Open lung ventilation does not increase right ventricular outflow impedance: An echo-Doppler study*

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Objective: **Ventilation according to the open lung concept (OLC) consists of recruitment maneuvers, followed by low tidal volume and elevated positive end-expiratory pressure (PEEP). Elevated PEEP is associated with an increased right ventricular afterload. We investigated the effect of OLC ventilation on right ventricular outflow impedance during inspiration and expiration in patients after cardiac surgery using transesophageal echo-Doppler.**

Design: **A prospective, single-center, crossover, randomized, controlled clinical study.**

Setting: **Cardiothoracic intensive care unit of a university hospital.**

Patients: **Twenty-eight patients scheduled for elective cardiac surgery with cardiopulmonary bypass.**

Interventions: **In the intensive care unit, each patient was ventilated for approximately 30 mins according to both OLC and conventional ventilation. During OLC ventilation, recruitment maneuvers were applied until Pao_2/Fio_2 was >375 torr (50 kPa); during conventional ventilation no recruitment maneuvers were performed.**

Measurements and Main Results: **Transesophageal echo-Doppler measurements were performed at end-inspiration and end-expiration in a steady-state condition, 20 mins after initiation of a ventilation strategy. Mean acceleration of flow was determined in the long axis of the pulmonary artery in a transverse axis view. During OLC ventilation, a total PEEP of 14 ± 4 cm H_2O was applied vs. 5 cm H_2O during conventional ventilation. Mean acceleration during expiration was comparable between groups. During inspiration, OLC ventilation did not cause a decrease of mean acceleration compared with expiration, whereas this did occur during conventional ventilation.**

Conclusions: **Despite the use of elevated PEEP levels, ventilation according to OLC does not change right ventricular outflow impedance during expiration and decreases right ventricular outflow impedance during inspiration. (Crit Care Med 2006; 34:2555–2560)**

Key Words: positive end-expiratory pressure; right ventricular afterload; echo-Doppler cardiography; open lung concept; recruitment maneuver; cardiac surgery; cardiopulmonary bypass

Mechanical ventilation using elevated positive end-expiratory pressure (PEEP) is especially known to increase right ventricular (RV) afterload (1–3). Several studies have demonstrated that 15 cm H_2O of PEEP increased RV afterload (4, 5) but did not affect RV contractility (6). This might be explained by overdistention of aerated lung areas in the presence of atelectatic lung areas. In this regard, ventilation according to the open lung concept (OLC) has been introduced to avoid atelectasis (7). This is achieved by short periods of high inspiratory pressures to open up collapsed alveoli followed by elevated levels of PEEP to keep the alveoli open. While maintaining the lung open, the lowest possible pressure amplitude is used to minimize overdistention.

Recently, we showed that this ventilation strategy had several beneficial effects (8) and did not affect pulmonary vascular resistance or RV ejection fraction, assessed with a pulmonary artery catheter (9). In this latter study, however, these volumetric measurements obtained with the pulmonary artery catheter were averaged over 5 mins. Therefore, the separate effects of PEEP and tidal volume on RV afterload during OLC ventilation remained unknown.

As RV afterload was not affected by OLC ventilation measured with pulmonary artery catheter (9), we hypothesized that OLC ventilation does not affect RV afterload during the entire respiratory cycle. Echocardiographic studies have shown that elevated PEEP leads to elevated RV outflow impedance mainly during inspiration (2) and less during expiration (10, 11). We therefore conducted a study assessing RV outflow impedance by the echo-Doppler flow signal in the pulmonary artery during OLC ventilation, and we compared this with conventional ventilation (CV). Doppler echocardiography allows beat-to-beat measurements, allowing inspiratory and expiratory measurements of the right ventricular impedance (2, 10, 12).

METHODS

The local Human Ethics Research Committee approved this study, and each patient gave written informed consent. We prospectively enrolled 28 patients scheduled for cardiac surgery with use of cardiopulmonary bypass using a median sternotomy, without suspicion of pulmonary hypertension (assessed by preoperative echocardiography) or

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The authors have not disclosed any potential conflicts of interest.

