Effects of PEEP on Respiratory Mechanics after Open Heart Surgery*

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Respiratory dysfunction, particularly atelectasis, is common after open heart surgery. Routine use of PEEP (5 to 10 cm H2O) in these patients has been advocated. We studied the effects of different levels of PEEP on respiratory mechanics in ten mechanically ventilated open heart surgery patients in the immediate postoperative period. PEEP was studied in increasing increments and decreasing decrements. This procedure was repeated three times. Flow, tidal volume, and airway pressure were measured. We used the rapid airway occlusion technique to determine static compliance of the respiratory system (Cst,rs) and intrinsic PEEP (PPEPI). The changes in end-expiratory lung volume (AEEELV) were measured with respiratory inductive plethysmography. Recruitment of lung units (Vrec) was estimated as the difference in lung volume between PEEP and zero end-expiratory (ZEPP) for the same static inflation pressure (15 cm H2O). We found that respiratory system and on recruitment of lung units in ten open heart surgery patients in the immediate postoperative period.

METHODS AND MATERIALS

Ten patients who were admitted to the intensive care unit (ICU) of Kuopio (Finland) University Hospital after open heart surgery were included in the study. Their entry data are shown in Table 1. All patients were male. Two patients were current smokers and seven were ex-smokers. All patients had normal vital capacity and forced expiratory volume in 1 s as measured preoperatively and none of them had a history of lung disease. The operative procedure was coronary artery bypass in eight patients and aortic valve replacement in two. The protocol was approved by the institutional ethics committee and informed consent was obtained from each patient preoperatively. During the operation, patients were anesthetized with high-dose Fentanyl (0.1 mg/kg) and halothane (up to 3 percent) if needed.

Table 1—Entry Data of Ten Patients*

<table>
<thead>
<tr>
<th>Data</th>
<th>Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>57 ± 3</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>77 ± 3</td>
</tr>
<tr>
<td>Height, cm</td>
<td>174 ± 2</td>
</tr>
<tr>
<td>Tidal volume, L</td>
<td>0.64 ± 0.03</td>
</tr>
<tr>
<td>f, breaths/min</td>
<td>12.2 ± 0.1</td>
</tr>
<tr>
<td>PEEP, cm H2O</td>
<td>5.1 ± 0.1</td>
</tr>
<tr>
<td>Fio2</td>
<td>0.41 ± 0.01</td>
</tr>
<tr>
<td>PaO2, mm Hg</td>
<td>152 ± 14</td>
</tr>
<tr>
<td>(a/A) PaO2</td>
<td>0.61 ± 0.06</td>
</tr>
</tbody>
</table>

*PEEP = positive end-expiratory pressure; f = frequency; Fio2 = fraction of inspired oxygen; PaO2 = arterial PO2; (a/A) PaO2 = ratio of arterial to alveolar PO2.

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Paralysis was achieved by the combination of alcuronium and pancuronium (0.25 mg/kg and 0.15 mg/kg, respectively). Toward the end of the operation, the lungs were inflated with several large tidal volumes and PEEP of 5 cm H₂O was applied for the rest of the operation. After the operation, patients were immediately transported to the ICU where they were maintained in controlled mechanical ventilation.

Patients were studied shortly after warming (peripheral temperature over 31°C) and becoming hemodynamically stable (within 3 to 5 h of admission to the ICU). During the study, patients were kept sedated with diazepam (0.1 to 0.2 mg/kg) and paralyzed with vecuronium bromide (0.1 to 0.2 mg/kg). They were tranorally intubated with cuffed endotracheal tubes (Portex, Portex Ltd; Hythe, Kent, England) (8.0 to 9.0 internal diameter [ID] and 31 cm long) and mechanically ventilated with a ventilator (Siemens Servo 900C, Siemens-Elema, Solna, Sweden). The TV was set at 12 ml/kg and frequency at 12/min with fractional inspiratory time of 35 percent, including an end-inspiratory pause of 10 percent. Other than changing PEEP level, baseline settings were kept constant in each subject throughout the experiment. Special care was taken to avoid gas leaks in the equipments and around the tracheal cuff. To reduce the effects of the compliance and resistance of the system connecting the patients to the ventilator on the mechanics measurements, a single length of standard low-compliance adult tubing was used (2 cm ID, 110 cm long) and the humidifier was omitted from the inspiratory line.

