanical ventilation or any need for supplementary tracer gases, as described in detail elsewhere.^{21,22} In all patients, EELV values were indexed according to predicted body weight, which was calculated for men as 50 + 0.91 (height [cm] – 152.4), and for women as 45.5 + 0.91 (height [cm] – 152.4), as described previously.²³ EELV measurements were performed immediately after arterial blood sampling to avoid any influence of the step change in FiO₂ on blood gas analysis. Previous studies have specifically evaluated the reproducibility, accuracy, and precision of EELV measurements provided by the module.^{22,23}

Peak airway pressure (P_{MAX}) and plateau end-inspiratory airway pressure were recorded 5 min after the onset of me-

Table 1. Patient Characteristics

	CON Group (n = 22)	NPPV Group (n = 22)	NPPV+RM Group (n = 22)	Overall Group Effect
Age (yr)	41 ± 9	42 ± 10	43 ± 11	<i>P</i> = 0.37
Sex (male/female)	5/17	6/16	8/14	<i>P</i> = 0.56
Height (m)	167 ± 10	167 ± 8	169 ± 8	<i>P</i> = 0.88
Weight (kg)	130 ± 28	128 ± 20	128 ± 17	<i>P</i> = 0.94
BMI (kg · m ⁻²)	46 ± 4	46 ± 2	45 ± 5	<i>P</i> = 0.78
Smoking history (no. patients)	7	5	6	<i>P</i> = 0.79
W/H ratio	0.94 ± 0.07	0.95 ± 0.07	0.97 ± 0.08	<i>P</i> = 0.28
Male	1.05 ± 0.04	1.02 ± 0.05	1.04 ± 0.08	<i>P</i> = 0.78
Female	0.91 ± 0.04	0.92 ± 0.05	0.93 ± 0.04	<i>P</i> = 0.16

Values are presented as mean ± SD or absolute value.

BMI = body mass index; CON = conventional preoxygenation; NPPV = noninvasive positive pressure ventilation; NPPV+RM = NPPV and recruitment maneuver; RM = recruitment maneuver; W/H ratio = waist-to-hip ratio.

chanical ventilation. The quasistatic elastance of the respiratory system was calculated as $\Delta P_{aw}/V_{Te}$ where ΔP_{aw} is the difference between plateau end-inspiratory airway pressure and end-expiratory pressure at a period of no flow (corrected for intrinsic PEEP), and V_{Te} is the expiratory tidal volume.

Statistical Analysis

The primary endpoint was arterial oxygenation (P_{aO_2}) 5 min after the onset of mechanical ventilation. To calculate the sample size, data from a previous study were used.¹² Assuming a 30% absolute increase in P_{aO_2} in the NPPV+RM group compared with the other groups, with a type I error of 5% and a power of 90%, a sample of 22 patients was required in each group. The secondary endpoints were P_{aO_2} at other time points (at the end of preoxygenation and after tracheal intubation), EELV after intubation and 5 min after the onset of mechanical ventilation, static elastance of the respiratory system, and P_{a-ETCO_2} .

All data are expressed as mean ± SD or median [interquartile range], according to the type of variable distribution. The chi-square test² was used to compare qualitative data. Qualitative and quantitative data were correlated using the Student *t* test or analysis of variance (ANOVA) when normally distributed (and variances equivalent), and the Kruskal-Wallis H-test was used in other circumstances. ANOVA was used to examine the effect of the treatment groups on arterial oxygenation. Comparisons of EELV, P_{a-ETCO_2} , respiratory mechanics, and hemodynamics were performed using the Kruskal-Wallis H-test or ANOVA as appropriate. The paired Student *t* test was used to compare data between two points in time. All *post hoc* analyses were performed with the Bonferroni correction when appropriate. Multiple regression analysis was carried out to correlate oxygenation (P_{aO_2}) with EELV after the 5-min period of mechanical ventilation. All tests were two-tailed, and *P* values less than 0.05 were considered statistically significant. All analyses were performed using SEM software (version 2.0, center Jean-Perrin; Clermont-Ferrand, France).²⁴

