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

The role of static lung complicance

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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 encrease 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 cmH_2O 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 increace 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 scare, 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 FiO_2 of between 0.3 to 0.7, and 5 patients were ventilated with PEEP (5–9 cmH_2O). At the time of the study, all patients had a PaO_2 above 60 mmHg. Ventilator settings were established by the primary physicians according to the

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	5	Multiple trauma, FE, ARDS
5	M	25	25	9	Multiple trauma, ARDS
6	F	64	68	7	SP-posterior fossa tumor, COPD
7	M	33	84	7	Severe head trauma
8	F	59	64	7	Sp-acoustic neuroma, pneumonia
9	M	64	64	6	Subarachnoid hemorrage, 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 P-CVA, COPD
14	M	71	41	8	Status eplilepticus, COPD

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

patients' 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 patients 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 FiO_2 was increased sufficiently to guarantee acceptable levels of PaO_2, 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 (ΔEELV–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 PEEPe were again measured. Finally, a PEEPe equal to the initial AP was applied, and again, the AP and the increase in EELV caused by PEEPe were measured. Minute ventilation was left unchanged during the procedure. In no case did the study take more than 1 h.

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) previously 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 patient's AP. In patients with an AP of 10 cmH_2O or more, the maximum difference tolerated between the three measurements was 2 cmH_2O. In patients with an AP below 10 cmH_2O, the maximum difference tolerated between the three measurements was 1 cmH_2O. If these limits were exceeded the measurement was repeated until the level of AP could be measured in accordance with this protocol.

After the measurement of AP and with the same pattern of ventilation, a pneumotachograph (Jaeger Screenmate-Box, Würzburg, Germany) was fitted in the expiratory port of the respirator, behind the expiratory valve, and the expired volume was measured by electrical integration of the flow signal and recorded using a Hewlett Packard 7045 A X-Y recorder. After recording at least 10 normal expirations, an expiration was prolonged for between 15–25 seconds, so that an above normal expiratory volume was obteined, since the gas causing the AP had been allowed to escape (b in Fig. 1). When ventilation was restarted, the first 3–4 expirations were below normal due to the gas trapping caused again by the AP (c, d and e in Fig. 1). The volume of gas trapped by the AP (ΔEELV–AP) was measured as the sum of the differences between the normal expiratory volume and each of the expirations below normal that appeared on restarting mechanical ventilation (ΔEELV–AP = c+d+e, Fig. 1).

AP during ventilation with PEEPe was measured as the difference between the airway pressure obtained on occluding the expiratory port at the end of expiration and the PEEPe.

The increase in EELV caused by the PEEPe (ΔEELV–PEEPe) was calculated as the difference between the expiratory volume during ventilation with PEEPe and the following 3–4 expirations after removal of PEEPe. This value was corrected by substracting the volume trapped in the circuits by the PEEPe (3.7 ml/cmH_2O). This figure was previously calculated using the expired volume/airway pressure ratio on allowing a tidal volume while keeping the patient connection closed. The expired volume obtained in these conditions is a consequence of the circuit's distensibility. This expired volume was measured using a Wright Spirometer fitted in the expiratory port of the respirator, and the airway pressure was measured with the respirator manometer. The value of 3.7 ml/cmH_2O was the mean of the measurements with the two types of single use circuits used (range 3.1–4 ml/cmH_2O).

All measurements of increases in EELV caused by AP or PEEPe were carried out twice and were considered valid if there was not a difference greater then 15% between them. The final result was the average of both measurements.

Given that the level of PEEPe applied varied from one patient to another according to their AP, the value of the increase in EELV was also calculated in relationship to the amount of PEEPe applied (increase in EELV per cmH_2O of PEEPe).

