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Original Article

The Effect of Low Tidal Volume Ventilation during Cardiopulmonary Bypass on Postoperative Pulmonary Function

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Abstract

Background: *Postoperative pulmonary dysfunction is one of the most frequent complications after cardiac surgery and it is believed to result from the use of cardiopulmonary bypass (CPB). In this study, we investigated the effect of low tidal volume ventilation during CPB on postoperative gas exchange and lung mechanics.*

Methods: *This prospective randomized study included 100 patients undergoing elective coronary artery bypass grafting. In 50 patients, low tidal volume ventilation [tidal volume (TV) = 3 ml/kg, respiratory rate (RR) = 12/min, fraction of inspiratory oxygen (FIO2)= 1.0, positive end expiratory pressure (PEEP) = 5 cmH_2O] was applied during CPB (group I); and in the other 50 patients (group II), the lungs were open to the atmosphere without ventilation. Measurements were taken preoperatively, after CPB, and before discharge.*

Results: *Post-bypass PaO2 (just after CPB 85 versus75) was higher significantly in group I (P value < 0.05). Decrease in postoperative forced expiratory volume in 1 second (25% versus 30%) and forced vital capacity (32% versus 35%) was less significant in group I. Also, time to extubation (5 hrs versus 5.5 hrs) was shorter ingroup I.*

Conclusion: *Continued low tidal volume ventilation during CPB improved post-bypass oxygenation and lung mechanics.*

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Introduction

Lung dysfunction after coronary artery bypass grafting (CABG) remains an important cause of postoperative morbidity despite continuing improvements in cardiopulmonary bypass (CPB) techniques and postoperative intensive care.[1] Pulmonary dysfunction not only prolongs mechanical ventilation, intensive care in intensive care unit(ICU), and total duration of hospitalization time but it also increases treatment costs.[2] The etiology of pulmonary dysfunction after open heart surgery is thought to be multi factorial, occurring as a result of the combined effects of anesthesia, CPB, and surgical trauma. CPB in particular is known to activate the inflammatory process, resulting in increased pulmonary capillary permeability and damage to lung parenchyma.[1] Even after uncomplicated cardiac surgery, a midline sternotomy causes significant reductions in total lung capacity, vital capacity, forced expiratory volume in 1 second (FEV_1), and functional residual capacity. These changes may give rise to postoperative atelectasis and frequently at least mild hypoxemia.[3] Atelectasis in the post-bypass period is the main cause of intrapulmonary shunt and poor arterial oxygenation.[2]

The physiologic disturbance after CPB can be categorized into abnormal gas exchange and poor lung mechanics.[1] Most interventions focus specially on airway management,

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endotracheal suctioning, extubation, and physiotherapy that includes deep breathing and coughing exercises, incentive spirometry, and the application of a range of measurement to achieve positive airway pressures and alveolar recruitment. More recently, the effects of postoperative position, pain management, and early ambulation on pulmonary complications have been evaluated.[4]

A wide range of ventilatory strategies while on CPB have been attempted; they include continuous positive airway pressure (CPAP) with pressures of 5-15 cmH_2o, high frequency low tidal volume ventilation (100 breaths/min), inspired oxygen concentrations from 21% to 100%, and bilateral extracorporeal circulation using the lungs to oxygenate the blood while on bypass. Although some small and transient benefits for CPAP with 10 cmH_2O have been demonstrated, no convincing clinical benefits for any of the ventilatory strategies have thus far emerged.[5] In a swine model, Lamarche et al. concluded that mechanical ventilation prevented the pulmonary endothelial dysfunction due to reperfusion after CPB.[6] The effects of ventilation during CPB have been tested in a number of studies using vital capacity maneuvers, CPAP, and continuous ventilation over the period of cardiac arrest.[7] Meanwhile, better postoperative gas exchange and less pulmonary shunting were observed in patients who received CPAP (10 cmH_2O), although the beneficial effects of CPAP were not evident in an animal CPB model.

To date, the evidence for the benefits of maintaining ventilation alone during CPB has been inconsistent, with most studies showing no significant preservation of the lung function.[7] It seems that further studies are necessary to find out which ventilation strategy could improve post-bypass lung function. The goal of this study was to evaluate whether continuous low tidal volume ventilation on CPB would prevent atelectasis, preserve lung mechanics, and improve oxygenation after cardiac surgery.

