Effects of positive end-expiratory pressure (PEEP) in cardiac surgery patients

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The role of positive end-expiratory pressure (PEEP) in the postoperative course of cardiac surgery patients remains questionable. In this prospective study, we examined the effect of different levels of PEEP on arterial oxygenation, \( S\text{O}_2 \) and \( P\text{O}_2 \) values, and on haemodynamic indices, during the early postoperative period in cardiac surgery patients. Upon transfer to the ICU, 67 adult patients with normal preoperative respiratory status were randomly assigned to receive zero PEEP (Group A), 5 cmH\(_2\)O (Group B), or 10 cmH\(_2\)O PEEP (Group C) during mechanical ventilatory support. \( Pa\text{O}_2/Fi\text{O}_2 \) ratio, mixed venous \( P\text{O}_2 \) and \( S\text{O}_2 \), and cardiac index, were measured 30 min, 4 h and 8 h after application of mechanical ventilation in the ICU, just prior to extubation, half an hour after extubation, and 4 h post-extubation. We found no statistically significant differences (\( P=\text{n.s.} \)) in arterial oxygenation expressed by \( Pa\text{O}_2/Fi\text{O}_2 \) ratio, \( S\text{O}_2 \) and \( P\text{O}_2 \) values, and in cardiac index among the three groups at any study interval. We conclude that low levels of PEEP have no advantage over zero PEEP in improving gas exchange in the early postoperative course of patients following open heart surgery.

Introduction

Respiratory dysfunction, particularly atelectasis, leading to early postoperative arterial hypoxaemia is commonly seen in open heart surgery patients (1). Routine use of 5-10 cmH\(_2\)O positive end-expiratory pressure (PEEP) has been advocated in these patients (2), but its utility is largely unknown. Valta et al. found that PEEP less than 10 cmH\(_2\)O was not effective to reopen atelectatic lung units after open heart surgery (3). On the other hand, the use of PEEP is sometimes avoided in cardiac surgery patients because of circulatory depression or increased risk of barotrauma. The objective of this study was to examine the effect of different levels of PEEP on arterial oxygenation, \( S\text{O}_2 \) and \( P\text{O}_2 \) values, and on haemodynamic indices, during the early postoperative period of cardiac surgery patients.

Patients and Methods

Following Institutional Ethical Committee approval and informed consent, we prospectively included in this study a group of 69 patients. There were 56 males and 13 females of mean age 61.3 (± 6.2) years. Two patients were excluded because of severe perioperative complications.

INCLUSION CRITERIA

Patients undergoing elective coronary bypass graft surgery with preoperative left ventricular ejection fraction ≥45%, New York Heart Association Class I or II, and with normal preoperative respiratory function (FVC ≥ 75% of predicted and FEV\(_1\)/FVC ratio ≥ 70%).

EXCLUSION CRITERIA

All patients who did not fulfill the above criteria, those with chronic renal, or hepatic failure, cerebral dysfunction, or those who underwent redo coronary artery bypass graft (CABG) surgery.

Premedication and anaesthesia procedures were standardized and comparable for all patients consisting of weight-dependent dosages of etomidate (0.1–0.2 mg kg\(^{-1}\)), fentanyl (20–40 \( \mu \)g kg\(^{-1}\)), midazolam and pancuronium bromide (0.1 mg kg\(^{-1}\)). In all patients, moderate (28°C) to mild (34°C) core hypothermia was achieved, while antegrade cold crystalloid cardioplegia was used. The same surgical technique was performed on all patients. Immediately after weaning from cardiopulmonary bypass, all patients were ventilated without PEEP. All these patients had a Swan–Ganz catheter inserted into the pulmonary artery via the internal jugular vein to evaluate haemodynamics; blood pressure was monitored by means of a radial arterial catheterization (20 g, Vygon).

Upon admission to the ICU, all patients were ventilated on the assist-control mode with a Siemens Ventilator Servo 900C. They were ventilated at a frequency (\( f \)) of 10 breaths min\(^{-1}\) with a tidal volume (\( V_t \)) adjusted to...
maintain normocarbia ($P_{aCO_2}$ between 35 and 40 mmHg). Fractional inspiratory time % was kept constant (25%), including an end-inspiratory pause 10%. The fraction of inspired oxygen ($FIO_2$) was adjusted to keep a $P_{aO_2}$ around 100 mmHg. When indicated, low doses of propofol (4 mg kg$^{-1}$ h$^{-1}$) or midazolam and morphine (1–2 mg) were administered intravenously for adequate sedation and analgesia.

Patients were randomly assigned to receive zero PEEP (Group A, $n=22$), 5 cmH$_2$O PEEP (Group B, $n=24$), or 10 cmH$_2$O PEEP (Group C, $n=21$) during mechanical ventilatory support. The groups were similar in terms of sex, age, NYHA class, and preoperative left ejection fraction (Table 1).