SLC was measured by connecting a 1 l syringe to the tracheal tube and manually inflating a volume of 10 ml/kg in not less than 30 s. The pressure reached was recorded by a manometer (Siemens-Elema) interposed laterally between the syringe and the tracheal tube. The syringe

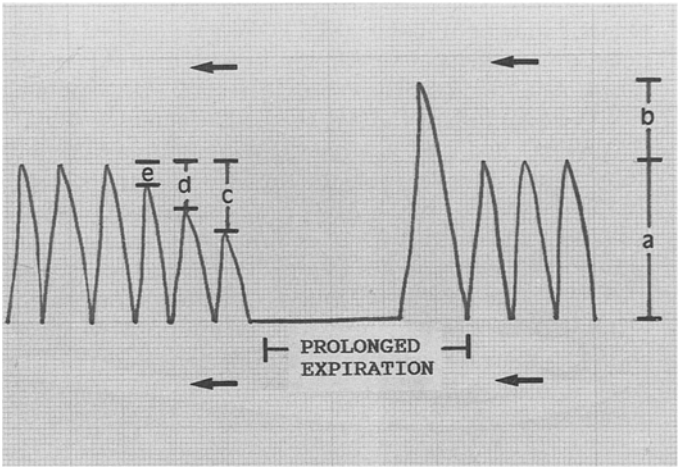

Fig 1. Graph showing method of calculating the volume of gas trapped by the AP. The *arrows* in the upper and lower part of the figure indicate the direction of the recorder (right to left). Note that no inspired volume is recorded. Only expired volume is recorded with the pneumotachograph fitted in the expiratory port of the respirator, behind the expiratory 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 mechanical ventilation. When ventilation is restarted, the first expirations are of smaller volume than normal, since part of the gas remains trapped in the lungs and originates the AP. *c*, *d* and *e* represent together the volume of gas causing the AP. Normally $b = c+d+e$, but on occasions, when expiration is prolonged, the escape of gas is momentarily interrupted 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 cmH_2O. On applying a PEEPe equal to half the AP (3.7 ± 0.9), the value of AP fell to 5.1 ± 2.3 cmH_2O, and on applying a PEEPe equal to the AP, it fell to 2.8 ± 1.3 cmH_2O ($p > 0.05$) (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 cmH_2O of PEEPe of 21 ± 19 ml/cmH_2O. On applying a PEEPe equal to the AP, the EELV increased by 178 ± 110 ml (Fig. 2) ($p > 0.05$), an increase in EELV per cmH_2O of PEEPe of 27 ± 15 ml/cmH_2O.

The SLC was 63.4 ± 26 ml/cmH_2O. 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 cmH_2O 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 cmH_2O and a SLC of 71 ml/cmH_2O in whom the application of 3 and 6 cmH_2O 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 (cmH_2O)	0.64 ± 0.6 (a)	3.7 ± 0.9	6.7 ± 2
AP (cmH_2O)	6.8 ± 2	5.1 ± 2.3	2.8 ± 1.3*
ΔEELV-AP (ml)	329 ± 155	–	–
ΔEELV-PEEPe (ml)	–	77 ± 64	178 ± 110*
Peak AW-P (cmH_2O)	33.9 ± 6.2	35 ± 6	37 ± 5.9*
Plateau AW-P (cmH_2O)	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 cmH_2O)

ΔEELV-AP, Volume of gas trapped by the AP obtained by pro longing 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

