The Effectiveness of Noninvasive Positive Pressure Ventilation to Enhance Preoxygenation in Morbidly Obese Patients: A Randomized Controlled Study

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BACKGROUND: Noninvasive positive-pressure ventilation (NPPV) with pressure support-ventilation and positive end-expiratory pressure are effective in providing oxygenation during intubation in hypoxemic patients. We hypothesized administration of oxygen (O_2) using NPPV would more rapidly increase the end-tidal O_2 concentration (ETo_2) than preoxygenation using spontaneous ventilation (SV) in morbidly obese patients.

METHODS: Twenty-eight morbidly obese patients were enrolled in this prospective randomized study. Administration of O_2 for 5 min was performed either with SV group or with NPPV (pressure support = 8 cm H_2O, positive end-expiratory pressure = 6 cm H_2O) (NPPV group). ETo_2 was measured using the anesthesia breathing circuit, and is expressed as a fraction of atmospheric concentration. The primary end-point was the number of patients with an ETo_2 >95% at the end of O_2 administration. Secondary end-points included the time to reach the maximal ETo_2 and the ETo_2 at the conclusion of O_2 administration.

RESULTS: A larger proportion of patients achieved a 95% ETo_2 at 5 min with NPPV than SV (13/14 vs 7/14, $P = 0.01$). The time to reach the maximal ETo_2 was significantly less in the NPPV than in the SV group (185 ± 46 vs 222 ± 42 s, $P = 0.02$). The mean ETo_2 at the conclusion of O_2 administration was larger in the NPPV group than the SV group (96.9 ± 1.3 vs 94.1 ± 2.0%, $P < 0.001$). A modest, although significant, increase in gastric distension was observed in the NPPV group. No adverse effects were observed in either group.

CONCLUSION: Administration of O_2 via a facemask with NPPV in the operating room is safe, feasible, and efficient in morbidly obese patients. In this population NPPV provides a more rapid O_2 administration, achieving a higher ETo_2.

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Noninvasive positive-pressure ventilation (NPPV) can be delivered via a facemask using a combination of pressure support ventilation and positive end-expiratory pressure (PEEP). NPPV has been increasingly used in the intensive care unit to avoid endotracheal intubation in patients with acute respiratory failure.[1–3] Pressure support is a ventilatory mode in which the patient's spontaneous inspiratory effort triggers the ventilator to provide a variable flow of gas that increases until airway pressure reaches a selected level. Thus, during each spontaneous inspiration, the patient receives a pressure-supported breath. Once the selected airway pressure is achieved, the patient can continue to breathe until his or her inspiratory flow rate decreases below a threshold level (usually a fixed level at 25% of the peak flow). Thus, the tidal volume achieved depends on the patient's own effort added to that of the ventilator's inspiratory pressure, which can be capped at a relatively low level.[4] NPPV increases in functional residual capacity (FRC) by recruiting collapsed alveoli, thereby increasing the oxygen (O_2) reserves held within the lungs.[5]

We reported that NPPV was more effective for oxygenation than spontaneous ventilation (SV) during tracheal intubation in hypoxemic critically ill patients.[5] The significant improvement in oxygenation with NPPV resulted from both the high delivered O_2 concentration and the increase in FRC. Administration of 100% O_2 before the induction of general anesthesia is intended to enhance O_2 reserves[6] to delay hypoxemia. Adequate O_2 administration is particularly critical if difficult airway management is anticipated.[7] Obese patients may present with difficult airways, and are more likely to desaturate than lean subjects.[8–12] Maximal O_2 administration is a major component of safety for these patients during induction of general anesthesia.[7,13]

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During O_2 administration, high values of end-tidal O_2 concentration (ETo_2, expressed as a percent of atmospheric pressure) indicate that the pulmonary volume at FRC is saturated with O_2. Values of ETo_2 above 90% define denitrogenation and should be obtained when difficulties for mask ventilation or intubation are anticipated.[7,13] The usual technique of O_2 administration is for the patient to spontaneously breathe 100% O_2 by mask for 3–5 min. This approach may not provide an ETo_2 above 90% in some obese patients.[7,14,15] Rusca et al.[16] showed that PEEP applied during induction prevents atelectasis and increases the duration of nonhypoxic apnea in both nonobese and obese patients.[17,18] However, the use of pressure support ventilation in the operating room has seldom been reported, whether during general anesthesia[19–21] or for an O_2 administration procedure before general anesthesia, because the pressure support mode has only recently been available in anesthesia ventilators. The latest generation of anesthesia ventilators provide a pressure support ventilation mode with a standard closed anesthesia system. We reported that the trigger sensitivity and the ability to meet inspiratory flow during pressure-supported breaths of contemporary anesthesia ventilators are comparable to performances of modern intensive care unit ventilators.[22]

