Increase in lung volume originated by extrinsic PEEP in patients with auto-PEEP

The role of static lung complianc e

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Received: 30 August 1991; accepted: 13 April 1992

Abstract. The use of extrinsic positive end expiratory pressure (PEEPe) in patients with auto-PEEP (AP) can reduce the respiratory work during weaning from mechanical ventilation. However, the application of PEEPe can produce a certain level of hyperinflation, an undesirable effect which can limit the efficacy of the reduction of respiratory work. The objective of the present study has been to determine if the increase in end expiratory lung volume (EELV) originated by the PEEPe is related to static lung compliance (SLC). We have studied 14 patients on mechanical ventilation in whom an AP of between 4 and 12 cmH2O was detected. On applying PEEPe equal to half the AP, the EELV increased slightly (77 ± 64 ml) and was not related to pulmonary compliance. When PEEPe equal to the AP was applied, the EELV increased by 178 ± 110 ml (range 45–375 ml, p < 0.05), and there was a significant correlation with SLC (r = 0.659, p < 0.05). In conclusion, the application of PEEPe equal to the AP causes a moderate increase in EELV. However, in patients with high pulmonary compliance this increase can be more important and must be taken into account when considering the use of PEEPe during weaning.

Key words: Mechanical ventilation — Auto-PEEP — Pulmonary compliance

The presence of positive end expiratory pressure not applied externally or auto-PEEP (AP), is a frequent and well-known phenomenon found both in patients receiving mechanical ventilation [1, 2] and in patients with chronic obstructive pulmonary disease (COPD) who are breathing spontaneously [3].

In patients weaning from mechanical ventilation, the presence of AP can contribute to respiratory muscle fatigue since for each spontaneous inspiration, the patient must generate a pressure of greater magnitude but opposite in sign to the alveolar pressure before any air intake is achieved. This involves an additional effort and may contribute to weaning failure. In this context, the application of extrinsic PEEP (PEEPe) similar to the AP can reduce the respiratory work by suppressing the pressure gradient between the alveoli and the exterior [4, 5]. The efficacy of this manoeuvre is optimal if the application of PEEPe originates no increase in lung volume, but given that the pulmonary parenchyma is not a homogeneous structure and that the commonest method of measuring AP (occluding the expiratory port of the respirator) measures the average AP, the application of a PEEPe equal to the AP can distend pulmonary zones with a below average AP, and so produce a certain level of hyperinflation which worsens the strength-length relationship of respiratory musculature [6, 7]. Data in this respect are scarce, although they generally show that the increase in end expiratory lung volume (EELV) is small [4, 5] but variable [8]. Among the factors governing the magnitude of this increase in EELV, it seems that the absence of airflow limitation may play an important part [8], however in our opinion, other factors like the quantity of zones with low AP as well as the compliance of such zones, can influence the increase of EELV. Given that the static lung compliance (SLC) is an easily quantifiable parameter, if it proved useful as a predictor of increases in EELV originated by PEEPe in patients with AP, it would be a valuable clinical aid. Therefore the objective of the present study was to find out if the increase in EELV originated by PEEPe in patients with AP is related to SLC.

Materials and methods

Fourteen patients receiving mechanical ventilation for different reasons (Table 1) and who were in stable haemodynamic condition were included in the present study. No patient was in the weaning phase. All patients were sedated with a continuous infusion of midazolam or midazolam plus morphine; 6 patients were paralyzed with pancuronium or atracurium; the remainder of the patients were paralyzed with atracurium i.v. for the purpose of the study. Mechanical ventilation was provided using Drager Evita or Bear-1 ventilators at FiO2 of between 0.3 to 0.7, and 5 patients were ventilated with PEEP (5–9 cmH2O). At the time of the study, all patients had a PaO2 above 60 mmHg. Ventilator settings were established by the primary physicians according to the
tion, a pneumotachograph (Jaeger Screenmate-Box, Würzburg, Ger-
many) was fitted in the expiratory port of the respirator, behind the expira-
tory valve. After each expiration, when no flow is detected by the
graph fitted in the expiratory port of the respirator, the airway
pressure was recorded by a manometer (Siemens-Elema) inter-
posed laterally between the syringe and the tracheal tube. The syringe
was prolonged for between 15–25 seconds, so that an above normal ex-
piratory volume was obtained, since the gas causing the AP had been
removed or the flow is so low, that the pneumotachograph returns to the
base line before all the gas has been recorded. In these cases, b does not
represent the entire volume of gas originating the AP, while the sum of
c+d+e does indeed reflect more accurately the increase in lung volume
(AEELV-AP = c+d+e, Fig. 1).