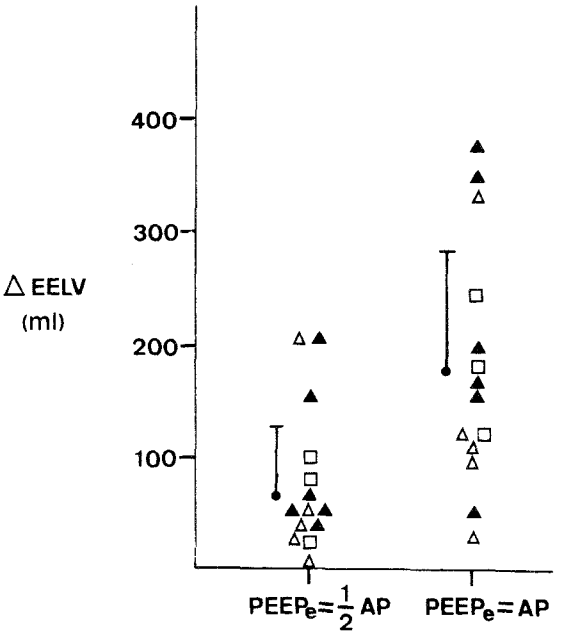

Fig. 2. Increase in EELV in each patient on the application of PEEPe equal to half the AP and of PEEPe equal to the AP. $p < 0.05$ between the latter and the base situation. ▲ = COPD; △ = ARDS; □ = other diagnoses

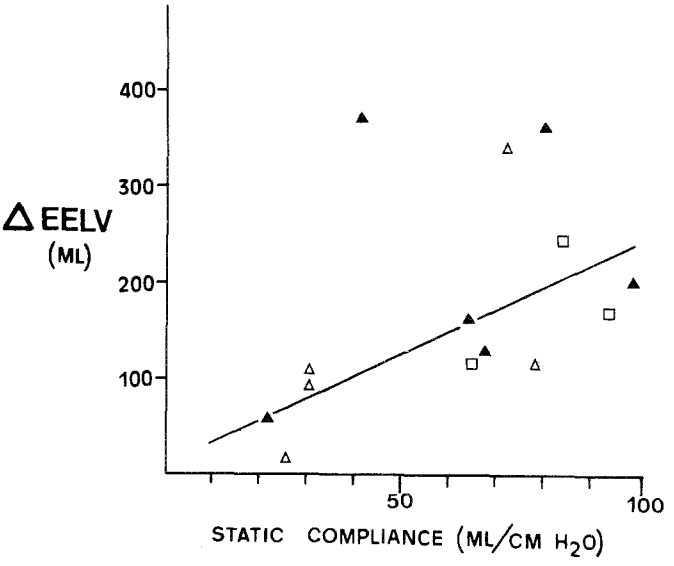

Fig. 3. Correlation between static lung compliance and the increase in EELV on the application of PEEPe = AP. $r = 0.659$; $p < 0.05$; $y = 20.85 + 2.09\,X$. ▲ = COPD; △ = ARDS; □ = other diagnoses

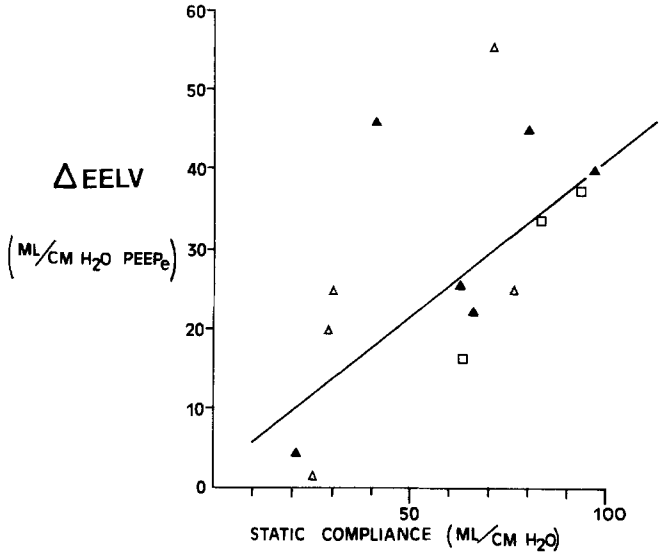

Fig. 4. Correlation between static lung compliance and the increase in EELV per cmH_2O of PEEPe on the application of PEEPe = AP. $r = 0.7358$; $p < 0.01$; $y = 1.85 + 0.38\,X$. ▲ = COPD; △ = ARDS; □ = other diagnoses

