EFFECTS OF A SIGH ON THE RESPIRATORY MECHANICAL PROPERTIES IN ALI PATIENTS

Vittorio Antonaglia, MD,¹ Sara Pascotto, MD,¹ Loredana De Simoni, MD,¹ and Walter A. Zin, MD, PhD²

Antonaglia V, Pascotto S, De Simoni, Zin WA. Effects of a sigh on the respiratory mechanical properties in ALI patients

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ABSTRACT. Objectives. The application of sighs during base-line ventilation was found to improve alveolar recruitment and oxygenation in patients with acute respiratory distress syndrome (ARDS). The present investigation evaluates if respiratory mechanics can be modified by a sigh. **Methods.** Ten consecutive patients with acute lung injury (ALI) admitted to the University Hospital Intensive Care Unit were studied during mechanical ventilation. Three sighs were administered to sedated-paralyzed patients during the measurement period. Respiratory mechanics were studied in regular breaths immediately before and after a sigh provided that a steady-state had been reached and by the airway pressure-time curve profile to evaluate the lung recruitment. Viscoelastic constants (elastic, resistive, and time), as well as elastance and resistances, were determined by the single breath method. Arterial blood gases were also determined pre- and post-sigh. **Results:** Elastic and resistive components of viscoelasticity decreased after a sigh (20 and 21%, respectively). As a result, the pressure required to overcome viscoelasticity and mechanical inhomogeneities also decreased in these patients (17%). The mechanical changes were associated with alterations in PaO₂. **Conclusions:** The sigh is useful to diminish viscoelastic impedance in ALI patients, thus allowing a smaller inflation pressure. Under the present experimental conditions it seems that viscoelastic mechanical alterations precede their elastic and resistive counterparts.

KEY WORDS. critical care, sigh, respiratory mechanics, resistance, elastance.

¹From the Istituto di Anestesia, Rianimazione e Terapia Antalgica, Laboratorio di Biomeccanica Respiratoria, Università degli Studi di Trieste, Trieste, Italy, ²Carlos Chagas Filho Institute of Biophysics, Federal University of Rio de Janeiro, Rio de Janeiro, Brazil.

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Address correspondence to Dr. Vittorio Antonaglia, Ospedale di Cattinara, Dipartimento di Anestesia e Rianimazione, Laboratorio de Biomeccanica Respiratoria, Strada di Fiume 447, I-34139 Trieste, Italy.

E-mail: v.antonaglia@libero.it

Recently the role of the sigh during mechanical ventilation in ARDS patients has been emphasized and the automatic delivery of sighs or sustained inflations by a ventilator can be considered a possible component of the ventilatory strategy in these patients [1–6].

The application of three sighs per minute during one hour of ventilation was found to improve alveolar recruitment and oxygenation in patients with ARDS in whom PEEP was adjusted using PaO₂/FiO₂ values [1, 2]. Pelosi *et al.* [1] also found a significant decrease in static lung elastance and respiratory resistance during the sigh period. Furthermore, it has also been demonstrated that in ARDS most of the resistance is provided by pulmonary tissue viscoelasticity [7]. Thus, in line with the results of the study of Pelosi *et al.* [1] it is possible to argue a modification of the viscoelastic properties of the lung during the sigh period. However, no determination of the viscoelastic constants of the respiratory system in ARDS and acute lung injury (ALI) patients has been performed in post-sigh breaths. Thus, the aims of the present investigation were to assess (1) whether sighs would modify the viscoelastic properties in

Table 1. Characteristics and diagnosis of patients with acute lung injury (ALI)

	Sex	Height (cm)	Weight (kg)	Age (yrs)	Diagnosis	PaO ₂ /FiO ₂ (mmHg)
1	M	181	100	45	multiple injury	198
2	F	155	68	60	pneumonia	185
3	M	184	89	39	multiple injury	215
4	F	165	69	61	sepsis	212
5	M	191	61	32	multiple injury	197
6	M	170	75	44	pneumonia	202
7	F	166	67	52	sepsis pneumonia	204
8	M	184	89	67	pancreatitis	191
9	F	159	62	70	sepsis	200
10	M	166	93	49	burns	227
Mean		172	77.3	51.9	—	200
SD		4	4.5	3.9	—	4

ALI patients with PEEP adjusted using PaO₂/FiO₂ values; (2) whether there were differences in the mechanical properties of the respiratory system after sighs in ALI patients in whom the steady-state portion of the dynamic volume-pressure (V-P) curve showed an upward concavity.