DOI: 10.1097/01.CCM.0000239118.05093.EE

severe airway obstruction (forced expired volume in 1 sec or vital capacity in the predicted normal range, taken as predicted value ± 2 SD). Patient characteristics are given in Table 1.

Anesthesia was induced with midazolam (0.1 $mg \cdot kg^{-1}$) intravenously and sufentanil (2 $\mu g \cdot kg^{-1}$). Pancuronium was given only at induction of anesthesia to facilitate endotracheal intubation (0.1–0.2 $mg \cdot kg^{-1}$). During operation, the lungs were ventilated in pressure-controlled mode with a respiratory frequency adjusted to maintain $Paco_2$ between 34 and 49 torr (4.5 and 6.5 kPa), tidal volume 8–10 mL/kg (ideal body weight), inspiratory/expiratory ratio 1:1, and PEEP 5 cm H_2O. FIO_2 was adjusted to achieve a Pao_2 of 75–98 torr (10–13 kPa). The pericardium was not closed. After surgery, patients were sedated with midazolam 0.05–0.1 $mg \cdot kg^{-1} hr^{-1}$, and analgesia was achieved with morphine 5–10 $\mu g \cdot kg^{-1} \cdot hr^{-1}$. Patients did not receive inhaled nitric oxide or phosphodiesterase inhibitors.

Table 1. Patient characteristics

Age, yrs	64 ± 11
Male/female	16/12
Weight, kg	76 ± 16
BMI, kg/m^2	26 ± 4
FEV_1, % predicted	92 ± 19
FVC, % predicted	92 ± 15
OR	
CABG	12
CABG ± valve	3
AVR	5
MVR	3
MVP	2
ASD	1
MAZE	1
Extirpation myxoma	1
Aortic clamp time, mins	95 ± 41

BMI, body mass index; FEV_1, forced expired volume in 1 sec; FVC, forced vital capacity; OR, odds ration; CABG, coronary artery bypass graft; AVR, aortic valve replacement; MVR, mitral valve replacement; MVP, mitral valvuloplasty; ASD, atrium septum defect repair; MAZE, atrial compartmentalization.

Thirty minutes after the patient's arrival in the intensive care unit, each ventilation strategy was applied once on each patient in a crossover design to minimize the effect of confounding variables. The order of the applied ventilation strategies was randomized by sealed envelopes. Each ventilation strategy commenced after disconnection from the ventilator for 15 secs, which has been shown to result in an immediate lung collapse (13). Each ventilation strategy was maintained for 30 mins. Conventional ventilation was started with volume control ventilation at the following settings: tidal volume 8 mL/kg (ideal body weight), PEEP 5 cm H_2O, and inspiratory/expiratory ratio 1:2. FIO_2 was set to achieve a Pao_2 between 75 and 98 torr (10–13 kPa), and respiratory rate was adjusted to achieve a $Paco_2$ between 34 and 49 torr (4.5–6.5 kPa).

Ventilation according to the OLC was started by switching the ventilator to a pressure-controlled mode with a respiratory frequency of 40 breaths/min. FIO_2 was set to achieve a Pao_2 between 75 and 98 torr (10–13 kPa), PEEP 10 cm H_2O, inspiratory/expiratory ratio 1:1, and a driving pressure to obtain a tidal volume of 4–6 mL/kg aiming at a $Paco_2$ of 34 to 49 torr (4.5–6.5 kPa). A lung recruitment maneuver was performed by increasing peak inspiratory pressure to 40 cm H_2O during 15 secs in order to increase the Pao_2/FIO_2 ratio to a value >375 torr (50 kPa), as this mimics an open lung (14, 15). If this value was not reached, a recruitment maneuver was repeated by adding 5 cm H_2O to the previous peak inspiratory pressure, up to a maximum peak inspiratory pressure of 60 cm H_2O. If the Pao_2/FIO_2 ratio decreased slowly below 375 torr (50 kPa) after recruitment, PEEP was increased with 2 cm H_2O and the recruitment maneuver (beginning at 40 cm H_2O) was repeated. If Pao_2/FIO_2 ratio decreased below 375 torr (50 kPa) after an (accidental) disconnection, PEEP was not increased and a new recruitment maneuver was performed.