Our hypothesis was that O_2 administration using NPPV would provide a more rapid increase in ETo_2 than O_2 administration consisting of 5 min of SV of 100% O_2 via facemask in morbidly obese patients during induction of a general anesthesia.

METHODS

The study was approved by institutional review board (CCPPRB, University Teaching Hospital of Montpellier). Signed informed patient consent was obtained at the time of inclusion in the study. Inclusion criteria were age older 18 yrs, body mass index (BMI) above 40 kg/m^2, and planned abdominal surgery under general anesthesia. The exclusion criteria were previous bariatric surgery, emergency procedures, digestive endoscopy not immediately followed by surgery, coronary artery or cerebrovascular disease, intracranial hypertension, epilepsy, history of difficult tracheal intubation, contraindication for the use of any of the protocol drugs, incomprehension of the protocol, and consent refusal.

Patients fasted for 6 h and received oral premedication 2 h before surgery with hydroxyzine and cimetidine in sodium citrate. The study was conducted in the operating room, with the patient lying supine, after placement of a five lead electrocardiogram, a pulse oximeter (M11914®, Philips Medizinesystems™, Boeblingen, Germany), and IV infusion of a crystalloid solution. A 20-gauge catheter (Leader-Cath®, Vygon™, Ecouen, France) was inserted in a radial artery under local anesthesia for continuous monitoring of arterial blood pressure (BP) and arterial blood gas (ABG) measurement.

Preoxygenation consisted of a single 5-min trial of SV (SV group) or NPPV (NPPV group), depending on randomization assigned from a sealed envelope in the operating room. The main circuit of the Primus® anesthesia ventilator (Dräger™, Lübbeck, Germany), including a 3-L balloon, was used in both groups. In both groups, masks surrounding the nose and mouth (Airvie®; Peters™, Bobigny, France) were tightly held on the face to prevent air leaks and entrainment of room air. In the SV group, the fresh gas flow was set at 18 L/min, which is the highest the ventilator could deliver, and the adjustable pressure-limiting valve was fully opened. Patients were asked to breathe at their usual tidal volume. In the NPPV group, pressure support and PEEP were set at 6 and 4 cm H_2O respectively during the first 20 s, after which PEEP was increased to 6 cm H_2O and pressure support was increased to 8–10 cm H_2O to attain an expiratory tidal volume (VTe) of at least 8 mL/kg of ideal body weight. The inspiratory trigger sensitivity was set at −2 L/min. The inspired concentration of O_2 (Fio_2) was set at 100%.

At the end of the 5 min administration of O_2, general anesthesia was induced with IV sodium thiopental and succinylcholine. The mask was maintained until the onset of apnea, defined by the loss of the expiratory flow curve on the monitor. No manual ventilation was attempted before laryngoscopy, which was performed after muscle fasciculation. After intubation, anesthesia was maintained with IV remifentanil (0.5 $\mu g/kg$ for 2 min followed by a continuous infusion of 0.25 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and IV cisatracurium (0.2 mg/kg), both based on ideal body weight. After intubation, the endotracheal tube was separated from the breathing circuit, and left open to room air, without manual ventilation. Apnea was maintained until arterial O_2 saturation (Spo_2) decreased to 95%. Patients were then connected to the breathing circuit, and assisted-controlled ventilation was started with the following settings: VTe = 8 mL/kg, based on ideal body weight, respiratory rate (RR) = 14 cycles/min, and PEEP = 5 cm H_2O. Anesthesia was maintained with desflurane in an air-O_2 mixture and a continuous infusion of IV remifentanil (0.15 $\mu g \cdot kg^{-1} \cdot min^{-1}$, based on ideal body weight). Intubation difficulty was evaluated with the intubation difficulty score.[23]