Flow was measured with a heated pneumotachograph (Hans Rudolph Pneumotachometer, model 3700; Hans Rudolph Inc, Kansas City, Mo) inserted via cones between the endotracheal tube and the Y-piece of the ventilator and connected to a differential pressure transducer (Validyne MP 45, ±2.5 cm H₂O; Validyne Co, Northridge, Calif). The response of the pneumotachograph was linear over the experimental range of flows. Volume was obtained by electronic integration of the flow signal (Validyne FV 156-571). Airway pressure (Pao) was measured at the mouth via a side hole proximal to the endotracheal tube by means of a pressure transducer (Validyne MP 45, ±88 cm H₂O). As previously described, changes in end-expiratory lung volume (∆EELV) were obtained using the sum signal of respiratory inductive plethysmography (RIP) (Respiograph, NIMS; Miami Beach, Fla) on the DC coupled mode. The RIP was calibrated against the pneumotachograph with the semiquantitative single-position calibration method. The calibration was validated and accepted only if the error was within 2 percent.

All signals were recorded on an 8-channel pen recorder (Mingograph; Siemens, Solna, Sweden) at a paper speed of 10 mm/s.

Experimental Procedure

Measurements were obtained first at zero end-expiratory pressure (ZEEP). PEEP was then applied in increments of 2 cm H₂O. At each PEEP level, measurements were done after patients were judged to have reached a steady state by stability of the RIP sum signal and Pao signal (usually within 1 min). End-inspiratory occlusions were obtained by pressing the inspiratory hold button of the ventilator for 5 to 6 s. During this period, the contribution of reduction in pressure due to volume loss by continuing gas exchange should be negligible. On reestablishing a steady state (usually within a few breaths), end-expiratory occlusions were done by pressing the end-expiratory hold button of the ventilator for 5 to 6 s. All patients were studied up to a PEEP of 10 cm H₂O. In seven patients, higher levels of PEEP were used (up to 12 to 18 cm H₂O). The above measurements were then performed while PEEP was reduced stepwise to zero in decrements of 2 cm H₂O. The whole procedure was repeated three times. Henceforth, these repetitions will be referred to as inflation-deflation runs 1, 2, and 3.

Data Analysis

End-inspiratory occlusions were followed by a rapid initial drop in the airway pressure followed by a gradual decrease to an apparent plateau. This plateau was reached in less than 5 s and hence the pressure at 5 s was taken to represent the end-inspiratory static elastic recoil pressure of the respiratory system (Pst,rs). Whenever the time required for passive expiration to proceed to completion is greater than the expiratory duration set by the ventilator, the end-expiratory lung volume during mechanical ventilation will exceed the relaxation volume (elastic equilibrium volume) of the respiratory system, and the respiratory system will exert a positive static elastic recoil pressure at end expiration. This pressure is termed intrinsic PEEP (PEEPi). PEEPi was measured with the end-expiratory occlusion technique. If PEEPi is present, Pao increases following airway occlusion until a plateau is reached that corresponds to PEEPi. The static compliance of the total respiratory system (Cst,rs) was calculated by dividing the tidal volume by the end-inspiratory Pst,rs after subtracting PEEP and PEEPi if present.

Change in end-expiratory lung volume due to PEEP (∆EELV) was measured from the RIP sum signal. The drift of the RIP signal in vitro is less than 0.1 ml/min and in vivo under similar conditions it is approximately 1 ml/min. The effect of drift was avoided by using the breaths immediately preceding and those 0.5 to 1 min after the change of PEEP. The cumulative sum of measured ∆EELVs at each PEEP level represents the total change in EELV.

