

## **Short-term effects of the prone positioning manoeuvre on lung and chest wall mechanics in ARDS patients**

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Little is known about changes in respiratory mechanics during the procedure of prone positioning in ARDS patients. This information is important in order to interpret changes in airway pressure that may occur in lateral and prone position during volume controlled ventilation. Indeed some changes may result from chest wall elastance alteration. We underwent the present study to assess lung and chest wall mechanics in a series of consecutive ARDS patients during the procedure of prone positioning.

## Methods

The study was approved by our local ethic committee (2014-AO-1714-43). Forty-one patients (26 men) of  $66 \pm 12$  years in age with moderate to severe ARDS(1) intubated and mechanically ventilated in volume controlled ventilation, sedated and paralyzed were included once clinician indicated prone positioning ( $\text{PaO}_2/\text{F}_\text{I}\text{O}_2 < 150$  mmHg under  $\text{PEEP} \geq 5$  cm  $\text{H}_2\text{O}$ ) after informed consent of the next of kin. Mean  $\pm$  SD tidal volume was  $6 \pm 0.6$  ml/kg ideal body weight,  $\text{PEEP}$   $11 \pm 3$  cm $\text{H}_2\text{O}$ , inspiratory flow  $1 \pm 0$  L/s (constant shape) and  $\text{F}_\text{I}\text{O}_2$   $73 \pm 15\%$ . Airway pressure ( $\text{Paw}$ ) was measured proximal to endotracheal tube and airflow by Fleish II pneumotachograph inserted between  $\text{Paw}$  port and Y-piece. Oesophageal pressure ( $\text{Pes}$ ) was measured by using air-filled catheter (Nutrivent, Sidam, Italy). Ventilator settings, except  $\text{F}_\text{I}\text{O}_2$ , were kept unaltered during the whole study. In our ICU the proning procedure is made routinely by 3 caregivers with one staying at the patient head for securing the endotracheal tube and avoiding any kinking. Furthermore, the trachea is systematically suctioned before the procedure without patient disconnection. Pressure and flow signals were continuously recorded on a datalogger (Biopac 150, Biopac inc, Goletta, USA) in supine  $0^\circ$  for 5-10 minutes then in the transient 3-minute  $90^\circ$  lateral position (23 patients with left lateral) and then during the first 5-10 minutes in prone  $0^\circ$ . The patients remained in prone in

a 0°-15° angulation for the next consecutive 16 hours. The reverse manoeuvre from prone 0° to supine 0° via the same previous 90° lateral position was also subjected to same recording.

Trans-pulmonary pressure was obtained by subtracting  $P_{es}$  from  $P_{aw}$ . Lung resistance ( $R_L$ ) and lung ( $E_L$ ) and chest wall ( $E_{cw}$ ) elastance were computed by fitting measurements with a resistance-elastance linear model. This procedure was done breath by breath using classical least square regression method (figure 1). The data were analyzed by using linear mixed model to take into account the fact that serial measurements were made in same patients. We investigated the effects of lateral and prone position, first when turning the patient prone, second when putting the patient back from prone to supine. The model included as fixed effects the position, the sequence (proning or back to supine) and the interaction between them, and the position by patient as random effect. For each position the mean value was compared to the mean of the corresponding reference.

## Results

$R_L$  and  $E_{cw}$  significantly increased in the lateral and prone positions from supine.  $E_L$  markedly increased in the lateral position but was not different in prone from supine (table 1). During the reverse procedure to put the patient back to supine, lateral positioning was associated with a significant decrease in  $E_L$  and no change in both  $R_L$  and  $E_{cw}$ ; supine repositioning was associated with a significant decrease in  $E_L$  and  $E_{cw}$  and no change in  $R_L$  (table 1). The corresponding raw values of  $P_{aw}$  and absolute  $P_{es}$  used to determine respiratory mechanics are shown in table 2.