During O_2 administration, Fio_2, ETo_2, inspired and expired concentrations of carbon dioxide ($Fico_2$ and $ETco_2$, respectively) 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. Tidal volumes were calculated by the ventilator's software by integrating the flow curve during expiration. RR was measured every minute and minute ventilation (MV) was calculated every minute as the product of RR and the mean tidal volume for the same minute. The duration of O_2 administration was defined as the sum of 5 min of administration of O_2 and the time between

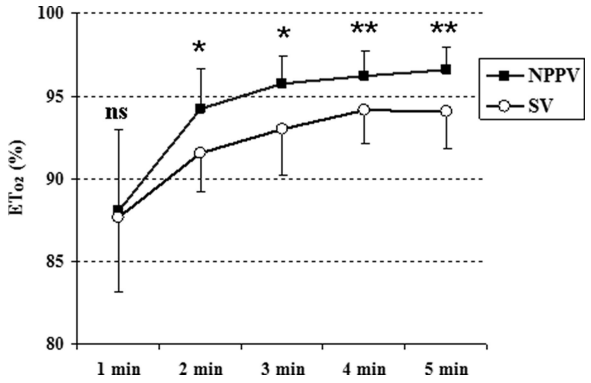

Figure 1. Variation in mean end-tidal oxygen concentration (ETo_2) during preoxygenation. After 2 min of preoxygenation and during all preoxygenation procedures, ETo_2 values were significantly higher in the noninvasive positive-pressure ventilation (NPPV) group (full square) than in the spontaneous ventilation (SV) group (empty circles). Data are mean ± SD. ns = not significant; *$P < 0.05$, **$P < 0.01$, comparison between the two groups at the same point.

induction and the onset of apnea. Time to desaturate was defined by the time between onset of apnea and a Spo_2 of 95%.

ABG was immediately analyzed (Omni-S®, Laboratoires Roche™, Neuilly, France) before O_2 administration (i.e., while on room air), at the end of O_2 administration, when Spo_2 reached 95% during apnea, and after 5 min of assisted-controlled ventilation (Fig. 1).

Gastric distension was evaluated by the surgeon blinded for the O_2 administration method at the beginning of laparoscopy and before insertion of a gastric tube. Distension was rated with a numerical scale from 0 (no distension) to 100 (maximal distension). Tolerance of O_2 administration was rated by the patient 6 h after the surgery, with a visual analogical scale, graded from 0 (poor tolerance) to 100 mm (good tolerance).

The primary end-point was the number of patients with an ETo_2 >95% at the end of O_2 administration. The study was designed with 90% power to detect a 90% relative difference between groups (with a significance level of 5%). Assuming an ETo_2 >95% at the end of O_2 administration for 50% of the patients in the SV group, we planned to enroll 30 patients. The secondary end-points were the time to reach the maximal ETo_2 and the mean ETo_2 at 5 min.

Data are expressed as mean ± SD or number (%). Categorical data were compared with χ^2 test. Continuous data were compared between groups with Mann-Whitney test or Wilcoxon's ranked sum test inside each group. Linear regression analysis was used to assess any relation between BMI and time to desaturate. The level of significance was set at $P = 0.05$. Statistical comparisons were performed with SAS©/STAT software version 8.1 (SAS Institute, Cary, NC).

RESULTS

We screened 102 patients between January and December 2005. Sixty-five were excluded based on our exclusion criteria, nine refused to give consent, and 28 patients were included in the study. Included and nonincluded patients were comparable for age, sex, weight, and BMI (data not shown). All included patients were scheduled for laparoscopic bariatric surgery, except one patient in the SV group, who was scheduled for a lipectomy. Demographic data, duration of O_2 administration, intubation difficulty score, and drug amounts were comparable between groups (Table 1). All patients underwent laryngoscopy and tracheal intubation before any desaturation, except one patient in the SV group who required intubation using a Fast-trach® device. For this patient, time to desaturation and minimal Spo_2 could not be recorded because of a transient dysfunction in the oximeter. In all patients, the measured Fio_2 was always 100%, and the measured $Fico_2$ was always 0. All patients in both groups reached an ETo_2 of at least 90%.