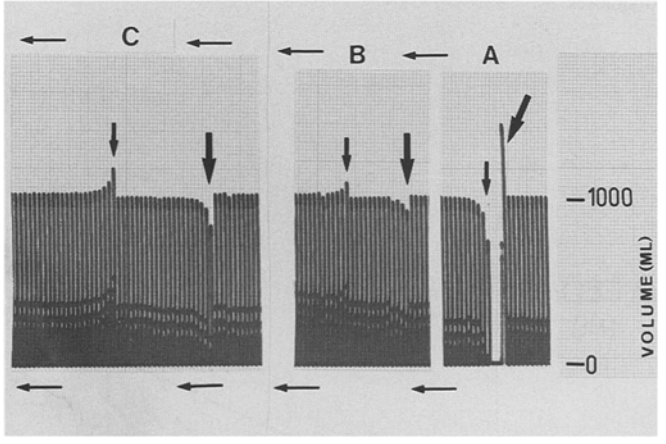

Fig. 5. Record of expiratory volume of patient number 10, in which an auto-PEEP of 6 cmH_2O and a static lung compliance of 71 ml/cmH_2O were measured. The *arrows* in the upper and lower part of the figure indicate the direction of the recorder (right–left). Recorder speed is 25 s/cm. In *A*, the *large arrow* shows the increase in expiratory volume on prolonging the expiration, which was causing an AP of 6 cmH_2O. The *small arrow* shows how on restarting mechanical ventilation, the four following expirations are below normal as a consequence of the gas trapped by the AP. In *B*, the *large arrow* shows the reduction in expiratory volume on applying 3 cmH_2O of PEEPe. The *small arrow* shows the increase in expiratory volume on removing the PEEPe of 3 cmH_2O. In *C*, we can see the reduction in expiratory volume on applying a PEEPe of 6 cmH_2O (*large arrow*) and the increase in expiratory volume on removing the PEEPe of 6 cmH_2O

25 ml/cmH_2O and AP of 9 cmH_2O in whom the application of 4 cmH_2O of PEEPe produced no increase in lung volume (the minimum variation to be seen is due to the distensibility of circuits, 3.7 ml/cmH_2O); and the application of a PEEPe of 9 cmH_2O 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

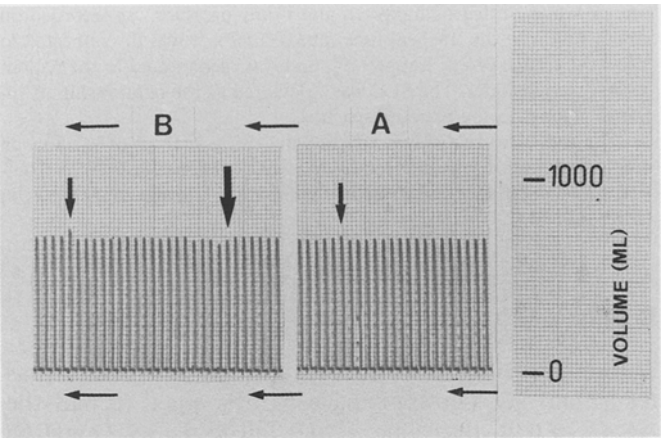

Fig. 6. Record of expiratory volume of patient number 5 who presented a static lung compliance of 25 ml/cmH_2O and an AP of 9 cmH_2O. The *arrows* in the upper and lower part of the record indicate the direction of the graph (right to left). Recorder speed is 25 s/cm. In *A*, the patient is being ventilated with a PEEPe of 4 cmH_2O. 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/cmH_2O of PEEPe). In *B*, the patient is being ventilated without PEEPe. The *large arrow* indicates the reduction in expired volume on applying PEEPe of 9 cmH_2O, and the *small arrow* indicates the increase in expired volume on removing the PEEPe

lung volume [4, 5, 8]. Gay et al. studied the effect of the patient's expiratory flow pattern on the increase in EELV originated by the PEEPe. From the data shown in their results table, it can be seen that the increase in EELV is extraordinarily variable from one patient to another, going from 130 ml/cmH_2O of PEEPe to 11.6 ml/cmH_2O of PEEPe. They found that in patients not presenting airflow limitation, any level of PEEPe produces an increase in EELV. On the other hand, in those patients with airflow limitation an increase in EELV is only seen when the PEEPe is increased to levels similar to those of the AP. We have not studied the expiratory flow pattern of our patients, but the moderate dispersal of some of our results can probably be related to the expiratory flow pattern. However, SLC has been a good indicator of the increase in lung volume originated by a PEEPe equal to the AP.