Echo-Doppler studies were performed by one investigator with a System Five Ultrasound (GE Vingmed, Holten, Norway) with a KN100062 5-MHz transesophageal probe (GE Medical, Holten, Norway). Airway pressure was measured at the end of the endotracheal tube, and the signal was displayed on the electrocardiogram channel of the echo device. During both ventilation strategies, echo-Doppler was performed after 20 mins of steady-state ventilation. End-expiratory images, defined as the last beat before inspiration, and end-inspiratory images, defined as the last beat before expiration, were stored electronically and on videotape. All images were reviewed by a cardiologist. Measurements were performed off-line by one investigator in triplicate and averaged.

Right ventricular outflow impedance was assessed by the mean acceleration (Ac_{mean}) of the pulmonary artery flow, measured with the ultrasound beam parallel to the long axis of the main pulmonary artery. The Doppler sample volume was placed beyond the pulmonary valve in the midlumen of the main pulmonary artery to record the pulmonary artery flow (Fig. 1). The pulsed Doppler spectrum was measured at a high sweep speed of 100 mm/sec. Ac_{mean} was calculated by dividing the peak velocity by the acceleration time (Fig. 1). We also measured the velocity time integral, which is the area under the flow curve of the pulmonary artery and which reflects the stroke volume. The superior vena cava (SVC) was examined in a long- and short-axis view. The maximal diameter during inspiration and expiration was measured in the short axis view using M-mode.

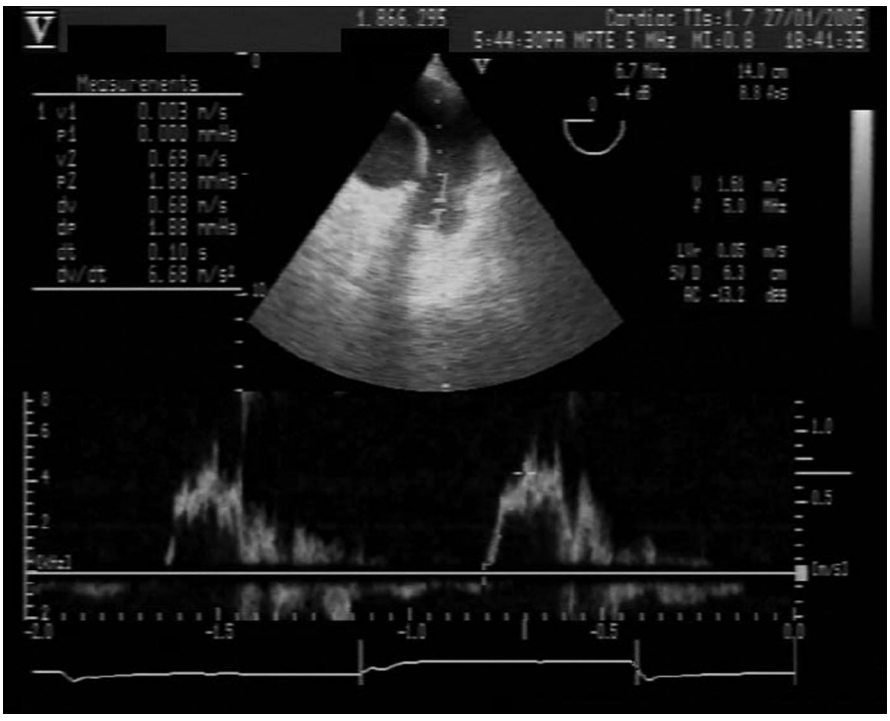

Figure 1. Example of mean acceleration measurement of the pulmonary artery on high-speed Doppler recording. The *bottom line* represents airway pressure. The measured beat on the *right* occurred during inspiration.

Fluid management during the study was guided by the collapsibility of the SVC. The SVC collapsibility index was calculated as diameter of the SVC during expiration minus diameter of the SVC during inspiration divided by diameter of the SVC during expiration times 100. An SVC collapsibility index >20% was taken to indicate hypovolemia (16, 17). Hypovolemia was treated with starch colloids (Voluven) with a bolus of 250 mL. Thereafter, measurement of the SVC collapsibility index was repeated until it was <20%.

Cardiovascular and respiratory measurements were made just before acquiring the echocardiographic images. After the echocardiographic images were acquired, patients were conventionally ventilated. Patients were weaned according to the local protocol, which was briefly described in a previous study (8).