Figure 1 shows the ETo_2 in each group during O_2 administration. ETo_2 increased significantly between the second and the fifth minute in both groups. More patients in the NPPV group had an ETo_2 value ≥95% at the conclusion of O_2 administration than in the SV group (50% vs 93%, $P = 0.01$). The time to reach the maximal ETo_2 was significantly less in the NPPV group than in the SV group (185.3 ± 46.1 vs 221.9 ± 41.5 s, $P = 0.02$). Patients in the NPPV group reached higher ETo_2 at the end of O_2 administration than patients in the SV group (96.9% ± 1.3% vs 94.1% ± 2.0%, $P < 0.001$) (Fig. 1) Mean $ETco_2$ values were always comparable between groups at all time points during O_2 administration, ranging between 35 and 38 mm Hg.

We were only able to place arterial catheters in nine patients in each group. ABGs were collected in these patients, and are presented in Table 2. Values of Pao_2, $Paco_2$, and pH before O_2 administration were comparable between groups. Pao_2 values increased significantly during O_2 administration for both groups. The trend toward higher Pao_2 values in the NPPV group did not reach statistical significance ($P = 0.12$ between groups). $Paco_2$ values of both groups were comparable before O_2 administration. During O_2 administration $Paco_2$ increased significantly in the SV group (37.9 ± 4.8 vs 41.7 ± 5.2 mm Hg, $P = 0.007$) but not in the NPPV group.

Figure 2 shows the ventilation variables in each group. Mean measured RR increased significantly more in the SV than the NPPV group, whereas VTe and MV were significantly higher in the NPPV group at the end of O_2 administration.

Time to desaturation was similar in both groups (154 ± 35 vs 161 ± 35 s, $P = 0.48$) (Fig. 3). Systolic BP and heart rate were comparable between groups (Fig.

Table 1. Demographic and Anesthetic Data

	SV group (n = 14)	NPPV group (n = 14)	P
Age (yrs)	42.9 ± 11.6	36.6 ± 11.7	0.21
Sex (men/women) (n)	2/12	3/11	0.99
Weight (kg)	142.5 ± 42.9	131.2 ± 23.4	0.95
Height (cm)	164.5 ± 10.1	166.6 ± 8.5	0.68
BMI (kg/m^2)	52.3 ± 13.7	47.1 ± 6.2	0.58
Ideal body weight (kg)	56.6 ± 10.3	58.7 ± 9.1	0.66
Current smokers	2	2	0.99
Sleep apnea syndrome	4	6	0.69
Nocturnal respiratory assistance	2	5	0.38
Hemoglobin rate (g/dL)	13.7 ± 1.9	12.6 ± 1.2	0.27
Hemoglobin saturation on room air before preoxygenation (%)	96.6 ± 1.8	97.3 ± 0.8	0.18
IDS score	2.4 ± 3.1	2.6 ± 2.5	0.69
Minimal hemoglobin saturation after period of apnea (%)	88.6 ± 2.9	86.9 ± 5.0	0.53
Thiopental (mg/kg of RBW)	6.9 ± 0.7	7.0 ± 0.3	0.87
Succinylcholine (mg/kg of RBW)	1.5 ± 0.1	1.5 ± 0.1	0.93
Cisatracurium (mg/kg of IBW)	0.20 ± 0.01	0.21 ± 0.02	0.82
Expiratory fraction of desflurane at 5 minutes of ACV (%)	4.2 ± 2.4	3.6 ± 1.4	0.48

Data are number (%) or mean ± SD.

NPPV = noninvasive positive-pressure ventilation; SV = spontaneous ventilation; RBW = real body weight; IBW = ideal body weight; ACV = assisted-controlled ventilation; BMI = body mass index; IDS = intubation difficulty score.

Table 2. Comparison of Blood Gas Results for Patients Randomly Assigned with the SV and NPPV Groups

	Before preoxygenation	After 5 min of preoxygenation	At Spo_2 = 95%	After 5 min of ACV
Pao_2 (mm Hg)				
SV	86.0 ± 9.3	454.3 ± 93.4	66.1 ± 7.8	130.8 ± 44.3
NPPV	87.6 ± 11.8	507.1 ± 81.0	69.2 ± 11.1	140.6 ± 47.3
P	0.93	0.12	0.56	0.67
Sao_2 (%)				
SV	96.4 ± 1.5	99.9 ± 0.3	89.6 ± 3.4	97.6 ± 3.2
NPPV	96.7 ± 1.3	99.9 ± 0.3	90.2 ± 4.9	97.0 ± 4.0
P	0.93	0.99	0.49	0.70
$Paco_2$ (mm Hg)				
SV	37.9 ± 4.8	41.7 ± 5.2	55.3 ± 7.6	42.5 ± 5.0
NPPV	39.4 ± 5.6	38.2 ± 7.0	55.3 ± 7.4	45.8 ± 7.7
P	0.69	0.17	0.69	0.26
pH				
SV	7.42 ± 0.04	7.40 ± 0.03	7.33 ± 0.04	7.39 ± 0.04
NPPV	7.41 ± 0.02	7.43 ± 0.04	7.32 ± 0.03	7.37 ± 0.03
P	0.44	0.08	0.77	0.29