Results

From January through August 2009, 70 consecutive patients were assessed for eligibility. Three patients were excluded because of refusal to participate, and one patient was excluded because of $FEV_1 < 80\%$ of expected value. Consequently, 66 patients who fulfilled the study inclusion criteria were enrolled (n = 22 in each group) and were included in the analysis. Complete follow-up data were available for all 66 patients. The characteristics of the patients were similar in the three groups (table 1). There was no significant difference in preoperative reference EELV values between groups (overall group effects, *P* = 0.96; fig. 2). There was no significant difference in the P_{aO_2} values at baseline between groups (fig. 2). The comparison of the variance for the three groups with the grouped variance was: *F* = 1.32 (*P* = 0.41), *F* = 1.62 (*P* = 0.19) and *F* = 1.61 (*P* = 0.18) in the CON, NPPV, and NPPV+RM groups, respectively. No relevant clinical problem occurred during the procedure, and neither preoxygenation nor RM was stopped prematurely. The duration of anesthesia induction was 215 ± 40 s, 219 ± 51 s, and 217 ± 44 s in the CON, NPPV, and NPPV+RM groups, respectively (*P* = 0.96). There was no significant difference in hemodynamics between the groups (table 2). No patient developed postoperative pulmonary complications defined as the need for mechanical ventilation postoperatively.

There was no significant difference in PSV (9 ± 2 vs. 9 ± 1 cm H₂O in the NPPV and NPPV+RM groups, respectively; *P* = 0.89) and PEEP levels (7 ± 1 cm H₂O in each group; *P* = 1.00) between the NPPV and NPPV+RM groups. At the end of the 5-min preoxygenation trial, P_{aO_2} was significantly higher in the NPPV and NPPV+RM groups compared with the CON group (382 ± 87 mmHg and 375 ± 82 mmHg vs. 306 ± 51 mmHg, respectively; both *P* < 0.001). In comparison with awake patients, P_{aCO_2} was significantly lower in the three groups (both *P* less than 0.001; table 2). Mean ET_{O_2} values were higher in the NPPV and NPPV+RM group compared with the CON group (95.6 ± 1.1% and 96 ± 1.0% vs. 92.3 ± 2.3%, both *P* < 0.01). In

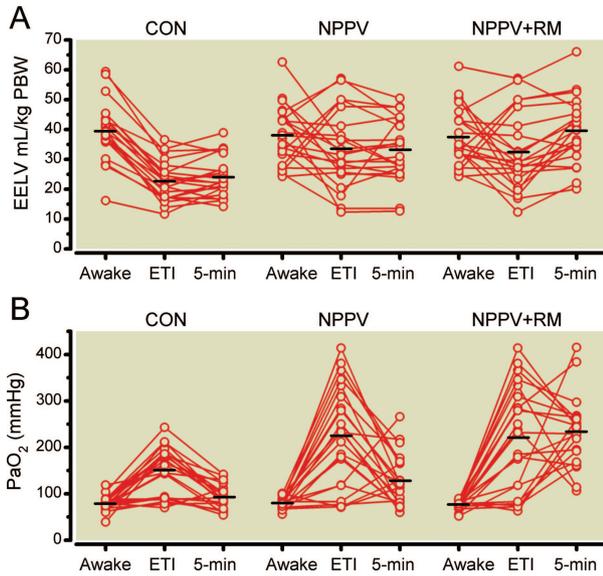


Fig. 2. Progression of EELV (A) and PaO_2 values (B) in individual patients throughout the experiment in the CON, NPPV, and NPPV+RM groups. Black dots represent the mean values for EELV and PaO_2 in each group of patients. Compared with conventional preoxygenation, NPPV and NPPV+RM increased both EELV after ETI (both P values less than 0.001), without significant difference ($P = 0.91$) between the two groups. In contrast to NPPV alone, NPPV+RM further increased EELV 5 min after mechanical ventilation ($P = 0.03$). After ETI, PaO_2 was also significantly higher in the NPPV and NPPV+RM groups compared with the CON group, without any significant difference ($P = 0.90$) between the NPPV and NPPV+RM groups. After initiation of mechanical ventilation, PaO_2 remained higher in the NPPV+RM group than in the NPPV and CON groups ($P < 0.0001$ and $P < 10^{-5}$, respectively). *Post hoc* analyses were performed using the Bonferroni correction test. CON = conventional preoxygenation; EELV = end-expiratory lung volume; ETI = endotracheal intubation; NPPV = noninvasive positive pressure ventilation; PaO_2 = arterial partial pressure of oxygen; PBW = predicted body weight; RM = recruitment maneuver.

addition, although all patients had ETO_2 values greater than 90%, more patients in the CON group had ETO_2 values less than 95% at the end of preoxygenation (72% vs. 14% and 9% in the NPPV and NPPV+RM group, respectively, $P < 0.01$). Five patients (23%) in the CON group, two (9%) in the NPPV group, and two (9%) in the NPPV+RM group had SpO_2 less than or equal to 92% before intubation ($P = 0.31$).