Statistics. This randomized clinical trial was designed to detect a 25% difference in mean acceleration of the Doppler flow signal in the pulmonary artery between OLC ventilation and CV during expiration. Under the assumption that the SD of the mean acceleration is about 3.4 m/sec^2 (10), power analysis established that a sample size of 28 patients would have 80% power of detecting a difference of the mean acceleration in the pulmonary artery of 25% at a significance level of $\alpha < .05$ between the two groups.

Between-group differences for hemodynamic variables were tested with a paired Student's *t*-test. Results are presented as mean ± SD.

RESULTS

Patient characteristics are displayed in Table 1. Ten patients were on β-blockade preoperatively. During OLC ventilation, 2.0 ± 1.3 recruitment maneuvers were applied to open the lung, with a mean peak inspiratory pressure of 45 ± 5 cm H_2O. A PEEP of 14 ± 4 cm H_2O was used to keep the lung open. Other respiratory data are given in Table 2. All patients had a regular rhythm (either sinus or sequential atrioventricular pacing) and no pulmonary valve regurgitation. Three patients had trivial tricuspid regurgitation, and five patients had mild tricuspid regurgitation. Tricuspid regurgitation was not affected by the ventilation strategy in any patient. No patient required reoperation within the first 72 hrs.

Ac_{mean} during expiration was comparable between both ventilation strategies. Inspiration did not cause a significant decrease in Ac_{mean} compared with expiration during OLC ventilation but did do so during CV (Fig. 2). Ac_{mean} during inspiration was significantly lower during CV compared with OLC ventilation.

During CV, velocity time integral showed a significant difference between inspiration and expiration, but not during OLC (Table 2). There were no significant differences in any variable when OLC as the first strategy was compared with OLC as the second strategy (Table 2).

Heart rate, mean arterial pressure, and right atrial pressure were comparable between both ventilation strategies (Table 2). Throughout the entire study period, nine patients received dobutamine (4.3 ± 1.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$), which was combined with phenylephrine in two patients (0.15 ± 0.07 $\mu g \cdot kg^{-1} \cdot min^{-1}$). No changes in any drugs dosages occurred during the study period. During OLC ventilation, two patients received a total of three fluid boluses, and during CV, four patients received a total of six fluid boluses.

One patient developed subcutaneous emphysema 6 hrs after we concluded the study, due to obstruction of the chest tube. Air leakage from the chest tube was present before initiation of the study and was not increased by recruitment maneuvers.

DISCUSSION

This study shows that an elevated PEEP during OLC ventilation did not affect Ac_{mean} during expiration in cardiac surgery patients. Furthermore, inspiration did not change Ac_{mean} during OLC ventilation compared with expiration, whereas this did occur during CV.

In this study, mean acceleration of the pulmonary flow is used as marker of RV outflow impedance. This impedance reflects ventricular afterload, which is defined as the ventricular wall tension during systole. The tension in the ventricular wall that the sarcomeres must overcome to shorten is related not only to the transmural pressure during systole but also to the cavity size through the Laplace relation. However, obtaining reproducible right ventricular cavity diameters using echocardiographic measurements is difficult. Aortic or pulmonary impedance has also been used to accurately measure afterload. The impedance is the pressure divided by the flow at that instant, so that this index of the afterload varies at each stage of the contraction cycle. Factors reducing flow, such as a high arterial pressure or outlet valve stenosis or loss of arterial compliance, will increase impedance and hence the afterload. An approximation can be made by using echocardiography to determine the blood flow at any instant during systole. Therefore, an average value for the impedance can be calculated by dividing the maximum velocity by the time from the onset of flow until the peak velocity is reached, the so-called Ac_{mean}. Ac_{mean} of the aortic flow is reduced by afterloading (18) and increased by unloading (19). In contrast to the left ventricle, studies validating Ac_{mean} for the right ventricle are still lacking. Although the method has not been formally validated, several authors have used Ac_{mean} to describe the changes during the respiratory cycle as a marker to good effect (2, 10, 12). As Ac_{mean} allows dynamic measurements during the respiratory cycle, we used Ac_{mean} as marker for RV outflow impedance.