MATERIAL AND METHODS

Ten patients with ALI (PaO₂/FiO₂ < 300, and bilateral infiltrates on the chest radiograph) [8], who were consecutively admitted to the intensive care unit of Cattinara Hospital, were studied. The study was always performed after hemodynamic stabilization for 1 day. The patients were sedated (propofol, 4.5 mg·kg⁻¹·h) and paralyzed (vecuronium 0.15 mg·kg⁻¹·h). Their anthropometric characteristics are provided in Table 1. None had a history or clinical evidence of either restrictive or chronic lung disease, nor cardiogenic pulmonary edema or active cardiac disease. Patients were ventilated according to the guidelines of the Consensus Conference of the American College of Chest Physicians [9], *i.e.*, FiO₂ below 0.6, adequate PEEP to support oxygenation, and low V_T to avoid a plateau pressure higher than 35 cmH₂O.

We performed a PEEP trial in each patient by applying 5, 10 and 15 cmH₂O PEEP during baseline ventilation, which consisted of a V_T of 5.2 ± 0.6 ml·kg⁻¹, constant inspiratory flow (*V'*) of 0.5 ± 0.03 L·s⁻¹, constant FiO₂ of 0.5, and respiratory rate amounting to 16 bpm. At PEEP = 15 cmH₂O, in any patients the plateau

pressure exceeded 35 cmH₂O. Thus, V_T was decreased until it equalled 35 cmH₂O. After 20 min at each ventilatory set we measured PaO₂, and finally chose the level of PEEP at which the larger PaO₂ improvement was observed, accepting possible hypercapnia as a side effect [9, 10]. Table 2 depicts the baseline ventilatory setting in ALI patients.

Experimental procedure

Patients lay in the supine position, were intubated with a cuffed Rusch tracheal tube (ID: 7.0–8.0 mm), and ventilated with constant flow inflation by means of a 900C Servo Ventilator (Siemens-Elcoma AB, Solna, Sweden). Rapid airway occlusions were performed using a solenoid valve (Airmatic SV, Airmatic-Allied, Wilmington, OH, USA) placed next to the oral end of the tracheal tube (TT). The solenoid valve has a closing time of 11 ms, which was measured at the Electronics Laboratory, Department of Energetics, Faculty of Engineering, Trieste, Italy, with an accelerometer (Brüel & Kjær 4332, Brüel & Kjær Italiana, Milan, Italy) and a current probe (AC-DC Fluke Y8100, Fluke Corporation; Everett, WA, USA). Flow (*V'*) was measured (linear range: ±3 L·s⁻¹) with a heated Jäger pneumotachograph (Würzburg, Germany), inserted between the TT and the solenoid valve, which was connected to a Validyne differential pressure transducer (MP-45, ±2 cmH₂O, Northridge, CA, USA) and to a carrier amplifier (13-4615-35, Gould Inc., Cleveland, OH, USA). Tracheal pressure (Ptr) was measured with a piezoresistive differential

Table 2. Pre and post-sigh ventilatory setting and respiratory mechanics

	Pre-sigh	Post-sigh
Flow ($\text{L} \cdot \text{s}^{-1}$)	0.51 ± 0.01	0.51 ± 0.01
TI (s)	0.79 ± 0.03	0.79 ± 0.03
V_T (L)	0.40 ± 0.02	0.42 ± 0.02
RR (breaths min^{-1})	16.1 ± 0.33	16 ± 0.37
V'_e ($\text{L} \cdot \text{min}^{-1}$)	6.47 ± 0.34	6.76 ± 0.34
PEEPi (cmH_2O)	$1.9 \pm 1, 2$	1.7 ± 0.9
PEEPe (cmH_2O)	7.5 ± 0.83	7.5 ± 0.83
Pst,rs (cmH_2O)	16.5 ± 0.9	16.4 ± 0.9
Est,rs ($\text{cmH}_2\text{O} \cdot \text{L}^{-1}$)	22.8 ± 1.1	21.5 ± 1.3
Rint,rs ($\text{cmH}_2\text{O} \cdot \text{L}^{-1} \cdot \text{s}$)	3.03 ± 0.24	3.01 ± 0.19
ΔP (cmH_2O)	2.76 ± 0.16	$2.28 \pm 0.16^*$
τ_2 (s)	1.05 ± 0.08	1.05 ± 0.06
R_2 ($\text{cmH}_2\text{O} \cdot \text{L}^{-1} \cdot \text{s}$)	10.64 ± 1.27	$8.41 \pm 0.78^*$
E_2 ($\text{cmH}_2\text{O} \cdot \text{L}^{-1}$)	9.99 ± 0.58	$8.03 \pm 0.60^*$
PaO ₂ (mmHg)	96.9 ± 3.5	97.4 ± 2.9
PaCO ₂ (mmHg)	44.5 ± 2.1	42.7 ± 1.4
Pam (mmHg)	81 ± 6	77 ± 10