After intubation, PaO_2 remained significantly higher in the NPPV and NPPV+RM groups compared with the CON group, without any significant difference ($P = 0.90$) between the NPPV and NPPV+RM groups (fig. 2). EELV was significantly higher in the NPPV and NPPV+RM groups compared with conventional preoxygenation (both $P < 0.001$), without a significant difference between the two groups ($P = 0.91$, fig. 2). EELV was 58%, 88%, and 87% of the preoperative EELV (awake) values in the CON, NPPV, and NPPV+RM groups,

respectively, with a significant difference ($P = 0.002$) between the groups.

After initiation of mechanical ventilation, PaO_2 was significantly higher in the NPPV group compared with the CON group (128 ± 54 mmHg vs. 93 ± 25 mmHg, $P = 0.035$) (fig. 2), whereas PaCO_2 was unaffected (table 2). Compared with the other two groups, gas exchange was further improved in the NPPV+RM group. Compared with the NPPV group, PaO_2 was significantly higher in the NPPV+RM group (234 ± 73 mmHg vs. 128 ± 54 mmHg, $P < 0.0001$). PaCO_2 was also significantly lower after RM was applied ($P = 0.038$, vs. NPPV group). Compared with the CON group, NPPV also significantly reduced Pa-ETCO_2 (7 ± 2 mmHg vs. 5 ± 3 mmHg, $P < 0.001$; table 2). RM further lowered Pa-ETCO_2 (3 ± 2 mmHg, $P = 0.024$ vs. NPPV group).

The main ventilation variables and respiratory mechanics are shown in table 3. There was no significant difference in the measured respiratory rate and V_{Te} between the groups ($P = 0.15$ and $P = 0.94$, respectively). Compared with the CON group, static elastance of the respiratory system was significantly lower in both the NPPV and NPPV+RM groups (both P values less than 0.0001), with a significant difference ($P = 0.023$) between the NPPV and NPPV+RM groups. RM-induced reduction in static elastance of the respiratory system was 21%. In contrast with NPPV alone, airway pressure was significantly lower in the NPPV+RM group than in the CON group (table 3). Compared with NPPV alone, plateau end-inspiratory airway pressure was also significantly lower in the NPPV+RM group ($P = 0.012$).

Irrespective of the group, compared with conventional preoxygenation, EELV was significantly higher in the NPPV and NPPV+RM groups after mechanical ventilation was induced (both $P < 0.001$, fig. 2). Compared with NPPV alone, NPPV+RM further increased EELV ($P = 0.03$). Compared with the CON and NPPV groups, in which changes in EELV (with EELV after intubation was performed as a reference) were not significant ($P = 0.49$ and $P = 0.71$, respectively), RM-induced change in EELV was 32% in the NPPV+RM group ($P = 0.028$). When considering the variation of EELV after RM was applied, two populations were identified: patients who responded to NPPV alone with no additional beneficial effect of RM (patients with the lowest variation, $n = 10$), and patients who did not respond to NPPV, but with larger effects of RM on EELV changes ($n = 12$). There was a statistical difference ($P = 0.004$) on EELV changes between the two populations.

Oxygenation, End-expiratory Lung Volume, and Static Elastance of the Respiratory System

A weak but significant correlation was found between PaO_2 obtained 5 min after the onset of mechanical ventilation and EELV ($R^2 = 0.41$, $P < 0.001$). EELV obtained after induction of mechanical ventilation was correlated

Table 2. Gas Exchange and Hemodynamics over the Different Steps of the Experiment