This study shows that the use of OLC ventilation with a higher PEEP level than CV is not associated with an elevation of RV outflow impedance during expiration. Elevated PEEP levels do not resolve atelectasis (15), but recruitment maneuvers followed by sufficient levels of PEEP avoid atelectasis in cardiac surgery patients (13, 20). Duggan and colleagues (21) showed that atelectasis causes a significant increase in RV afterload (assessed with pulmonary artery catheter and echocardiography) and that this may even lead to RV failure in healthy rats. This effect of atelectasis on RV afterload can be explained by two mechanisms: a) local hypoxic pulmonary vasoconstriction in nonaerated lung areas (22–24); and b) overdistention in aerated lung areas. High tidal volume ventilation in aerated lung areas occurs in the presence of atelectasis, compressing the surrounding vasculature and thus increasing RV afterload. This could explain the results of Huemer et al. (12), who found no increased RV afterload using 12 cm H_2O continuous positive airway pressure in healthy volunteers (without atelectasis), assessed by echo-Doppler. In the present study, atelectasis was avoided by OLC ventilation and could explain the fact that the RV outflow impedance during expiration is comparable between the two ventilation strategies.

During CV, RV outflow impedance (as assessed by Ac_{mean}) increased significantly during inspiration. These changes in RV outflow impedance during the respiratory cycle were also found by Poelaert et al. (11) in cardiac surgery patients and by Vieillard-Baron et al. (10) in patients with acute respiratory distress syndrome (ARDS). High PEEP levels even enhance this RV outflow impedance increment dur-

Table 2. Respiratory data

	Mean (n = 28)		OLC First Strategy (n = 14)		OLC Second Strategy (n = 14)	
	Exp	Insp	Exp	Insp	Exp	Insp
Ac_{mean}						
OLC	9.6 ± 2.2	10.0 ± 2.9[a]	9.5 ± 2.5	10.6 ± 3.6[a,b]	9.8 ± 1.9	9.4 ± 2.0
CV	10.2 ± 3.3	8.6 ± 2.9[b]	10.3 ± 4.4	8.5 ± 3.2[b]	10.0 ± 1.9	8.7 ± 2.8[b]
VTI						
OLC	15.1 ± 3.9	15.4 ± 4.1	14.1 ± 4.1	14.8 ± 4.5	16.1 ± 3.7	15.9 ± 3.6
CV	16.1 ± 4.0	15.1 ± 4.2[b]	15.4 ± 4.3	14.5 ± 4.7	16.9 ± 3.7	15.7 ± 3.8[b]
AcT						
OLC	0.08 ± 0.003[a]	0.08 ± 0.03[a]	0.08 ± 0.02[a]	0.08 ± 0.03[a]	0.09 ± 0.03	0.09 ± 0.03
CV	0.10 ± 0.02	0.10 ± 0.002	0.09 ± 0.02	0.1 ± 0.02	0.1 ± 0.02	0.1 ± 0.02
Vmax						
OLC	0.80 ± 0.22	0.76 ± 0.19[b]	0.76 ± 0.2[a]	0.74 ± 0.2[b]	0.84 ± 0.2	0.79 ± 0.2
CV	0.84 ± 0.20	0.80 ± 0.25	0.80 ± 0.2	0.79 ± 0.3	0.87 ± 0.2	0.82 ± 0.3
HR						
OLC	87 ± 13		85 ± 13		88 ± 14	
CV	85 ± 12		85 ± 14		86 ± 10	
MAP						
OLC	80 ± 13		76 ± 10		85 ± 14	
CV	82 ± 11		83 ± 12		81 ± 9	
RAP						
OLC	14 ± 3[a]		13.6 ± 2.5		14 ± 3[a]	
CV	11 ± 3		11.6 ± 17		11 ± 4	
RR						
OLC	38 ± 4		38 ± 4		38 ± 4	
CV	13 ± 1		13 ± 1		13 ± 1	
VT/kg						
OLC	5.1 ± 0.9		5.1 ± 1.0		5.1 ± 0.8	
CV	8.4 ± 1.1		8.3 ± 1.1		8.6 ± 1.1	
PIP						
OLC	25 ± 3[a]		26 ± 4		24 ± 4	
Pplat						
CV	17 ± 3		18 ± 3		17 ± 4	
Pmap						
OLC	18.5 ± 2.3[a]		19.0 ± 2.4[a]		17.8 ± 2.3	
CV	10.4 ± 2.2		10.8 ± 2.6		9.6 ± 1.5	
PEEPext (PEEPint)						
OLC	12 ± 2[a] (3 ± 1)[a]		13 ± 2[a] (3 ± 1)[a]		12 ± 2[a] (3 ± 2)[a]	
CV	5 ± 0 (0 ± 0)		5 ± 0		5 ± 0	
CRS						
OLC	38 ± 10		35 ± 10		40 ± 10	
CV	34 ± 6		33 ± 5		35 ± 7	
Pa_{O_2}/F_{IO_2}						
OLC	422 ± 62[a] (56 ± 8)[a]		408 ± 66[a] (54 ± 9)[a]		437 ± 58 (58 ± 8)[a]	
CV	343 ± 97 (45 ± 12)		342 ± 83 (45 ± 11)		358 ± 106 (48 ± 14)	
Pa_{CO_2}						
OLC	(38 ± 6) 5.0 ± 0.7		39 ± 6 (5.2 ± 0.8)		36 ± 4 (4.9 ± 0.6)	
CV	(38 ± 5) 5.1 ± 0.8		39 ± 6 (5.2 ± 0.9)		37 ± 5 (4.9 ± 0.7)	