Methods

The study was approved by the institutional Ethics Committee, and informed written consent was signed by all the patients. In total, 100 patients undergoing elective CABG were enrolled in this prospective, randomized clinical trial. On the day of surgery, the patients were randomized via the closed envelope method into either group I (n = 50) or group II (n = 50). Exclusion criteria were comprised of the American society of anesthesiologists (ASA) class > III, age > 70 years, poor left ventricular function (left ventricular ejection fraction < 40%), valvular heart disease (any disease process involving one or more of the valves of the heart), reoperation, renal impairment (elevated serum creatinine), and significant pulmonary disease as defined by preoperative FEV_1 or forced vital capacity (FVC) values < 40% of the predicted value. All the patients received education and underwent spirometry on the day before surgery. Arterial and central venous cannulation was conducted for all the patients, who also received a standard anesthetic technique, using premedication with intramuscular morphine (0.15 mg/kg) and promethazine (0.5 mg/kg) 2 hours before the induction of anesthesia. After preoxygenation for at least 3 minutes, anesthesia was induced by using sufentanil (25µg), propofol (1.5 mg/kg), and atracurium (0.5 mg/kg). This was followed by manual ventilation at a fraction of inspired O_2 (FIO_2) of 1.0 for 3 minutes and subsequent tracheal intubation. After intubation, mechanical ventilation was started with volume control ventilation at the following setting: tidal volume (TV) = 7 ml/kg (lean body mass), positive end expiratory pressure (PEEP) = 5 cmH_2O, inspiratory/expiratory (I/E) ratio = 1:2, FIO_2 = 1.0, and Respiratory Rate (RR) = 12/min. Anesthesia was maintained by a constant infusion of sufentanil, propofol, and atracurium as required. CABG was performed via a median sternotomy. During CPB, the patients in group I (n = 50) underwent low tidal volume ventilation (TV = 3 ml/kg [lean body mass], RR = 12, FIO_2 = 1.0, PEEP = 5 cmH_2O, and I/E ratio = 1:2), and the patients in group II (n = 50) underwent typical (no ventilation) CPB. In both groups, a standard CABG procedure was performed using the roller pump (Affinity, USA) and cold crystalloid cardioplegia (Martindale, U.K) for myocardial protection. After surgery, all the patients were transferred to the ICU and were managed and monitored according to usual routine requirements. In the ICU, the lungs were ventilated using the synchronized intermittent mandatory ventilation (SIMV) ventilation mode. The initial setting was: FIO_2 = 1.0, RR = 12, TV = 7 ml/kg (lean body mass), and PEEP = 5 cmH_2O. Respiratory rate and FIO_2 were then adapted to obtain an arterial $PaCO_2$ = 35-40 and PaO_2 > 90 mmHg. Four hours after the completion of CPB measurements, FIO_2 was 0.4 using oxygen enriched air. The decision for extubation was made according to the protocol such as adequate gas exchange (PaO_2 > 70 mmHg, FIO_2 < 0.4, $PaCO_2$ = 35-40 mmHg, and spontaneous RR < 25), hemodynamic stability (i.e. adequate cardiac output, controlled arrhythmias, and no pulmonary edema), and absence of bleeding.[8]

PaO_2 was measured after the induction of anesthesia and endotracheal intubation, before CPB, just after CPB, 5 minutes, 2 and 4 hours after the completion of CPB, and after extubation in the ICU. Time of extubation, FEV_1, and FVC measured on the 5th postoperative day were recorded. Pre and postoperative spirometries were done by someone blinded to the groups. The anesthesia protocol and surgery were done by one anesthesiologist and one surgeon, respectively. ICU management as well as data collection was performed by the nursing staff, who were blinded to the study groups.

The values are expressed as mean and standard deviation. The data were analyzed with SPSS 10th version, and p values < 0.05 were considered statistically significant.

Results

The demographic data between the two groups were comparable (Table 1). There were no significant differences in age, sex, body mass index, history of cigarette smoking, preoperative FEV_1 and FVC, pre-CPB PaO_2, mean CPB time, mean aortic cross-clamp time, and mean operation time between the two groups (Table 2). Post-CPB PaO_2 was higher significantly in group I (Figure 1). Postoperative FEV_1 and FVC decreased in both groups, but the decrease in group I was smaller (p value = 0.04, p value = 0.03). Time of extubation (p value = 0.01) was shorter in group I (Table 3). There were no complications in the two groups.

Table 1. Preoperative patients> characteristics*

	Ventilated group	Not ventilated group	p value
Age (y)	59.8±12.2	57.4±14.4	0.39
Gender (M/F)	38/12	36/14	0.94
Body mass index	26.2±5.4	25.3±7.3	0.41
History of cigarette smoking (%)	46	42	0.98
FEV_1 (L)	2.6±0.7	2.4±0.8	0.23
FVC (L)	3.3±0.9	2.9±0.8	0.08

*Data are presented as mean±SD
FEV_1, Forced expiratory volume in 1 second; FVC, Forced vital capacity

Table 2. Intraoperative patients> characteristics*

	Ventilated group	Not ventilated group	p value**
Cardiopulmonary bypass time (min)	47.83±15.58	52.00±10.56	0.81
Aortic cross-clamp time (min)	28.50±10.87	29.17±5.52	0.93
Operation time (min)	172.17±20.18	168.05±19.48	0.93