Data were obtained from arterial blood sample before preoxygenation in nine patients of each group. Data are presented as mean ± SD.

After 5 min of assisted controlled ventilation (ACV), Fio_2 values were 0.68 ± 0.10 in SV group and 0.60 ± 0.08 in NPPV group (difference was not significant).

NPPV = noninvasive positive-pressure ventilation; SV = spontaneous ventilation; Pao_2 = arterial oxygen tension; Sao_2 = arterial oxygen saturation; $Paco_2$ = arterial carbon dioxide tension.

4). Tolerance of O_2 administration was similar between groups (82.3 ± 24.4 vs 90.5 ± 8.9 mm, P = 0.81). Preoxygenation was never stopped prematurely. Transient air leaks were identified in two patients upon initiation of NPPV, and were eliminated by adjusting the position of the mask.

Gastric distension evaluation was not performed in four patients in the SV group because of extraperitoneal surgery (lipectomy, one patient), esogastric fibroscopy with gas insufflation between intubation and onset of surgery (two patients), or need to ventilate manually before intubation (one patient). A modest, although significant, increase in gastric distension was measured in the NPPV group compared with the SV group (3.8 ± 5.6 vs 17.6 ± 13.5, P = 0.01). No episodes of nausea, regurgitation, or vomiting were observed. Air was always suctioned from the stomach, except in one SV group patient for whom surgery was postponed because of small bowel distension after ventilation for desaturation and difficult intubation.

DISCUSSION

Main Results

Our results show that 5 min of NPPV with moderate levels of pressure support and PEEP provides a higher ETo_2 than SV, and accelerates the increase in ETo_2 during O_2 administration. Several authors have

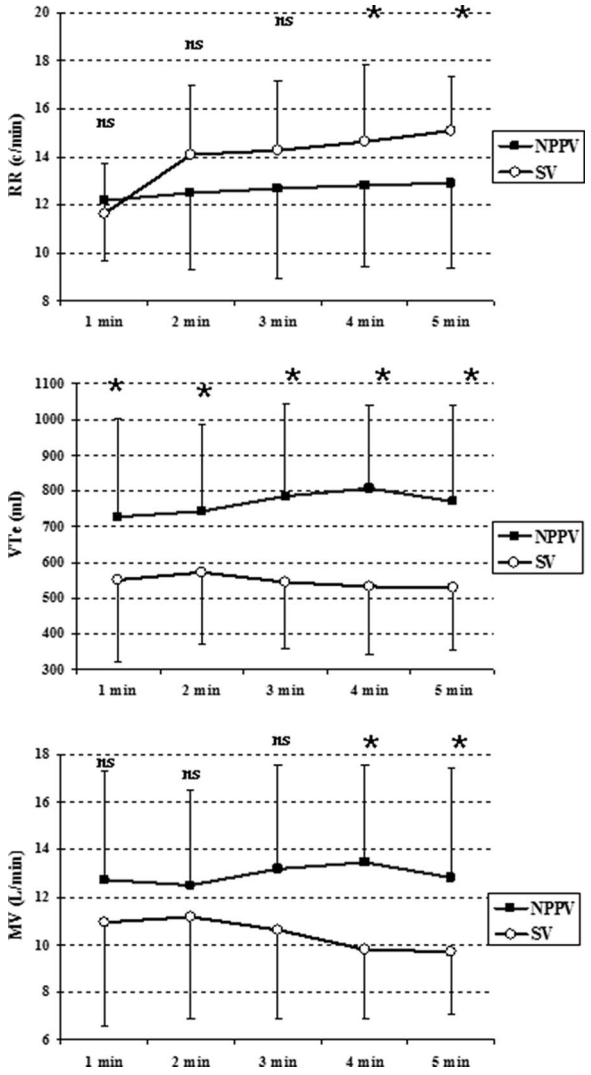

Figure 2. Comparison of breathing pattern for patients randomly assigned to the spontaneous ventilation (SV) and noninvasive positive-pressure ventilation (NPPV) groups. Data are mean ± SD. ns = not significant; $*P < 0.05$, comparison between the two groups at the same point. RR = respiratory rate; VTe = expiratory tidal volume; MV = minute ventilation.