Statistical Analysis

Data are mean ± SE. Statistical analysis was done using two-way analysis of variance (ANOVA) and followed, if significant, by Duncan's test for multiple comparisons. In comparing results obtained at PEEP with those at ZEEP, the paired t test as modified by Dunnett was used. Significance was defined as p<0.05.

Results

At ZEEP, the initial values (ie, before run 1) of PEEPi and Cst,rs of the ten patients were 1.6±1.1 cm H₂O (range, 0.3 to 5.0 cm H₂O) and 60±2 ml/cm H₂O (range, 48 to 78 ml/cm H₂O), respectively. Figure 1 depicts the changes in EELV, Cst,rs, and PEEPi measured at ZEEP after the three subsequent inflation-deflation runs with PEEP. All increased progressively after runs 1 and 2 with no further change after run 3. While Cst,rs increased significantly already after run 1, EELV and PEEPi increased significantly only after run 2. In line with the classic study of Mead and Collier, it is likely that the parallel increase of EELV and Cst,rs after runs 1 and 2 reflects recruitment of previously atelectatic alveoli. Thus, in this case, ∆EELV should reflect recruited lung volume (Vrec). The increase of PEEPi after runs 1 and 2 probably reflects the concomitant increase in Cst,rs and hence a decrease in expiratory driving pressure.

Figure 2 illustrates the relationship between changes in lung volume (∆V) and Pst,rs obtained at end-inspiration and end-expiration in a representative patient at different levels of PEEP during the first stepwise inflation (A) and deflation (B) run with PEEP. Changes in volume are expressed relative to the initial EELV at ZEEP. Up to PEEP of 6 cm H₂O, the data points moved essentially along a fixed line, ie, along a
fixed static volume-pressure (V-P) curve, indicating that recruitment of lung units (if any) was negligible (Fig 2, A). At higher values of PEEP, the data points were displaced progressively upward, suggesting that there was progressive recruitment of previously atelectatic lung units.24 We quantified the "recruited" volume (Vrec) with PEEP as the difference in lung volume between the V-P curves at PEEP and ZEEP at fixed Pst,rs, as described by Ranieri et al.24 We chose Pst,rs of 15 cm H₂O (broken lines in Fig 2) because this allowed us to obtain Vrec in all patients at all levels of PEEP. With increasing PEEP in this patient, there was no appreciable Vrec at PEEP up to 6 cm H₂O because the corresponding data points fell along the ZEEP line. During PEEP decrease (Fig 2, B) the V-P lines moved downward (derecruitment), but at ZEEP did not return to the initial position, reflecting the increase of EELV after run 1 (Fig 1). Similar to the preceding inflation, the data points obtained at PEEP levels between 6 and 0 cm H₂O fell along a single V-P line. In Figure 2, the upward shift of the static V-P-relationships with increasing PEEP (panel A) may be interpreted as recruitment of previ-
inflation runs 2 and 3 were significantly higher than the corresponding values obtained in run 1. The changes in Cst,rs at ZEEP following the three consecutive inflation-deflation runs with PEEP are also depicted in Figure 1.

The values of Cst,rs, Vrec, and ΔEELV during stepwise PEEP decrease were significantly higher at all levels of PEEP compared with those obtained during PEEP increase, as shown in Figure 5 for PEEP of 5 cm H₂O. At this level of PEEP, the values of Cst,rs, Vrec, and ΔEELV obtained in the three runs with decreasing PEEP were not significantly different, although ΔEELV and Vrec tended to be higher during run 1.

**Discussion**

In normal awake supine subjects, Cst,rs amounts to 108 ± 7 ml/cm H₂O. In our patients, lower values were obtained (60 ± 2 ml/cm H₂O), in line with a previous report by Marvel et al. This reduction in Cst,rs can be explained by reduced lung volume postoperatively, altered surfactant function due to residual effects of anesthesia, reduced chest wall compliance due to surgery, and atelectasis.