	CON Group (n = 22)	NPPV Group (n = 22)	NPPV+RM Group (n = 22)	Overall Group Effect
Awake				
Paco ₂ , mmHg	37 ± 4	37 ± 3	38 ± 3	P = 0.33
pHa	7.43 [7.42–7.46]	7.43 [7.41–7.45]	7.43 [7.41–7.45]	P = 0.63
MAP, mmHg	91 ± 11	97 ± 9	96 ± 9	P = 0.08
HR, beats per min ⁻¹	82 ± 12	80 ± 14	81 ± 13	P = 0.90
After preoxygenation				
Paco ₂ , mmHg	32 ± 2	29 ± 4*	30 ± 6*	P = 0.008
pHa	7.44 [7.44–7.45]	7.50 [7.47–7.53]*	7.50 [7.47–7.54]*	P < 0.01
MAP, mmHg	94 ± 9	93 ± 10	95 ± 11	P = 0.94
HR, beats per min ⁻¹	85 ± 11	82 ± 13	84 ± 14	P = 0.73
After ETI				
Paco ₂ , mmHg	45 ± 4	43 ± 6	43 ± 5	P = 0.40
pHa	7.40 [7.37–7.41]	7.39 [7.35–7.41]	7.39 [7.37–7.42]	P = 0.55
MAP, mmHg	73 ± 7	76 ± 15	75 ± 14	P = 0.67
HR, beats per min ⁻¹	80 ± 14	78 ± 15	79 ± 15	P = 0.95
5 min after MV				
Paco ₂ , mmHg	44 ± 2	42 ± 3	40 ± 3*†	P = 0.0001
pHa	7.40 [7.38–7.41]	7.45 [7.43–7.47]	7.46 [7.42–7.49]	P = 0.19
Pa-ETCO ₂ , mmHg	7 ± 2	5 ± 3*	3 ± 2*†	P < 0.001
MAP, mmHg	68 ± 7	73 ± 12	70 ± 13	P = 0.36
HR, beats per min ⁻¹	78 ± 14	82 ± 19	74 ± 18	P = 0.54

Data are presented as mean ± SD or median [interquartile range] as appropriate (see also Materials and Method, Statistical section).

* Significant vs. CON group, P < 0.05. † Significant vs. NPPV group, P < 0.05.

CON = conventional preoxygenation; NPPV = noninvasive positive pressure ventilation; NPPV+RM = NPPV and recruitment maneuver; RM = recruitment maneuver; ETI = endotracheal intubation; HR = heart rate; MAP = mean arterial pressure; MV = mechanical ventilation; Paco₂ = arterial partial pressure of carbon dioxide; Pa-ETCO₂ = arterial-to-end-tidal partial pressure of carbon dioxide; pHa = arterial pH.

with static elastance of the respiratory system (R² = 0.40, P < 0.001).

Discussion

The current study demonstrates that, in morbidly obese patients, NPPV followed by early RM improves arterial oxygenation after initiation of mechanical ventilation compared with NPPV alone; oxygenation with NPPV is improved in comparison with conventional preoxygenation; and NPPV followed by RM further improves EELV and respiratory mechanics compared with NPPV alone.

The use of NPPV during induction of general anesthesia in obese patients has seldom been reported in the operating room, mainly because PSV has only recently been available on anesthesia ventilators.²⁵ Coussa *et al.*⁸ showed that, in 18 obese patients, the application of a 5-min trial of continuous positive airway pressure during preoxygenation improved PaO₂ after tracheal intubation compared with control patients. However, differences in PEEP levels applied during mechanical ventilation in the continuous positive airway pressure (10 cm H₂O of PEEP) and control (no PEEP) groups could also explain this difference in oxygenation. In a recent study, Delay *et al.*¹² found no significant increase in

Table 3. Respiratory Mechanics

	CON Group (n = 22)	NPPV Group (n = 22)	NPPV+RM Group (n = 22)	Overall Group Effect
P _{max} , cm H ₂ O	35 [32–39]	32 [29–36]	29 [28–34]*	P = 0.03
P _{plat} , cm H ₂ O	26 [25–28]	20 [18–22]*	16 [16–18]*†	P < 0.001
E _{rs} , cm H ₂ O ml ⁻¹	34 [27–39]	20 [17–25]*	13 [11–20]*†	P < 0.001
VTe, ml	485 [430–546]	458 [420–550]	475 [425–550]	P = 0.94
RR, c min ⁻¹	20 [18–20]	20 [20–20]	20 [20–21]	P = 0.15

Data are presented as median [interquartile range].

* Significant vs. CON, P < 0.05. † Significant vs. NPPV, P < 0.05.

CON = conventional preoxygenation; NPPV = noninvasive positive pressure ventilation; NPPV+RM = NPPV and recruitment maneuver; RM = recruitment maneuver; E_{rs} = static elastance of the respiratory system; P_{max} = peak airway pressure; P_{plat} = end-inspiratory plateau airway pressure; RR = respiratory rate; VTe = expiratory tidal volume.