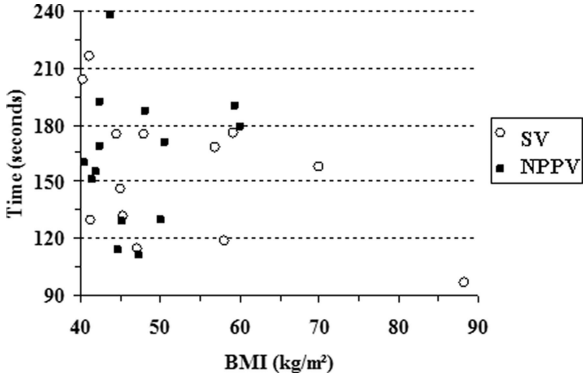

Figure 3. Individual values for time to desaturate from the onset of apnea to Spo_2 reached 95%. Values were obtained from 13 patients in the spontaneous ventilation (SV) group and from 14 patients in the noninvasive positive-pressure ventilation (NPPV) group (see Results section for details). BMI = body mass index.

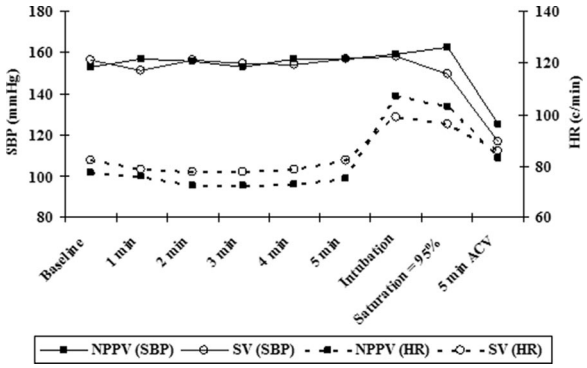

Figure 4. Variation in hemodynamic variables during the study period. Data are presented as mean for heart rate (HR) and systolic blood pressure (SBP). No significant difference was observed between noninvasive positive-pressure ventilation (NPPV) and spontaneous ventilation (SV) groups for all variables during all procedures. ACV = assisted-controlled ventilation.

suggested that ETo_2 is an appropriate surrogate marker for denitrogenation.[24–26] Although ETo_2 is a common marker for O_2 administration efficiency, study comparisons are difficult because of differences in O_2 flows, breathing patterns, and durations of O_2 administration trials. In lean adults, SV with deep breaths for 1 or 2 min provides a higher ETo_2 than an equal duration of tidal volume breathing (TVB).[15,27] In morbidly obese adults[14] and pregnant women,[28] 1 min of deep breaths provides higher ETo_2 levels than 1 min of TVB. In previous studies, 1 or 2 min of deep breaths did not provide higher ETo_2 values than 3 min of TVB.[6,14,27,28] The effect of 3 min of deep breaths has not been evaluated, but prolonged peak inspiratory efforts might exhaust morbidly obese patients.[29]

In the present study, the benefits of NPPV accrue largely from the generous tidal volumes (Fig. 2) which reduce the elastic work of breathing[30] and may enhance the more efficient washing-out of nitrogen. Moreover, reducing the elastic work of breathing may permit obese patients to execute a more effective deep breathing strategy.[30]

All patients in our study reached an ETo_2 of 90%, even with SV. Patients in the SV group breathing 18 L/min O_2 reached higher values of ETo_2 than what was reported with 9 or 10 L/min O_2.[14,28] Nimmagadda et al.[27] showed that ETo_2 increased with O_2 flow during deep breath ventilation but not during tidal ventilation breathing, but O_2 flow in this study was limited to 10 L/min. In the present study, we compared a new O_2 administration technique, i.e., NPPV with O_2 administration using SV and a high fresh gas flow (18 L/min). The O_2 flow of 18 L/min, which is the highest the Primus ventilator could deliver, could have helped to enhance O_2 administration with SV. This flow was also always above MV and thereby avoided the reduction of Fio_2 previously shown with O_2 flows below 10 L/min.[27] Moreover, avoidance of air leaks with a good mask seal avoided

dilution of inspired O_2 by room air and reduction of Fio_2.[31] It would also be interesting to examine another morbidly obese control group, i.e., where SV is used with standard O_2 flow rates (5–10 L/min).