Postoperative pulmonary atelectasis was first described by Pasteur in 1908. The incidence of atelectasis, particularly in the left lower lobe, after open heart surgery is well documented. The pathogenesis, however, is not well understood. Reduced lung volume and residual effects of anesthesia are possible causes. Further insight concerning atelectasis stems from recent work of Brismar et al and Hedenstierna. In elegant experiments with computed tomography (CT scan), they showed that in supine subjects, atelectasis occurred within 5 min of induction of anesthesia in the dependent parts of both lungs. The atelectasis was most pronounced in the caudal segments of the lung. Application of 10 cm H₂O of PEEP reduced substantially the degree of atelectasis which, however,
was rapidly reinstated after removal of PEEP.

In all three inflation runs, Vrec at PEEP of 5 cm H2O was small and not statistically significant (Fig 3). At PEEP of 10 cm H2O, Vrec amounted to 165 ± 57 ml during inflation run 1, but less during inflation runs 2 and 3 (104 ± 35 ml and 121 ± 41 ml, respectively). In the three patients studied at PEEP of 15 cm H2O, the Vrec amounted to 446 ± 123 ml during inflation run 1 and slightly less during runs 2 and 3.

At ZEEP, EELV increased by the end of run 1 by 109 ± 44 ml, and after run 2 by an additional 32 ± 30 ml (Fig 1). This increase in EELV probably reflects in part an increase in volume due to persistent recruitment of collapsed alveoli, ie, alveoli that have been recruited with increasing PEEP and that remain open upon return to ZEEP. The changes observed in Vrec and ΔEELV during each individual run (Fig 3) are considerably smaller than the cumulative changes. Assuming that the volumes of EELV in Figure 1 reflect entirely recruitment of previously collapsed lung units, at PEEP of 5 and 10 cm H2O during inflation runs 2 and 3, the cumulative Vrec at PEEP of 5 cm H2O should amount to 116 ± 40 ml and 216 ± 29 ml, respectively. At PEEP of 10 cm H2O, the corresponding values would be 245 ± 29 ml and 264 ± 48 ml.

During stepwise decrease of PEEP, Vrec at any given PEEP was higher than during the corresponding inflation with PEEP, especially during run 1 (Fig 5, B). Although part of this difference could be explained by other mechanisms (surface tension phenomena, tissue hysteresis, etc), recruitment of atelectatic lung units was probably paramount because with small tidal volumes, the other mechanisms play a small role, as indicated by a small difference between the static inflation and deflation V-P curves of the respiratory system (ie, static hysteresis) in the absence of atelectasis.

It has been shown that a minimum critical opening transpulmonary pressure (Pt) is needed before any gas could enter gas-free lungs. In humans, this minimum critical opening pressure is about 20 cm H2O. Since atelectasis occurs preferentially in the dependent lung zones, where the end-expiratory values of Pt at ZEEP are around zero in the supine position, the end-inspiratory Pst,rs required to reopen the atelectatic alveoli should exceed 20 cm H2O, considering that Pst,rs includes a component due to the chest wall. In our patients, the initial values of end-inspiratory Pst,rs at ZEEP and PEEP of 5 cm H2O amounted to 17.0 ± 1.3 cm H2O and 20.9 ± 1.2 cm H2O, respectively. Thus, even if we assume that our patients had normal chest wall compliance, their values of static end-inspiratory Pt were necessarily lower than the critical opening pressure needed for recruitment. This probably explains the lack of recruitment at PEEP of 5 cm H2O. In contrast, at PEEP of 10 cm H2O, the initial values of end-inspiratory Pst,rs amounted to 24.6 ± 1.0 cm H2O, which was probably sufficient to reopen collapsed alveoli (Fig 3). At PEEP of 15 cm H2O, end-inspiratory Pst,rs in three patients ranged between 28 and 35 cm H2O, and resulted in a further substantial increase in Vrec (Fig 3). Clearly, higher PEEP values than the recommended prophylactic PEEP (5 to 10 cm H2O) should be more effective in terms of recruitment. The adverse effects of high PEEP, however, on ventricular performance and cardiac output (and hence oxygen transport) are well recognized.