PaO₂ with NPPV compared with conventional preoxygenation. The use of the beach chair position instead of the supine position in the current study could explain the different beneficial effects of NPPV. Previous studies clearly showed that head-up positioning improves oxygenation and lung function during preoxygenation.^{26,27} Furthermore, the beach chair position and PEEP were shown to individually improve lung volumes in obese patients.¹⁷ We measured both oxygenation and lung volume directly and found that, despite the same level of PEEP applied after intubation, NPPV significantly improved oxygenation before intubation and after initiation of mechanical ventilation compared with 5 min of 100% oxygen spontaneous breathing. The clinical relevance of a benefit of NPPV on oxygenation is negligible compared with conventional oxygen administration. However, these effects might be different in sicker patients with higher amounts of alveolar collapse at the induction of anesthesia.²⁸ In addition, in the control group, a small number of patients reached levels of oxygenation near safety threshold values, even 5 min after tracheal intubation, the primary evaluation criteria in this study (fig. 2).

When considering the SD for PaO₂ in the current study, it is likely that NPPV introduced higher variability in oxygenation because of possible different effects among patients. Although NPPV should increase both EELV and oxygenation by recruiting collapsed alveoli,¹¹ its beneficial effect might not be associated with an increase in oxygenation: alveolar recruitment, an anatomic phenomenon that exhibits as restored aeration on CT, may not coincide with functional recruitment as defined by improved gas exchange.²⁹

Few data are available regarding the effects of NPPV on EELV. In acute respiratory failure, NPPV was suspected to improve oxygenation by unloading respiratory muscle and recruiting alveoli in parallel with an increase in lung volume.³⁰ We found that 5 min of NPPV was associated with an average increase in EELV of approximately 700 ml compared with conventional preoxygenation. EELV measurements, which reflect the amount of gas present in the lungs, were obtained using a modified nitrogen wash-out/wash-in method²¹ that was found to correlate well with EELV as measured by CT²² and with changes in lung aeration and consolidation on CT scans.³¹ Because body habitus might affect EELV variability, EELV measurements were standardized for the predicted body weight.²³ In most clinical studies that address the EELV measurements, data were presented without the measured EELV values for individual patients.^{32–34} We found comparable EELV data at comparable PEEP levels, as previously reported by Bikker *et al.*²³ It can be postulated that the improvement in EELV observed after preoxygenation using NPPV could be attributed to the recruitment of collapsed alveoli with oxygen administration,⁸ thereby allowing an increase in oxygen reserves.¹¹

Initiation of mechanical ventilation on a collapsed lung may cause or worsen lung injury, leading to ventilator-induced lung injury.³⁵ Although the beneficial effect on PaO₂ remained 5 min after the onset of mechanical ventilation compared with conventional preoxygenation, suggesting a residual effect of NPPV in recruiting alveoli, concerns have been expressed regarding possible lung recollapse after discontinuation of NPPV.³⁶ Coussa *et al.*⁸ showed that after intubation was performed, areas of atelectasis on CT, although more pronounced in the control group, increased after discontinuation of continuous positive airway pressure. We found that RM performed early after intubation improved arterial oxygenation after initiation of mechanical ventilation. In comparison with NPPV alone, we also found significant improvements in secondary evaluation criteria, especially EELV and respiratory mechanics. Previous studies have shown a beneficial effect of RM on lung collapse in both anesthetized patients and those with acute respiratory distress syndrome,^{15,37} whereas PEEP alone is insufficient to reopen the nonventilated lung.¹⁵ Reinius *et al.*¹⁵ found that after induction of anesthesia, PEEP alone increased the normally aerated lung fraction in parallel with a reduction of poorly aerated lung tissue while atelectasis remained unchanged. These authors also found that the increase in normally aerated lung tissue was not accompanied by an increase in oxygenation.¹⁵ This finding is in agreement with our results, which indicate that the RM followed by PEEP significantly increased PaO₂ in contrast to NPPV followed by PEEP alone.