OLC, open lung concept; Exp, end-expiratory; Insp, end-inspiratory; Ac_{mean}, mean acceleration (m/sec^2); CV, conventional mechanical ventilation; VTI, velocity time integral (cm); AcT, acceleration time (secs); Vmax, maximal flow velocity (m/sec); HR, heart rate (beats/min); MAP, mean arterial pressure (mm Hg); RAP, right atrial pressure (mm Hg); RR, respiratory rate (min^{-1}); VT/kg, tidal volume divided by ideal body weight (mL/kg); PIP, end-inspiratory pressure (cm H_2O): for the OLC group (pressure-controlled ventilation), this was the peak inspiratory pressure, and for the CV group (volume-controlled ventilation), this was the plateau pressure (Pplat, cm H_2O); Pmap, mean airway pressure (cm H_2O); PEEPext, extrinsic positive end-expiratory pressure (cm H_2O); PEEPint, intrinsic positive end-expiratory pressure (cm H_2O); CRS, respiratory compliance (mL/cm H_2O).

[a] $p < .05$ OLC vs. CV; [b] $p < .05$ Insp vs. Exp. Pa_{O_2}/F_{IO_2} ratio is the Pa_{O_2} divided by fractional F_{IO_2} (torr, in parenthesis kPa). Pa_{CO_2} expressed in torr (kPa).

ing inspiration in ARDS patients (2). In contrast to these studies, OLC ventilation did not increase RV outflow impedance during inspiration in the present study. In the present study, the tidal volume used was lower during OLC ventilation compared with CV, which may explain the lack of increase in RV outflow impedance during inspiration. On the other hand, alveolar overdistention during inspiration is even reduced by application of OLC ventilation, despite the use of elevated PEEP levels. De Matos et al. (25) demonstrated in ARDS patients that the degree of overdistention during inspiration decreased when a recruitment maneuver was performed first, followed by high PEEP levels. This implies that during OLC ventilation RV outflow impedance is not increased during inspiration due to a) the reduction of tidal volume ventilation in aerated lung areas caused by homogenization of pulmonary gas distribution; and b) the use of lower tidal volume, set on the ventilator. Furthermore, these two effects of OLC ventilation act in synergy: Homogenization of pulmonary gas distribution reduces tidal volume ventilation of aerated lung areas, which is reduced even further by the lower tidal volume ventilation set on the ventilator.