Values are means $\pm$ SD of ten patients in each instances (3 determinations/subject). ALI, acute lung injury; TI, inspiratory time; V_T tidal volume; RR, respiratory rate; V'_e , minute ventilation; PEEPi, intrinsic positive end-expiratory pressure; PEEPe, external positive end-expiratory pressure Pst, elastic recoil pressure; Est, static elastance; Rint, Newtonian resistance; ΔP , $P1$ -Pst,rs pressure dissipated against viscoelastic and inhomogeneous components; τ_2 , R_2 , and E_2 viscoelastic time constant, resistance, and elastance, respectively; Pam, mean arterial pressure. *Significantly different from pre-sigh value ($p < 0.05$).

pressure transducer (Microswitch 142PC05D, Honeywell Ltd., Scarborough, Ontario, Canada) via a polyethylene catheter protruding 2–3 cm beyond the tracheal tip of the TT. The tracheal catheter (ID: 1.5 mm) had six side holes and an occluded tip at its distal end. The system used to measure Ptr had a frequency response flat up to 20 Hz. The overall dead space of the measuring equipment (excluding the TT) was 35 ml. The resistance of the equipment was $8 \text{ cmH}_2\text{O} \cdot \text{L}^{-1} \cdot \text{s}$ at a flow of $1 \text{ L} \cdot \text{s}^{-1}$. V' and Ptr signals were fed through a 12-bit analogue-to-digital converter (DT2801A, Data Translation Inc., Marlboro, MD, USA) into a personal computer. Sampling frequency was 200 Hz. Volume (V) was obtained by numerical integration of the flow signal. All data were analyzed using ANADAT data analysis software (RHT-InfoData Inc., Montreal, Quebec, Canada).

Care was taken to avoid leaks around the tracheal cuff and in the equipment. During the sampling period the cuff pressure was $40 \text{ cmH}_2\text{O}$. Additionally, during the end-inspiratory occlusions the pressure reached an apparent plateau, further confirming the airtightness of the experimental assembly. In all patients, ECG and arterial blood

pressure were continuously monitored as well as peripheral O₂ saturation and end-tidal PCO₂ (Ohmeda 5250 RGM, Louisville, CO, USA). An anesthetist not involved in the experiment was continuously present to provide patient care.

The investigation was approved by the institutional review board of the Università degli Studi di Trieste, and informed consent was obtained from each patient or their next of kin.

Standard respiratory mechanics and viscoelastic parameters

The subjects were ventilated according to the aforementioned ventilatory settings. Under PEEP and before the sighs a series of consecutive breaths was collected, and V' and Ptr signals were averaged to determine the dynamic inflation V-P curve, as reported by Ranieri *et al.* [11]. Equation 1 was fitted to the experimental data points obtained during constant flow inflation (steady-state portion of the dynamic V-P curve):

$$V = a + b \cdot \text{Ptr} + c \cdot \text{Ptr}^2 \quad (1)$$

where coefficients a , b , and c are constants.

We performed baseline end-inspiratory occlusions to study respiratory mechanics before and after sighs by means of the single breath method (SBM) [12]. The sampling time-line was: (i) an end-expiratory occlusion for assessment of PEEPi; (ii) five breaths at baseline ventilatory settings; (iii) a 4-s end-inspiratory occlusion; (iv) five to six baseline breaths; (v) sigh; (vi) another series of 5–6 baseline breaths; (vii) an occlusion at baseline V_T ; (viii) 5–6 baseline breaths; (ix) end-expiratory pause (measurement of PEEPi). In each patient this sequence was repeated three times. The volume loss due to the continuous gas exchange had a negligible impact on Ptr. Heart artefacts were corrected according to Beydon *et al.* [13].