*Data are presented as mean±SD
**Mann Whitney test

Table 3. Postoperative patients> characteristics*

	Ventilated group	Not ventilated group	p value
Extubation time (hr)	5	5.5	0.01
Decrease in FEV_1 (%)	25	30	0.04
Decrease in FVC (%)	32	35	0.03

*Data are presented as mean±SD
FEV_1, Forced expiratory volume in 1 second; FVC, Forced vital capacity

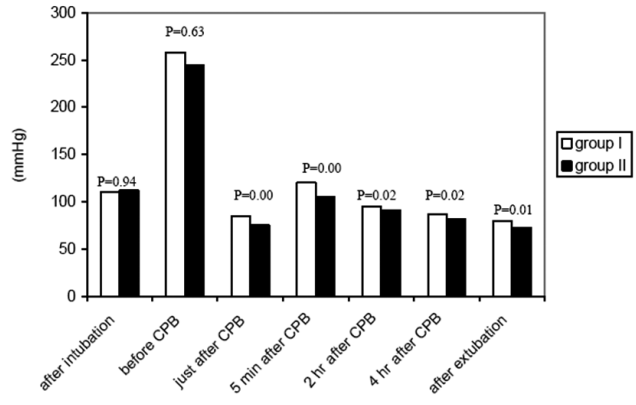

Figure 1. Values for oxygenation (mean PaO_2) as a function of time

Discussion

In order to evaluate the influence of continued low tidal volume ventilation during CPB on post-bypass lung function and oxygenation, we designed the present study. We evaluated changes in pre and postoperative FEV_1 and FVC on the basis of the theory that continued ventilation on CPB could reduce the decrease in these parameters. Also, the measurement of PaO_2 and extubation time was the simplest and least expensive predictor of postoperative lung function.

In adults undergoing coronary bypass procedures, there is a reduction of at least 25-50% in FVC and FEV_1 postoperatively.[8] In our study, the decrease in FEV_1 and FVC was 25% and 32% in the ventilated group and 30% and 35% in the non-ventilated group. Gagnon et al. argued that low tidal volume ventilation (3 ml/kg) without PEEP ventilation per CPB could not significantly change in pulmonary vascular resistance index (PVRI), PaO_2/FIo_2 ratio, mean pulmonary artery pressure (MPAP), pulmonary complications, and total length of stay when comparing to the non-ventilated group.[9] We applied low tidal volume ventilation (3 ml/kg) plus PEEP = 5 cmH_2O in the ventilated group. Improvement in oxygenation and lung volumes in the ventilated group in our study may be due to the application of PEEP.

In both Magnusson's and Berry's studies, there was a trend toward improved post-bypass pulmonary function by using CPAP.[10, 11] The provisional results of this study are suggestive of a benefit from continued low tidal volume ventilation during bypass. It appears that lung inflation decreases the bronchial blood flow and that this is probably due to purely mechanical effects on the bronchopulmonary arterial anastomoses with both compression and stretching of these vessels. This raises the possibility that the repetitive inflation and deflation of lungs at physiological intra alveolar pressure is necessary for normal bronchial arterial flow secondary to the cyclical compression and relaxation of the vessels. In that case, cessation of ventilation during bypass would reduce the bronchial flow and predispose the patient to ischemic lung

injury.[12] In one study, Lindsay et al. concluded that continued ventilation during bypass might reduce lung injury. They applied a higher tidal volume than that utilized in our study (5 ml/kg) and measured extra vascular lung water and extubation time.[12] We chose low tidal volume ventilation (TV = 3 ml/kg) because movement from mechanical ventilation with higher volumes during CPB may interfere with surgery. Also, we used some simple and available clinical parameters because of our limited facilities.

There was a significantly shorter extubation time in the ventilation group, as was the case in the Lindsay study.[12] This was probably due to the improvement in postoperative oxygenation. Shorter extubation time decreases stay time in ICU and total duration of hospitalization and relative costs. Because patient selection was according to our inclusion and exclusion criteria, the patients enrolled in the study were those who were relatively stable and fortunately none of them had predicted complications (cardiovascular or pulmonary).

Although the evidence for the benefits of maintaining ventilation alone during CPB is inconsistent, we found a correlation between the continued low tidal volume ventilation on CPB and post-bypass oxygenation and lung volumes.

Conclusion

In conclusion, the results of this study showed that continued low tidal volume ventilation during CPB improved post-bypass oxygenation and lung mechanics. This simple and inexpensive method could be used to reduce post-bypass lung injury.

The completion of studies that evaluate more clinical parameters is necessary to confirm or otherwise any benefits.

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