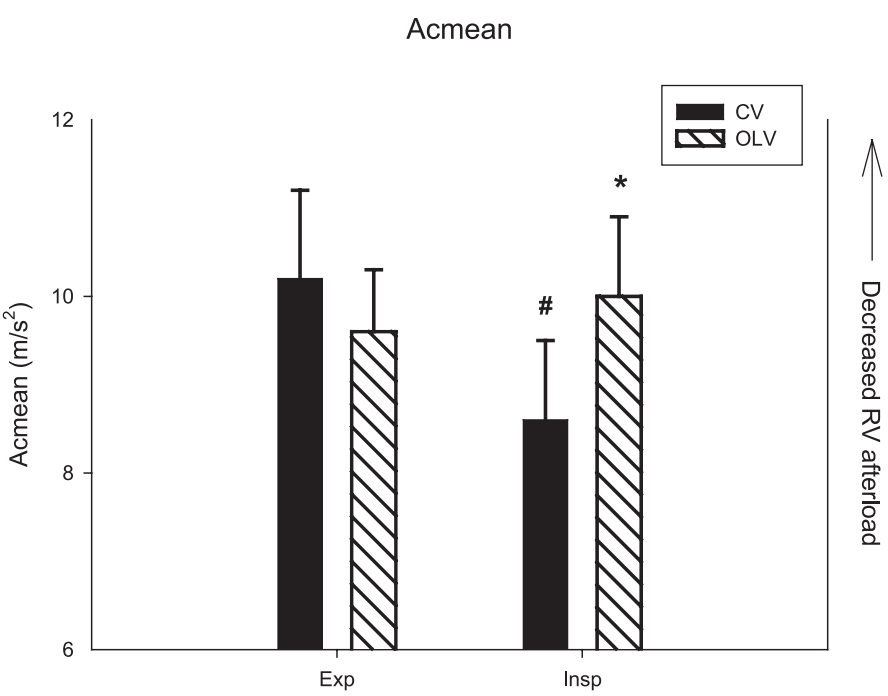

Figure 2. Mean acceleration (*Acmean*) of the pulmonary flow; high value reflects low right ventricular (*RV*) afterload. *CV*, conventional ventilation; *OLC*, open lung concept; *Exp*, expiration; *Insp*, inspiration. *$p < .05$ vs. CMV; #$p < 0.5$ vs. exp.

Ventilation according to the OLC is a ventilation strategy designed for ARDS patients (7). In a previous study, we showed that this ventilation strategy had beneficial effect on postoperative lung volumes and oxygenation in cardiac surgery patients (8). Interleukin release was also attenuated by application of OLC (26); in this latter study no increased incidence of myocardial infarction or pneumothorax was seen. OLC also did not lead to an increased duration of ventilation or an increased weaning time (8). Also, other studies using recruitment maneuvers followed by elevated PEEP levels have not reported an increased rate of these complications in cardiac surgery patients (9, 13, 20, 27, 28). In this study, however, one patient with a proven pneumothorax before recruitment developed subcutaneous emphysema 6 hrs after recruitment. The link between subcutaneous emphysema and a recruitment maneuver cannot be excluded but is unlikely: Air leakage did not increase after recruitment, and a chest tube obstruction during the fast development of subcutaneous emphysema was observed. The ongoing development of subcutaneous emphysema stopped after relieving the obstruction.

The aim of this study was not to investigate the hemodynamic effect of recruitment maneuvers but to evaluate the effect of a ventilation strategy. Experimental (29) and clinical (30) studies suggest that during a recruitment maneuver, RV outflow impedance is increased for 1–2 mins. We supported these findings in an earlier study: RV contractility was not affected 30 mins after a recruitment maneuver (9) in cardiac surgery patients, and in this present study, RV afterload was not increased 20 mins after a recruitment maneuver. Although a potential effect of a recruitment maneuver on RV afterload is very transient in cardiac surgery patients without known RV failure, it is questionable whether this can be extrapolated to patients with RV failure. The safety of recruitment maneuvers in patients with RV failure remains to be established. In addition, in patients who have undergone cardiac surgery, the pericardium has been opened. Therefore, the effect of OLC ventilation on RV outflow impedance with an intact pericardium (such as ARDS patients) still remains unknown.

CONCLUSION

Despite the use of elevated PEEP levels, ventilation according to OLC does not change RV outflow impedance during expiration and decreases RV outflow impedance during inspiration.

ACKNOWLEDGMENT

We thank Laraine Visser-Isles (Department of Anesthesiology) for correcting the English in this article.

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