Immediately after the end-inspiratory occlusions (steps iii and vii above) Ptr plummeted from its peak value (Ptr, max) to an inflection point (P1). The delay between the beginning and the end of valve closure was allowed for by backextrapolation of the pressure records to the actual time of occlusion and the corrections in pressure, although very minute, were performed as previously described [14]. Thereafter a gradual decrease in Ptr led to an apparent plateau reflecting the elastic recoil pressure of the respiratory system (Pst). The difference between P1 and Pst (ΔP) represents the pressure dissipated against viscoelastic mechanical components and a minute degree of mechanical inhomogeneities [7, 12]. Additionally the following mechanical variables were computed: (a) Newtonian resistance [$R_{\text{int}} = (\text{Ptr, max} - P1)/V'$], and (b)

static elastance of the respiratory system (E_{st}), calculated as the difference between end-inspiratory and end-expiratory airway plateau pressures divided by V_T . Using the same occlusions the resistive component and the time constant of viscoelasticity/inhomogeneity (R_2 and τ_2 , respectively) were determined, and E_2 (viscoelastic elastance) was computed as R_2/τ_2 [12]. Arterial blood gases were analyzed immediately before and after data collection and 10 min after.

Sigh

During steady state conditions a series of three sighs was carried out in each individual. Five to six breaths under baseline condition were given between two consecutive sighs. While the baseline flow was kept constant V_T was increased to values ranging from 0.88 to 1.35 L. This was obtained by appropriately changing the frequency setting on the ventilator. After the inspiratory limb of the sigh a 5-s end-inspiratory occlusion was triggered. To choose the respiratory frequency, we combined two criteria: the inspiratory time (T_I) > 2.5 s, to optimize the potential recruitment [15] and the product $T_I \times V'$ had to yield a V_T resulting in a P_{st} smaller than 40 cmH₂O in all instances [1]. All measurements were repeated three times under the same experimental conditions. Between each test V' , V_T and P_{tr} were allowed to return to baseline values.

Statistical analysis

The normality of the data was assessed with the Kolmogorov-Smirnoff test (with Liliefors' correction), and the homogeneity of the variances evaluated by means of Levene's median test. In all cases both conditions were valid. Paired t -test was used to compare the data gathered before and after the sighs. Regression analysis was performed using the least squares method. The significance level was established at 5% ($\alpha = 0.05$).

RESULTS

In all subjects the slow decay in P_{tr} after end-inspiratory occlusion was closely fitted by a mono-exponential function of time ($p < 0.001$). The data also closely fitted the single breath method equation, the root mean square error ranged from 0.15 to 0.21 cmH₂O.

The mean values $\pm$ SD's of PEEP_i were similar before and after sigh (1.9 ± 1.2 and 1.7 ± 0.9 cmH₂O, respectively).

In 7 ALI patients the dynamic V-P curves showed an upward concavity with a positive value of the nonlinear coefficient c in Equation 1 (ranging between 0.002 to 0.004; mean value $\pm$ SD: 0.0025 ± 0.0005). In 3 patients the dynamic V-P curves exhibited a downward concavity reflected by a negative value of c (range: -0.0004 to -0.0006).

Table 2 depicts the mean values ($\pm$ SEM) of the mechanical parameters and ventilatory data obtained before and after sighs. ΔP , R_2 , and E_2 decreased significantly after sighs ($p < 0.0001$, $p = 0.005$, and $p < 0.001$, respectively).

The mean values $\pm$ SD of inspiratory duration and volume of sighs were 2.73 ± 0.29 s and 1.29 ± 0.16 L, respectively. The mean value ($\pm$ SD) of differences between the plateau pressures during basal ventilation and sigh was 10.9 ± 2.1 cmH₂O. Considering the patients with the dynamic V-P curves showing an upward or downward concavity the mean values of these differences were 10.3 ± 2.1 cmH₂O and 12.5 ± 1.3 cmH₂O, respectively.

Although the overall average pre- and post-sigh values of PaO₂ did not differ significantly [96.9 ± 11.1 (SD) mmHg and 97.4 ± 8.9 (SD) mmHg, respectively], it is noteworthy that only the 3 patients that presented a negative non-linear coefficient had a smaller PaO₂ after sigh. If the remaining 7 patients were analyzed independently, the sigh promoted a significant increase in PaO₂ [from 92.1 ± 9.5 (SD) mmHg to 97.4 ± 9.7 (SD) mmHg; $p = 0.041$]. After ten minutes the patients with a negative non-linear coefficient presented no differences in gas exchange in comparison to the pre-sigh data, while the remaining 7 patients maintained a significant increase in PaO₂ [from 92.1 ± 9.5 (SD) mmHg to 97.1 ± 9.1 (SD) mmHg; $p = 0.045$].