The measurement of AP without PEEPe was made by occluding the
expiratory port of the respirator at the end of the expiration and by
reading the pressure registered by a manometer (Siemens-Elema) previ-
ously interposed laterally between the tracheal tube and the respirator
circuits. This expiration was prolonged, reducing the frequency of the
respirator or changing the ventilation mode to spontaneous to avoid the
respirator sending a new inflation. The procedure was repeated twice
more, allowing 8–10 normal respirations before each measurement.
The median figure, to the nearest whole number, was taken as the pa-
tient's clinical situation. The respiratory rate varied between 12–18
breaths per min and the I:E ratio was below 1:1 in 8 patients, in 4 pa-
tients was 1:1 and was above 1:1 in 2 (1.5:1 and 1.59:1).

Procedure

Once the patient was sedated and relaxed, the Fio2 was increased suf-
ficiently to guarantee acceptable levels of PaO2, and if the patient was
on PEEP, this was removed 10–15 min afterwards. Then the AP and
the increase in EELV caused by the AP (AEELV–AP) were measured
(see below). A PEEPe equal to half the AP was then applied and the
AP and the increase in EELV caused by the application of this PEEPes
were again measured. Finally, a PEEPe equal to the initial AP was ap-
plied, and again, the AP and the increase in EELV caused by PEEPe
were measured. Minute ventilation was left unchanged during the pro-
cedure. In no case did the study take more than 1 h.

The measurement of AP without PEEPe was made by occluding the
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respirator or changing the ventilation mode to spontaneous to avoid the
respirator sending a new inflation. The procedure was repeated twice
more, allowing 8–10 normal respirations before each measurement.
The median figure, to the nearest whole number, was taken as the pa-
tient’s AP. In patients with an AP of 10 cmH2O or more, the maxi-

Table 1. Clinical characteristics of the patients

| No. | Sex | Age (yr) | SLC | AP | Diagnosis                        |
|-----|-----|---------|-----|----|----------------------------------|
| 1   | M   | 59      | 94  | 5  | Severe head trauma               |
| 2   | M   | 71      | 98  | 5  | Multiple trauma, COPD            |
| 3   | F   | 73      | 21  | 12 | Cerebellar hematoma, COPD        |
| 4   | M   | 44      | 77  | 7  | Multiple trauma, FE, ARDS        |
| 5   | M   | 23      | 25  | 9  | Multiple trauma, ARDS            |
| 6   | M   | 64      | 68  | 7  | SP-posterior fossa tumor, COPD   |
| 7   | M   | 33      | 84  | 7  | Severe head trauma               |
| 8   | F   | 59      | 64  | 7  | Sp-acute neuroma, pneumonia      |
| 9   | M   | 64      | 64  | 6  | Subarachnoid hemorrhage, COPD    |
| 10  | M   | 30      | 71  | 6  | Tetanus, ARDS, HIV(+)            |
| 11  | M   | 23      | 30  | 6  | Thoracic trauma, ARDS            |
| 12  | M   | 26      | 31  | 4  | Multiple trauma, ARDS            |
| 13  | F   | 69      | 81  | 8  | Respiratory infection, hemiplegia|
| 14  | M   | 71      | 41  | 8  | Status epilepticus, COPD         |

SLC, Static lung complianc; AP, auto-PEEP; COPD, chronic obstructive pulmonary disease; ARDS, adult respiratory distress syn-
drome; FE, fat embolism; SP, status postoperative; HIV(+), human immunodeficiency virus seropositive; P-CVA, post cerebral vascular accident