Our results show that maximal O_2 administration can require more than 3 min in morbidly obese patients (Fig. 1). Such an improvement was not found in lean patients who reached maximal ETo_2 levels in <3 min.[15,27,32] Lengthening the time of O_2 administration may be important for the safety of morbidly obese patients.

Gas Exchange and Breathing Pattern

NPPV decreased elastic work such that normal CO_2 levels were easily maintained. This contrasted with the SV group, in whom the lower tidal volumes resulted in decreased MV and increased CO_2 levels (Table 2, Fig. 2). The absence of a significant increase in Pao_2 with NPPV may indicate a lack of power, as ABG samples were obtained in nine patients of each group, but high values of Pao_2 in the SV group also prevented attaining a significant difference (Table 2). Values of Pao_2 were higher in the control group than in previous studies of O_2 administration with 3 min of TVB in obese patients[13] or 1 min of deep breaths with 10 L/min O_2.[33] It is likely that both the 5-min duration and the high O_2 flow were responsible for the high Pao_2 with TVB, but this improvement could also be explained by a PEEP effect caused by the high fresh gas flow.[34] Finally, because of the increase of $Paco_2$ during the TVB trial, an improvement of Pao_2 with SV could have also been related to a rightward shifting of the hemoglobin dissociation curve (Bohr effect). Contrary to previous work performed in hypoxemic patients, the observed improvement of oxygenation in the present study, obtained after 5 min of O_2 administration with NPPV did not persist after intubation. These discrepancies may be explained by the difference in the study designs. In the present study, the period of deliberate apnea to a Spo_2 of 95% caused the reduction in lung volume back to the prior FRC. This suggests that the lung volume recruited using positive pressure ventilation was lost with the endotracheal tube was disconnected from the ventilator.

Time to Desaturation

Apnea after induction was designed to simulate a "cannot ventilate, cannot intubate" emergency. Several reasons may explain the lack of benefit in time to desaturation after O_2 administration using NPPV. In the present study, the deliberate prolonged period of apnea to a Spo_2 of 95% led to reduction in lung volume back to the prior FRC and thus the recruited lung volume because of positive pressure ventilation (pressure support + PEEP) was lost. In addition, FRC decreases rapidly after induction of general anesthesia and muscle relaxation in supine obese patients.[35] Also, the Spo_2 threshold of 95% to end the apnea may have been too high to obtain a significant difference of time to desaturation between groups. A threshold of 90% might have increased the difference, because of the shape of the desaturation curve,[36] but that might have been an unacceptable risk in these patients.

Side Effects

Tolerance was good and similar between groups. Most of the patients were unfamiliar with long-term support for sleep apnea, but this did not reduce tolerance. Tolerance of a tightly held facemask was previously rated identically before and after surgery.[37] As the mask must be held tightly to avoid leaks and to maintain maximal Fio_2,[31] it seems important to reassure patients before and during preoxygenation of their ability to breathe with the facemask.

We did not find any significant cardiovascular depressant effects of NPPV with respect to BP and heart rate (Fig. 4).

The low degree of gastric distension in the SV group is consistent with previous trials showing that residual gastric volume is low in fasted obese patients.[38] Gastric distension was greater after NPPV, but the increase was modest. O_2 administration with PEEP alone did not increase gastric air insufflation,[39] suggesting that the pressure support component promoted gastric distension during NPPV. Positive-pressure ventilation may increase gastric air content and hence may promote pulmonary aspiration during endotracheal intubation. There is a risk with an insufflation pressure of more than 20 cm H_2O, which can be easily obtained using manual ventilation.[40,41] In our present study, NPPV was used in a pressure-limited mode. As a result, no patients in the NPPV group received an airway pressure of more than 16 cm H_2O.

CONCLUSION

O_2 administration using NPPV in the operating room is safe, feasible, and efficient in morbidly obese patients. NPPV using moderate levels of pressure support and PEEP provide a higher ETo_2 than tidal volume spontaneous breathing, and accelerate the increase in ETo_2 during O_2 administration.

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