When the differences between the pre- and post-sigh values of R_2 , E_2 , and ΔP (ΔR_2 , ΔE_2 , and $\Delta P'$, respectively) were plotted against the difference between pre- and post-sigh PaO₂ (ΔPaO_2), significant inverse linear relationships were found: $\Delta PaO_2 = 7.6 - 3.6 \cdot \Delta R_2$, $r = -0.73$, $p = 0.01$, *filled circles*, Figure 1; $\Delta PaO_2 = 23.5 - 12.2 \Delta E_2$, $r = -0.64$, $p = 0.04$, *open circles*, Figure 1; and $\Delta PaO_2 = 24.3 - 51.8 \Delta P'$, $r = -0.83$, $p = 0.003$, Figure 2.

DISCUSSION

The present investigation showed that within the experimental period studied and following the PEEP trial, sighs did not change significantly the mean values of PaO₂ and E_{st} in our population of patients with acute lung injury (Table 2). Nevertheless, after sighs the viscoelastic resistive and elastic constants, as well as the pressure dissipated to overcome viscoelasticity and/or mechanical inhomogeneity

Fig. 1. Relationships between the variation between the pre- and post-sigh values of the viscoelastic resistive and elastic constants (ΔR_2 and ΔE_2 , respectively) and the difference in PaO_2 measured under the same conditions (ΔPaO_2) in 10 patients with acute lung injury. The solid line and circles pertain to ΔPaO_2 vs. ΔR_2 ($\Delta \text{PaO}_2 = 7.6 - 3.6 \pm \Delta R_2$, $r = -0.73$, $p = 0.01$), whereas the broken line and open circles refer to the ΔPaO_2 vs. ΔE_2 relationship ($\Delta \text{PaO}_2 = 23.5 - 12.2\Delta E_2$, $r = -0.64$, $p = 0.04$).

Fig. 2. Relationships between the variation between the pre- and post-sigh values of the pressure (ΔP) dissipated against viscoelastic and inhomogeneous mechanical components ($P_1 - P_2$) and the difference in PaO_2 measured under the same conditions (ΔPaO_2) in 10 patients with acute lung injury. The solid line represents the regression line ($\Delta \text{PaO}_2 = 24.3 - 51.8 \pm \Delta P$, $r = -0.83$, $p = 0.003$).

geneities within the system, decreased in ALI patients. Additionally, in the patients in who the differences between these variables were larger, the increase in PaO_2 was higher.

Our analysis was based on the assumption that the respiratory system behaves as a linear viscoelastic model, which clearly should not be regarded as a complete and perfect representation of respiratory mechanics. More complex nonlinear viscoelastic [16] and viscoplastoelastic models [17] have been used to explain volume- and time-dependence of energy dissipation within the respiratory system. Nevertheless, the linear viscoelastic model has been shown to provide an accurate description of the time-dependence of resistance and elastance over the lung volume and flow ranges used in the present study [18, 19].

The baseline values of the respiratory mechanical parameters reported in Table 2 are not different from those previously found in ALI patients [12, 20]. The introduction of a sigh into a regular pattern of baseline breaths decreased ΔP in ALI patients, although not back to normal values [12, 18], indicating that the viscoelastic properties and/or inhomogeneities of the system could be improved by the maneuver, *i.e.*, impedance decreased. In this context, no report has been found in the literature concerning the measurement of the viscoelastic constants (τ_2 , R_2 , and E_2) after sighs. These constants, however, were determined in normal subjects [12, 16, 17], ARDS [7] and COPD patients [21] to evaluate the time-, flow-, and volume-dependence of respiratory resistance and elastance.

In the present study τ_2 , R_2 , and E_2 were not different from those obtained in previous studies in ALI patients [12, 20]. However, we obtained post-sigh values of R_2 , and E_2 significantly smaller than the pre-sigh ones, while τ_2 was not significantly different (Table 2). This probably resulted from either a resetting of the viscoelastic mechanical components of the respiratory system. *i.e.*, returning their elastic and resistive components to baseline mechanical conditions, and/or to the recruitment of atelectatic air spaces in the diseased lungs.

In previous studies in ARDS patients [1–6] lung volume recruitment during sighs was associated with a significant increase in PaO_2 . Interestingly enough we found that the higher the change in PaO_2 measured before and after sighs, the greater the decrease in ΔP (Figure 2). The same behavior was presented by ΔE_2 and ΔR_2 (Figure 1). ΔP is determined by the actual values of E_2 and R_2 at any given time throughout the breathing cycle, and, hence, according to the viscoelastic model of the respiratory system, it is conceivable that the constants assume new values after a deep breath, yielding a change in ΔP . On the other hand, the sigh will recruit collapsed air spaces and minimize mechanical inhomogeneities [1], thus decreasing ΔP , E_2 and R_2 . Neumann et al. [15] found that recruitment occurred mainly within the first 4 s of breath-holding procedures in

different lung injury models. In our investigation the mean value of the inspiratory time was of 2.73 ± 0.29 s and presumably the potential recruitment occurred mainly within this time.