Fig 1. Graph showing method of calculating the volume of gas trapped by the AP. The arrows in the upper and lower part of the figure indicate the direction of the recorder (right to left). Note that no inspired volume is recorded. Only expired volume is recorded with the pneumotachograph fitted in the expiratory port of the respirator, behind the expirato-
ry valve. After each expiration, when no flow is detected by the pneumotachograph, the recording pen automatically returns to the base line. a, expired volume; b, increase in the expired volume on prolonging the expiration; c, d and e, the volumes of gas trapped on restarting me-
chanical ventilation. When ventilation is restarted, the first expirations are of smaller volume than normal, since part of the gas remains trap-
ed in the lungs and originates the AP. Normally b = c+d+e, but on occasions, when expiration is prolonged, the escape of gas is momentarily inter-
rupted or the flow is so low, that the pneumotachograph returns to the base line before all the gas has been recorded. In these cases, b does not represent the entire volume of gas originating the AP, while the sum of c+d+e does indeed reflect more accurately the increase in lung volume responsible for the AP.
was previously filled with oxygen and if any pressure was recorded on connecting it to the tracheal tube (auto-PEEP), it was disconnected to allow the escape of gas trapped [9], and was reconnected to the patient several seconds later. The SLC was calculated as the relationship of inflated volume to the pressure reached.

The statistical analysis was carried out using analysis of variance to compare the variations induced by sequential changes in PEEPe. Coefficients of correlation were used to compare the different increases in EELV and SLC.

**Results**

Before applying PEEPe the value of AP found was 6.8±2 cmH2O. On applying a PEEPe equal to half the AP (3.7±0.9), the value of AP fell to 5.1±2.3 cmH2O, and on applying a PEEPe equal to the AP, it fell to 2.8±1.3 cmH2O (p>0.05) (Table 2).

The increase in EELV originated by AP was 329±155 ml. On applying a PEEPe equal to half the AP, the EELV increased by 77±64 ml (Fig. 2), an increase in EELV per cmH2O of PEEPe of 21±19 ml/cmH2O. On applying a PEEPe equal to the AP, the EELV increased by 178±110 ml (p>0.05), an increase in EELV per cmH2O of PEEPe of 27±15 ml/cmH2O.

The SLC was 63.4±26 ml/cmH2O. We have found no statistical significance in the correlation of SLC with the increase in EELV originated by the AP or by an application of PEEPe equal to half the AP. By contrast, the correlation between the SLC and the increase in EELV on applying a PEEPe equal to the AP was statistically significant (r=0.659, p>0.05) (Fig. 3). The correlation was even more significant when SLC was related to increases in EELV per cmH2O of PEEPe (r=0.7358, p>0.01) (Fig. 4).

**Discussion**

In the present study we have found that in patients with AP, the application of a PEEPe equal to half the AP generally produces a negligible increase in EELV. However, when the level of applied PEEPe is similar to the AP, the increase in EELV is more important and is closely related to the SLC. In figs. 5 and 6, the influence of SLC in lung volume variations can be seen on the application and subsequent removal of increasing levels of PEEPe. Figure 5 concerns a patient with an AP of 6 cmH2O and a SLC of 71 ml/cmH2O in whom the application of 3 and 6 cmH2O of PEEPe originated an increase in lung volume of 145 and 270 ml respectively. In contrast, in fig. 6 can be seen the record for patient with a SLC of

| Table 2. Results obtained at different levels of PEEPe |
|------------------------------------------------------|
| Without PEEPe | PEEPe = 1/2 AP | PEEPe = AP |
| PEEPe (cmH2O) | 0.64±0.6 (a) | 3.7±0.9 | 6.7±2 |
| AP (cmH2O)    | 6.8±2          | 5.1±2.3 | 2.8±1.3* |
| ΔEELV-AP (ml) | 329±155        | 77±64   | 178±110* |
| Peak AW-P (cmH2O) | 33.9±6.2 | 35±6 | 37±5.9* |
| Plateau AW-P | 26±6.4         | 27.8±5.8 | 28±5.5 |

(a), Value corresponding to the PEEPe imposed by some respirators without having been prescribed. This has occurred in 6 patients (range between 0.5 and 1.5 cmH2O)

ΔEELV-AP, Volume of gas trapped by the AP obtained by prolonging the expiratory time (as described in Fig. 1); ΔEELV-PEEPe, Volume of gas trapped by the PEEPe, calculated as the difference between the expiratory volume during ventilation with PEEPe and the following 3–4 expirations after removal of PEEPe; AW-P, airway pressure. *P<0.05 between the situation without PEEPe and PEEPe = AP