Blood samples were obtained for arterial and mixed venous gas analysis at 30 min, 4 h and 8 h after application of mechanical ventilation in the ICU, just before extubation (BE), half an hour after extubation (AE), and at 4 h after extubation. $P_{aO_2}$/Fr$O_2$ ratio, mixed venous $P_{vO_2}$ and $S_vO_2$ and cardiac index were measured. Cardiac index was measured by thermodilution using four injections of 10 ml normal saline 0.9% injected at end-expiration, as previously suggested (4).

Mean arterial pressure was maintained between 70 and 90 mmHg, and pulmonary artery wedge pressure between 12 and 15 mmHg, by administration of blood and/or colloids and crystalloids. Blood transfusion was administered when haematocrit was less than 27%. The total amount of any fluid given during this period was recorded for every patient.

Assist-control (A/C) mode followed by synchronized intermittent mandatory ventilation (SIMV) plus pressure support ventilation (PSV) mode when the patient was awake, usually during the 4th or 5th postoperative hour. The weaning procedure for all patients started 6–8 h following ICU admission. The decision for tracheal extubation depended on the existence of the following conditions for at least 2 h: (1) a good consciousness level, (2) a satisfactory tidal volume ($V_t>5$ ml kg$^{-1}$), (3) a respiratory frequency less than 25 breaths min$^{-1}$, (4) arterial pH>7.36, (5) an arterial oxygen tension ($P_{aO_2}$) greater than 80 mmHg, with an inspired oxygen fraction ($FIO_2$) equal or less than 40%, (6) an arterial carbon dioxide tension ($P_{aCO_2}$) less than 45 mmHg, (7) haemodynamic stability, (8) chest drainage less than 50 ml h$^{-1}$, and (9) temperature greater than 36.5°C. Only then, was the patient placed on a PSV mode with 5 cmH$_2$O, for at least half an hour, before extubation.

### Statistical Analysis

Data are reported as the mean ± SD. Statistical analysis was done using two-way analysis of variance (ANOVA) using the BM DP Statistical Software (Los Angeles, CA) (5). A $P$-value <0.05 was considered significant (6). Powers were estimated for one-way (between-groups) ANOVA that omitted consideration of the repeated measure. Alpha error was set at 0.05 and the mean sample size of 22 per group was used in the calculations. Based on the observed findings, the control group score was assumed to be 300 while the common standard deviation was set at 70. Powers were estimated for (1) a 25% improvement in both treatment groups, (2) a 20% improvement in both groups, (3) a 25% improvement in one treatment group and a 12.5% improvement in the other, and (4) improvements of 20% and 10%. Computed powers were $95, 82, 88, and 70$, for estimates 1–4, respectively. Thus, the design appears to have had sufficient power to detect differences between groups if those differences had been as great as 20%–25%.

### Results

Mean $P_{aO_2}$/Fr$O_2$ values at these six time intervals were 320, 317, 322, 325, 277 and 228 mmHg in Group A, 339, 324, 322, 327, 286 and 229 mmHg in Group B, and 321, 319, 305, 315, 285 and 221 mmHg in Group C, respectively. Mean $S_vO_2$ values were found to be 67, 65, 67, 65, 67, 68 and 69 in Group A, 68, 69, 68, 69, 67 and 67 in Group B, and 68, 68, 67, 67, 67 and 67 in Group C, respectively. In addition, mean $P_vO_2$ values were found to be 36, 37, 37, 37, 35 and 35 mmHg in Group A, 36, 37, 36, 37, 36 and 36 in Group C and 35, 35, 36, 37, 36 and 35 mmHg in Group C, respectively. Mean cardiac index values (in 1 min$^{-1}$ m$^{-2}$) were found to be 2.7, 3.1, 3.1, 3.2, 3.2 and 3.4 in Group A, 2.5, 2.8, 2.9, 3, 3.1 and 3.1 in Group B, and 2.6, 3, 3, 3, 3.1 and 3.1 in Group C, respectively.
Mean $PaO_2/FIO_2$, $SVO_2$ and $PV_O2$ values among the three groups of patients at any study interval ($P=\text{n.s.}$). In addition, mean cardiac index values were similar among the groups ($P=\text{n.s.}$). We also found no differences in the duration of mechanical ventilation in these groups ($P=\text{n.s.}$). No pneumothorax was found in any group. Early postoperative atelectasis associated with severe hypoxaemia ($PaO_2/FIO_2 < 100$) needed extensive chest physiotherapy and application of CPAP developed in two patients of Group A (9.1%), in three patients of Group B (12.5%), and in two patients (9.5%) of Group C ($P=\text{n.s.}$). No reintubation or readmission to the ICU was recorded. Mean bypass time was similar in these three groups ($P=\text{n.s.}$). Patients received, during the first postoperative day in both OR and in the ICU, a mean total amount of fluids (blood, blood products, crystalloids and colloids): 5.2 l (Group A), 5.4 l (Group B) and 5.1 l (Group C) ($P=\text{n.s.}$). There were no deaths in any group.