With increasing PEEP level, there was a progressive increase in EELV. At low levels of PEEP, the changes in EELV occurred along a fixed static V-P relationship (up to 6 cm H2O in Fig 2, A). At higher levels of PEEP, however, there was an upward shift of the V-P curves, indicating that part of the increase in EELV reflected recruitment. Due to the persistent increase in EELV after runs 1 and 2, the EELV at any given level of

![Figure 5. Average values of static compliance of the respiratory system (Cst,rs), recruited alveolar volume (Vrec), and changes in end-expiratory lung volume (ΔEELV) obtained at PEEP of 5 cm H2O during inflation (filled bars) and deflation (open bars) runs I, II, and III in ten subjects. Bars = 1 SE. P values refer to comparisons between inflation and deflation. Asterisk = p<0.05; two asterisks = p<0.01.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21649/)
PEEP was higher in run 3 than in runs 1 and 2.

It should be noted that in all patients, the EELV at ZEEP was higher than the elastic equilibrium volume of the respiratory system because of presence of PEEFi (range, 0.3 to 5.0 cm H2O) (Fig 2). The values of PEEFi were much lower than those commonly observed in mechanically ventilated patients with chronic obstructive pulmonary disease (up to 22 cm H2O). Nevertheless, PEEFi in our patients had to be taken into account for the correct measurements of the Cst,rs. In the two patients with the highest PEEFi (4.6 and 5.0 cm H2O), the difference between the corrected and uncorrected Cst,rs was 22 and 24 percent at initial ZEEP. There are no previous measurements of PEEFi in postoperative open heart surgery patients, and the nature of PEEFi exhibited by our patients is not clear. It should be noted, however, that nine of our patients had a smoking history. Furthermore, our baseline ventilation included an end-inspiratory pause of 10 percent of the total cycle duration, which, according to a previous report, should have promoted the development of PEEFi.

In conclusion, the main findings of this study are as follows: (1) In postoperative open heart surgery patients, high values of PEEP appear to be potentially useful for reopening atelectatic lung units. (2) In the immediate period after open heart surgery, static compliance of the respiratory system is lower than normal, probably reflecting the presence of atelectasis. (3) The application of prophylactic PEEP of 5 cm H2O results in virtually no recruitment of collapsed alveoli. By contrast, PEEP of 10 cm H2O elicits significant alveolar recruitment. However, at PEEP of 10 cm H2O, not all of the atelectatic alveoli are recruited because higher values of PEEP caused a further substantial increase in Vrec. Although high levels of PEEP are effective in terms of recruitment, there may be adverse circulatory effects and hence high levels of PEEP are contraindicated. An alternative approach is to perform periodically large sustained lung inflations. In this connection, it should be noted that in spontaneously breathing postoperative patients, periodic deep breaths, in the form of incentive spirometry, are an effective and common modality for treating atelectasis. (4) In our patients, application of PEEP produced an increase in EELV and Cst,rs, at ZEEP, which probably reflects a persistent reopening of atelectatic lung units. This finding is consistent with the observations of Mead and Collier and Bendixen et al, who found that lung compliance increased immediately after forced inflation of the lungs, and attributed it to recruitment of previously closed lung units. Based on that study, the use of periodic inflations with large tidal volume (sighs) was adopted during mechanical ventilation. Recently, however, with the administration of large tidal volumes (10 to 15 ml/kg) and PEEP, the use of sighs has been generally abandoned. Our study suggests that even when relatively large tidal volumes are used (in the order of 12 ml/kg in our patients) in association with low levels of PEEP, large periodic inflations (sighs) might be beneficial in reopening atelectatic units. This, however, needs to be confirmed in further clinical investigations.

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