Previous studies in ARDS patients demonstrated a decrease in Est after the delivery of a deep breath [1–4]. We did not find such a result (Table 2). The disagreement between the reports could be secondary to different experimental designs: (i) they used a distending pressure around 40 cmH₂O for about 40 s, whereas in the present investigation an inspiratory duration of 2.7 s was applied instead. The longer distending pressure could have opened more air spaces to a greater extent [3]; (ii) Pelosi *et al.* [1] used three sighs per minute for at least one hour; (iii) Patroniti *et al.* [4] used sighs during pressure support ventilation; and (iv) our patients were less severe than theirs (ALI $\times$ ARDS), so that a recruitment maneuver could be more effective in ARDS.

Ranieri *et al.* [11] reported that in ARDS patients the dynamic volume–pressure curve represents a clinical tool to detect hyperinflation and to predict the effects of PEEP on alveolar recruitment. In accordance with them [11] we found that the patients with negative ϵ values (Equation 1) had the highest values of static elastance and end-inspiratory plateau pressure, and a tendency to show a fall in PaO₂ after a sigh. Moreover, we found that the patients with the dynamic V–P curves showing an upward concavity had a lower difference between the values of the plateau pressure during sigh and basal tidal volume (10.3 ± 2.1 cmH₂O) in comparison to those of the patients with dynamic V–P curves showing a downward concavity (12.5 ± 1.3 cmH₂O). The higher sigh plateau pressure in the latter patients can be related to the overstretching pressure in the ventilated alveolar districts. Thus, hyperinflation probably increased with sigh in the latter patients. In the other 7 patients, presenting a positive ϵ value, recruitment probably occurred [11], explaining the increase in PaO₂ after the sigh. Conversely, the mean value of PaCO₂ had the tendency to decrease after sigh (Table 2), but in the patients with dynamic V–P curves showing a downward concavity PaCO₂ remained unchanged (before sigh 44.2 ± 1.9 , after sigh 44.4 ± 1.7 mmHg). A possible explanation is that the potential increase of PaCO₂ related to hyperinflation in these patients was counterbalanced from the changes in hemodynamics due to reduction in mean arterial pressure after sigh, that in these patients was of 15 ± 3 mmHg (Table 2). These results were confirmed from the modifications of gas exchange ten minutes after sigh. PaO₂ remained higher and PaCO₂ slightly lower than the pre-sigh values in the patients with V–P curves showing an upward concavity, while PaO₂ and PaCO₂ returned to the values pre-sigh in the patients with V–P curves showing a downward concavity, indicating a strong de-

pendency by the hemodynamic fluctuation in these latter patients.

Potential limitations of the present investigation can be considered: (a) lung recruitment was assessed by a single method; (b) the measurement of ΔP was obtained with a precise experimental method. We used the airway pressure–time curve profile [11] to evaluate the lung recruitment. Other more sophisticated methods to assess a potential alveolar recruitment can be used [1, 22–25] and further studies have to be performed to compare the results of these methods with the modifications of the viscoelastic properties after a sigh. The measurement of P1 represents a critical point in the assessment of ΔP . In our experimental setting P1 was obtained by backextrapolation. The results of the present study have to be confirmed by measuring ΔP in clinical practice. On the other side, the airway pressure–time curve profile was recently confirmed as a good predictor of recruitment and/or hyperinflation [26] and our results can be considered the first analysis of the viscoelastic properties after a sigh and, taking into consideration the bedside evaluation of the changes in viscoelastic constants, the measurement of ΔP can represent an easier approach than the actual determination of E_2 and R_2 . Thus, if further studies will confirm our results, it will be possible to consider the measurement of ΔP as a very helpful when evaluating the diverse sigh patterns presently available to recruit collapsed air spaces [1, 3, 6].

In conclusion, in ALI patients the pre- and post-sigh determination of the viscoelastic constants and/or the pressure dissipated to overcome mechanical inhomogeneities might indicate an improvement in the mechanical impedance of the respiratory system earlier than the results yielded by the traditional static elastance or resistance.

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