Concerns might be raised regarding the effectiveness of a single RM. Previous data showed that when RM was applied without PEEP, the effect was of short duration in morbidly obese patients.^{15,16} Conversely, a single RM followed by PEEP reduced atelectasis and improved oxygenation significantly for at least 20 min in obese patients,¹⁵ whereas PEEP alone or RM alone did not.¹⁵

The study design did not allow us to determine the real extent of alveolar recruitment with the RM. The increase in EELV (approximately 30%; fig. 2) could be associated with increased alveolar size and/or alveolar recruitment. The reduction in Pa-ETCO₂, a suitable indicator of dead space,²⁰ may suggest that recruitment played a relevant role. An increase in ventilated lung regions due to re-aeration with alveolar recruitment (higher regional alveolar ventilation) decreased dead space variables,²⁰ whereas an increase in dead space variables would have been expected if no recruitment (or overdistension) had occurred. Although 10 cm H₂O of PEEP was applied after intubation in each patient, these results suggest that possible recruitable lung regions remained even after NPPV was used. Changes of EELV in the NPPV and NPPV+RM groups were variable among subjects. This finding suggests that in some patients EELV was likely associated with recruitment, whereas it was just an increase in alveolar volume in others. Furthermore, elastance

of the respiratory system was different among patients, likely affecting the effects of RM and PEEP on EELV, as previously suggested.³⁸

There are several concerns about the potential negative effects of the RM. The possibility of lung overdistension with RM seems unlikely as indicated by a reduction in elastance of the respiratory system in parallel with the increase in EELV, as suggested previously.³⁹ It has been shown that a pressure of 55 cm H₂O during the RM was effective at reopening atelectasis after induction of anesthesia in morbidly obese patients.¹⁵ Nevertheless, because of the beneficial effects of NPPV before tracheal intubation, it is estimated that a pressure of 40 cm H₂O would be sufficient while minimizing possible negative effects in terms of barotrauma. Furthermore, it has been suggested that an RM applied to a substrate with low potential for alveolar recruitment could worsen gas exchange by a diversion of blood flow toward lesser aerated regions.⁴⁰ However, arterial oxygenation is a complex physiologic parameter affected by several factors such as the extent of lung aeration, regional pulmonary flow, cardiac index, and oxygen delivery whereas recruitment *per se* depends exclusively on penetration of gas into poorly aerated and nonaerated lung regions.²⁸ We found that in contrast with NPPV alone, oxygenation was increased by approximately 50% after the RM was applied, which was consistent with previous studies in morbidly obese patients.¹⁵ Excessive pressure may cause transient hemodynamic instability, especially in hypovolemic patients.⁴¹ Special care was therefore taken to ensure that our patients were normovolemic throughout the study, and none of the patients showed hemodynamic instability during the RM.

Our study has several limitations. (1) The study investigators but not the anesthesiologists were blinded to treatment assignment to ensure patient safety. In addition, the anesthetists were all experienced in the management of morbidly obese patients. Although the study investigators did not take part in clinical procedures, this may have influenced the results. (2) Our group recently found that the RM alone may be sufficient to improve oxygenation after intubation of hypoxemic patients in the intensive care unit.²⁸ Nevertheless, several studies have shown that in morbidly obese patients NPPV enhanced the conditions of intubation^{7,9,12} but not respiratory function after intubation. It is suggested that NPPV is used to improve the safety of intubation and RM is used to further improve respiratory function. (3) Only EELV was measured, whereas its changes do not necessarily indicate atelectasis formation. Although CT is considered the technique of choice because it is accurate and precise, it is not easy to perform at the patient's bedside. Moreover, EELV measurement using the modified nitrogen wash-in/wash-out method was found to correlate well with EELV as measured by CT.²² Some limitations of EELV technique, such as the presence of severe airflow obstruction caused by chronic obstructive pulmonary disease or PEEP more than 20 cm H₂O,

have been reported.²³ (4) Opposite trends for subgroups of patients can be individualized with NPPV. Unfortunately, our data do not allow us to speculate about the possible determinants for responsiveness to NPPV. Nevertheless, previous data emphasized the variable and inconstant effect of NPPV among patients.^{11,12} (5) The investigation period was relatively short and limited to the first minutes after initiation of mechanical ventilation. However, previous data demonstrated that RM followed by PEEP could provide sustained effects.¹⁵ (6) Tolerance of oxygen administration was good, and preoxygenation was never stopped prematurely because of claustrophobia. Possible side effects such as gastric distension were not evaluated in detail. Although oxygen administration with PEEP alone did not increase the volume of gastric gas,¹² the PSV component may promote gastric distension.¹² We used a pressure-limited mode that allows control of insufflation pressures (PSV and PEEP). Insufflation pressures of no more than 20 cm H₂O are recommended to limit pulmonary aspiration.¹² In our study, no patient received insufflation pressures of more than 18 cm H₂O. (7) Anesthesia induction was performed using 100% oxygen, which greatly influenced the formation of atelectasis.⁴² However, although FIO₂ reduction will prevent atelectasis, this is not recommended in morbidly obese patients because of a decrease in nonhypoxic apnea duration with a reduced margin of safety.⁴²