Discussion

All cardiac surgery patients develop acute postoperative hypoxaemia, mainly due to atelectasis in dependent lung areas (1). Magnusson et al. reported recently that atelectasis is the major cause of hypoxaemia and shunt after cardiopulmonary bypass (7). However, the use of cardiopulmonary bypass, the associated systemic inflammatory response syndrome (SIRS) and ischaemia–reperfusion injury in the lung have been reported as minor causes of early postoperative arterial hypoxaemia following open heart surgery. The incidence of atelectasis, particularly in the left lower lobe, following open heart surgery, is well documented (8). The pathogenesis however, is not well defined. Reduced lung volumes (9) and chest wall compliance due to surgery, residual effects of anaesthesia (10), ciliary dysfunction, and incisional pain are possible causes. These patients also have decreased static compliance of the respiratory system (Cst, Rs) postoperatively, which can be explained by reduced lung volume, altered surfactant function due to residual effects of anaesthesia, and atelectasis (5,11). Goyal et al. found an overall decrease in pulmonary function of 30%-40% during the postoperative period in cardiac surgery patients (12).
PEEP introduced in 1967 for the treatment of ARDS, has been applied for the management of acute postoperative respiratory failure. However, various endpoints have been proposed for titration of PEEP (best PEEP), each of which is of unproved clinical benefit, and remain controversial. On the other hand, PEEP application frequently does not produce a proportional increase in PaO₂. Instead, as PEEP is added, PaO₂ does not change or decreases slightly. It has been speculated that optimization of PEEP should include pulmonary recruitment (13).

In this study, we examined the effects of different levels of PEEP on arterial and tissue oxygenation during the early postoperative period after elective coronary artery bypass surgery and found no differences (P=n.s.) between the three groups, at any study interval. This could be explained by the fact that the main cause of postoperative hypoxaemia in cardiac surgery patients is atelectasis. It is recognized that PEEP decreases the size of atelectasis but does not necessarily improve shunt (9) and thus, there is no improvement in arterial oxygenation (14). This is in agreement with the finding that PEEP less than 10 cmH₂O was not effective to reopen atelectatic lung units in cardiac surgery patients (3). Also, PEEP does not usually improve arterial oxygenation in patients under general anaesthesia (15). In addition, PEEP may have a rather modest effect on hypoxaemia due to the systemic inflammatory response syndrome or to ischaemia–reperfusion injury following cardiac surgery.

Ranieri et al. found application of 10 cmH₂O PEEP significantly increased PaO₂ and PaO₂ in ARDS patients (16), but, the aetiology of acute respiratory failure in ARDS is totally different from that of postoperative hypoxaemia.

Although application of PEEP expands the alveoli and may improve oxygenation, extravascular lung water accumulation may be augmented as a result of even moderate levels of PEEP by increase in pulmonary capillary pressure and pulmonary venous resistance. Koganov et al. found in a recent study that pulmonary capillary pressure increased gradually with the increase in PEEP in patients after coronary artery bypass grafting surgery (17). Increased pulmonary venous resistance has been observed in different experimental models of lung injury (18) and cardiopulmonary bypass has been identified as a cause of lung injury. Our findings regarding the non-significant effect of different levels of PEEP on cardiac index in the early postoperative period of cardiac surgery patients is in agreement with the study done by Torelli et al., who found the application of 10 cm PEEP in mechanically ventilated trauma patients compared with ZEEP, did not alter significantly central venous pressure (CVP), cardiac output, or SvO₂ (19). The unchanged cardiac output may be due to volanic expansion reversing the negative effects of PEEP, restoring normal values (20). This is also supported by the finding that alterations of PEEP lead to corresponding changes in pulmonary artery wedge pressure only in low intravascular volumes. On the contrary, at high intravascular volumes, pulmonary artery wedge pressure is independent of, or varies inversely with, changes in the level of applied PEEP (21).

Several studies have examined the effects of PEEP on right ventricular function in humans (22). These effects are complex, variable, and are related to numerous factors associated with underlying cardiorespiratory function, ventilatory parameters, and coronary blood flow. This variable response has been demonstrated in experimental animals and humans, in whom right ventricular end-diastolic pressure and volume have been reported to increase, decrease, or not change as PEEP is administered (23).

PEEP may alter right ventricular function if end-expiratory lung volume increases, through a number of different pathways. Pinsky et al. found no significant differences in cardiac output, heart rate, right ventricular end-diastolic and end-systolic volumes, or right ventricular ejection fraction, in cardiac surgery patients following administration of zero (ZEEP), 5 cmH₂O, and 10 cmH₂O PEEP. They also found lung compliance increased significantly at the transition from 10 to 15 cmH₂O PEEP (24).

Boldt et al. found that haemodynamic changes related to PEEP ventilation are minimal in the intact right ventricle (25), while Schulman et al. found a non-significant decline in cardiac output with increasing PEEP (up to 20 cmH₂O) for the entire population studied (26). On the contrary, Biondi et al. found low levels of PEEP have a predominant preload, reducing effect on the right ventricle (27) and Schuster et al. found decreased cardiac output by PEEP ventilation (28). The effects of PEEP on left ventricular preload, compliance and afterload have been extensively studied, but conflicting results have been reported (29).

In conclusion, low levels of PEEP have no advantage over ZEEP in improving gas exchange and tissue oxygenation during the early postoperative period in elective CABG surgery patients with normal preoperative respiratory status.

References