Contrary to expectations, we have found no significant relationship between the volume of gas liberated on prolonging the expiration (the volume of gas generating the AP) and SLC. The cause of this lack of relationship is perhaps the fact that the act of prolonging expiration does not necessarily guarantee the escape of all the gas that is originating the AP. It is possible that in some alveolar zones, the AP is contributing to their stabilisation, so that the escape of part of this gas may destabilise them, provoking an airways collapse which prevents its complete emptying. Another factor which could have partly affected the determination of the volume of gas trapped by the AP is the difference between the oxygen consumption and the CO_2 inflow into the lungs during the period of apnea on the prolongation of the expiration [10]. It is known that during apnea, the CO_2 inflow to the lung is very small, while in contrast the oxygen uptake from the lung continues at a similar rate as prior to the

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 CO_2, as the alveolar gas is not renewed, the alveolar CO_2 pressure rapidly equilibrates with the venous CO_2 pressure and the inflow of CO_2 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 cmH_2O who were in spontaneous breathing during weaning, found that the application of continuous positive airway pressure (CPAP) of 10 cmH_2O produced an increase in EELV of 242 ± 55 ml, which is equivalent to 24 ml/cmH_2O of CPAP, an increase similar to that which we have found (27 ml/cmH_2O 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.

References

1. Pepe PE, Marini JJ (1982) Occult positive end expiratory pressure in mechanically ventilated patients with airflow obstruction. The auto-PEEP effect. Am Rev Respir Dis 126:166–170
2. Broseghini C, Brandolese R, Poggi R, Polese G, Manzin E, Milic-Emili J, Rossi A (1988) Respiratory mechanics during first day of mechanical ventilation in patients with pulmonary edema and chronic airway obstruction. Am Rev Respir Dis 138:355-361
3. Haluszka J, Chartrand DA, Grassino AE, Milic–Emili J, (1990) Intrinsic PEEP and arterial PCO2 in stable patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 141:1194–1197
4. Smith TC, Marini JJ (1988) Impact of PEEP on lung mechanics and work of breathing in severe airflow obstruction. J Appl Physiol 65:1488–1499
5. Petrof BJ, Legaré M, Goldberg P, Milic–Emili J, Gottfried BS (1990) Continuous positive airway pressure reduces work of breathing and dyspnea during weaning from mechanical ventilation in severe chronic obstructive pulmonary disease. Am Rev Respir Dis 141:281–289
6. Roussos C, Macklem PT (1982) The respiratory muscles. N Eng J Med 307:786–797
7. Penguelly LD, Alderson AM, Milic-Emili J (1971) Mechanics of the diaphragm. J Appl Physiol 30:797–895
8. Gay PC, Rodarte JR, Hubmayr RD (1989) The effects of positive expiratory pressure on isovolume flow and dynamic hyperinflation in patients receiving mechanical ventilation. Am Rev Respir Dis 139:621–626
9. Fernandez R, Mancebo J, Blanch Ll, Benito S, Calaf N, Net A (1990) Intrinsic PEEP on static pressure-volume curves. Intensive Care Med 16:233–236
10. Gattinoni L, Mascheroni D, Basilico E, Foti G, Presenti A, Avalli L (1987) Volume/pressure curve of total respiratory system in paralysed patients: artefacts and correction factors. Intensive Care Med 13:19–25
11. Bishop B, Hirsh J, Thursby M (1978) Volume, flow, and timing of each breath during positive pressure breathing in man. J Appl Physiol 45:495–501

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