In conclusion, 5 min of NPPV using PSV and PEEP ensures better oxygenation and greater EELV early after tracheal intubation compared with conventional preoxygenation. Furthermore, NPPV combined with RM further improves oxygenation and respiratory functions after tracheal intubation compared with NPPV alone. Additional studies are warranted to demonstrate longer lasting effects of such a strategy.

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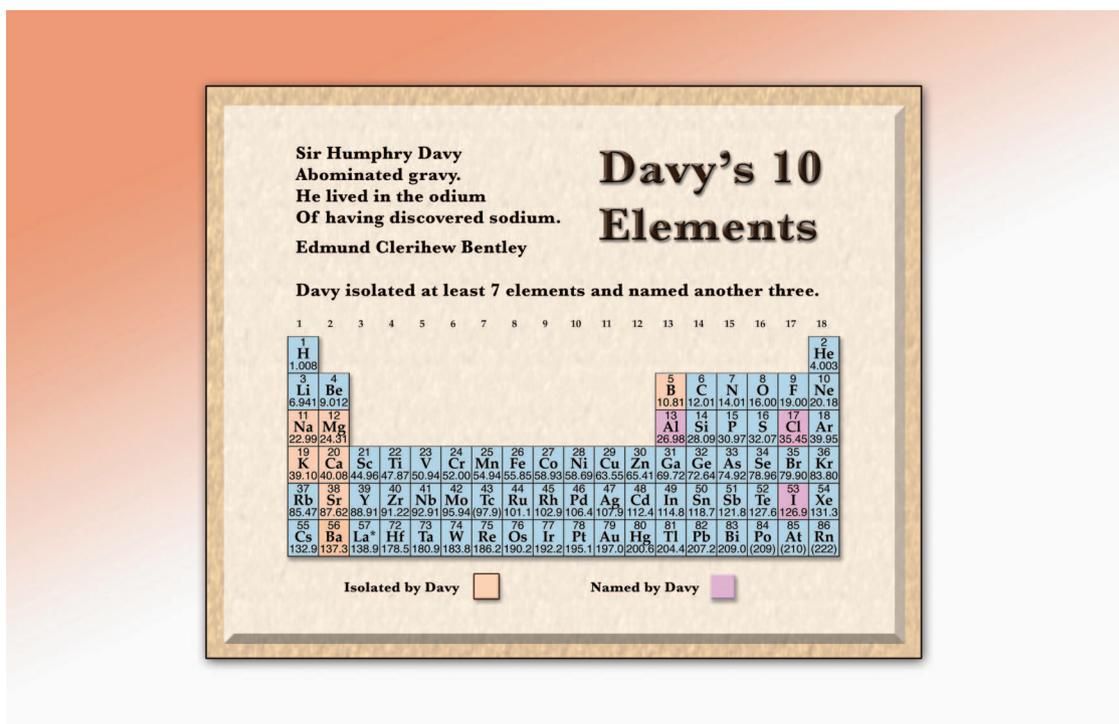
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ANESTHESIOLOGY REFLECTIONS

Davy's Ease Discovering Elements and Difficulty Naming Alumi(n)(i)um



Humphry Davy isolated or named more chemical elements than any chemist before or since. From electrolysis and other techniques, Davy had good fortune in discovering “new” elements. However, Davy had bad luck determining a name for, of course, the 13th chemical element. In 1807 Davy named that element “Alumium”; in 1812 he renamed it “Aluminum.” Later that year, editors encouraged Davy to adopt “Aluminium,” the spelling and pronunciation which most of the world uses to this day. Unfortunately for the United States, in 1925 “isolationists” in the American Chemical Society resurrected Davy’s second name for the 13th element, “Aluminum.” (Copyright © the American Society of Anesthesiologists, Inc. This image also appears in the *Anesthesiology Reflections* online collection available at www.anesthesiology.org.)

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