[Fig. 2. Increase in EELV in each patient on the application of PEEPe equal to half the AP and of PEEPe equal to the AP. p<0.05 between the latter and the base situation. ▲ = COPD; △ = ARDS; □ = other diagnoses]

[Fig. 3. Correlation between static lung compliance and the increase in EELV on the application of PEEPe equal to the AP. r=0.659; p<0.05; y=20.85+2.09X. ▲ = COPD; △ = ARDS; □ = other diagnoses]
Fig. 4. Correlation between static lung compliance and the increase in EELV per cmH₂O of PEEPe on the application of PEEPe = AP. 
\[ r = 0.7358; \ p < 0.01; \ y = 1.85 + 0.38 \times \]

\[ \Delta = \text{COPD}; \ \Delta = \text{ARDS}; \ \square = \text{other diagnoses} \]

Fig. 4. Correlation between static lung compliance and the increase in EELV per cmH₂O of PEEPe on the application of PEEPe = AP. The arrows in the upper and lower part of the record indicate the direction of the graph (right to left). Recorder speed is 25 s/cm. In A, the patient is being ventilated with a PEEPe of 4 cmH₂O. On removing the PEEPe (arrow), a slight variation in the expired volume is produced, which corresponds to the volume trapped in the circuits by the PEEPe (3.7 ml/cmH₂O of PEEPe). In B, the patient is being ventilated with PEEPe. The large arrow indicates the reduction in expired volume on applying PEEPe of 9 cmH₂O, and the small arrow indicates the increase in expired volume on removing the PEEPe.

25 ml/cmH₂O and AP of 9 cmH₂O in whom the application of 4 cmH₂O of PEEPe produced no increase in lung volume (the minimum variation to be seen is due to the distensibility of circuits, 3.7 ml/cmH₂O); and the application of a PEEPe of 9 cmH₂O produced a slight increase in lung volume of only 45 ml.

Other authors have found that the application of PEEPe in patients with AP produces slight increases in
apnea. This difference in behaviour is because in the case of oxygen, the alveolar-capillary pressure gradient is maintained by the continuous renewing of capillary blood. By contrast, in the case of CO₂, as the alveolar gas is not renewed, the alveolar CO₂ pressure rapidly equilibrates with the venous CO₂ pressure and the inflow of CO₂ to the lung is consequently minimal. This difference could have had an unequal influence on the determination of EELV–AP, having a proportionally greater effect on determinations from patients with less gas volume trapped by AP.

As other authors have previously described [4, 5], the application of PEEPe in patients with AP causes a progressive reduction of the AP. However, although the AP diminishes, the effective PEEP at the alveolar level (AP+PEEPe) increases slightly, in our case from 6.8±2 to 9.7±3 (p<0.001), which doubtless contributes to the increase in EELV. In the same way, the increase in airways pressure has been lower than expected on adding PEEPe, a result previously noted by other authors [4].

We have found no relationship between the volume of gas trapped by PEEPe and the etiology of the respiratory failure, since as can be seen in figs. 2, 3 and 4, patients with a similar base illness show a wide variation in their increase in EELV. By contrast, SLC has indeed been a reliable predictor of the increase in EELV independent of patient diagnosis. One factor to take into account is that our study has been carried out with sedated and relaxed patients who therefore have no muscular tone. It could be thought that the results would be different in patients with spontaneous breathing, since in these circumstances, the increase in EELV may originate an increase in expiratory muscle activity [11], which would diminish the volume of trapped gas. However, Petrof et al. [5], in a group of 7 patients with an AP of 9.9±1.1 cmH₂O who were in spontaneous breathing during weaning, found that the application of continuous positive airway pressure (CPAP) of 10 cmH₂O produced an increase in EELV of 242±55 ml, which is equivalent to 24 ml/cmH₂O of CPAP, an increase similar to that which we have found (27 ml/cmH₂O of PEEPe). Therefore it is to be expected that in patients breathing spontaneously, SLC would also be a good predictor of the level of hyperinflation originated by PEEPe.

In conclusion, the application of a PEEPe equal to half the AP produces a minimal increase in EELV. However, on applying a PEEPe equal to the AP, the increase is more important, especially in patients with a high pulmonary compliance. This must be taken into account when considering its use, carefully balancing the benefits of reducing respiratory work against the possible negative effects of a moderate hyperinflation.

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