## Discussion

Present study primarily intended to describe how much respiratory mechanics would change during the procedure of proning and in particular at the time of immediate installation in

prone position and back to supine when the measurements were performed at 0° inclination. We found that  $E_{cw}$  increased in prone as previously observed (2-4). According to Pelosi et al(2) the increase in  $E_{cw}$  is the trigger of the redistribution of ventilation and the improvement in oxygenation in prone. This effect would result from the limited expansion of the sternum because in prone the lung operates between two rigid bars. This could be a mechanism by which the distribution of tidal volume is more homogenous making the resulting overall lung stress (transpulmonary pressure) better distributed in prone. In this way prone position may contribute to lung protection. Regional pleural pressure and stress raisers (5), which in inhomogeneous lung parenchyma convert a safe level of transpulmonary pressure for an homogenous lung into a locally injurious stress, play an important role in the lung stress distribution. These factors were not assessed in present study. Our study brought up the new findings that the effect of prone on  $E_{cw}$  was immediate and did not vary over time for a prone session as long as 16 hours. In our present cohort of ARDS patients the same  $E_L$  and the higher  $E_{cw}$  in prone as compared to supine would obviously result in higher plateau pressure of the respiratory system, without any increase in the degree of alveolar stress. Actually, we found that trans-pulmonary plateau pressure increased in prone when a decrease would rather be expected. This somehow questions the predominant protective role of the increase in  $E_{cw}$ . It should be mentioned that the increase was very small and at any rate below the upper safety limit suggested from theoretical considerations(5). Our study showed that not only  $E_{cw}$  but also  $R_L$  increased in prone in ARDS patients, a finding which has not been previously described. As the inspiratory flow was set of 1 L/s the 1.2 cmH<sub>2</sub>O/L/s increase in  $R_L$  corresponds to a 1.2 cmH<sub>2</sub>O resistive pressure increase, which can contribute to higher peak  $P_{aw}$  in volume controlled mode (table 2). Even it was statistically significant the clinical importance of this increase is doubtful. This increase in  $R_L$  can be due

to the reduction in airways diameter and loss in lung volume(6). However, in studies where end expiratory lung volume was measured prone positioning was in general associated with not a decrease (2, 3). It could be that prolonged lateral position period could have resulted in some loss of aeration of the dependent lung and this effect carried over to the prone position resulting in this possible loss in lung volume. Going back to supine from prone essentially offset the effect of the previous supine-prone steps except for  $R_L$ .

In present study we found that  $R_L$ ,  $E_L$  and  $E_{cw}$  all increased significantly from supine to lateral. Previous observations showed reduction in compliance of the respiratory system after 30 minutes in lateral position(7) or during continuous rotating mobilization (8). Our study extends these findings by showing that both lung and chest wall compliance are impaired. Furthermore, our findings suggest that the increase in  $E_L$  and  $R_L$  observed in the further prone took place at this early step of changing position. It should be mentioned that there were no kinking or massive tracheal secretions at the endotracheal tube at the time respiratory mechanics was measured in the lateral position.

Our study is limited by the short time observation period and the lack of measurement of lung volume and gas exchange. This was planned because our primary aim was to describe respiratory mechanics during the routine procedure of prone positioning. Measurement of end expiratory lung volume requires time and would have prolonged the duration of the procedure beyond the routine care.

Further studies should explore the mechanism of these changes and in particular the relationships of the changes to end expiratory lung volume.

In conclusion, during the prone position manoeuvre  $R_L$ ,  $Ecw$ , and  $E_L$  increased immediately in lateral position.  $R_L$  did not change further in prone and back to supine.  $Ecw$  and  $E_L$  returned to baseline values back to supine.

## Legends for figure

**Figure 1.** A. Superimposed tracings over time of airway pressure (Paw, dark grey line), esophageal pressure (Pes, white grey line) and flow rate (open circles) during a single breath of mechanical mechanical ventilation in volume-controlled mode at constant flow inflation.

B. Trans pulmonary pressure (Paw-Pes) (dark grey line) and Pes (white grey line) above PEEP to which the following models are tested (dotted and broken dark lines, respectively).

$$\Delta (Paw-Pes) = R_L \times Flow + E_L \times \Delta V$$

$$\Delta Pes = E_{cw} \times \Delta V$$

where is  $R_L$  lung flow resistance,  $E_L$  lung elastance,  $E_{cw}$  chest wall elastance and  $\Delta V$  tidal volume

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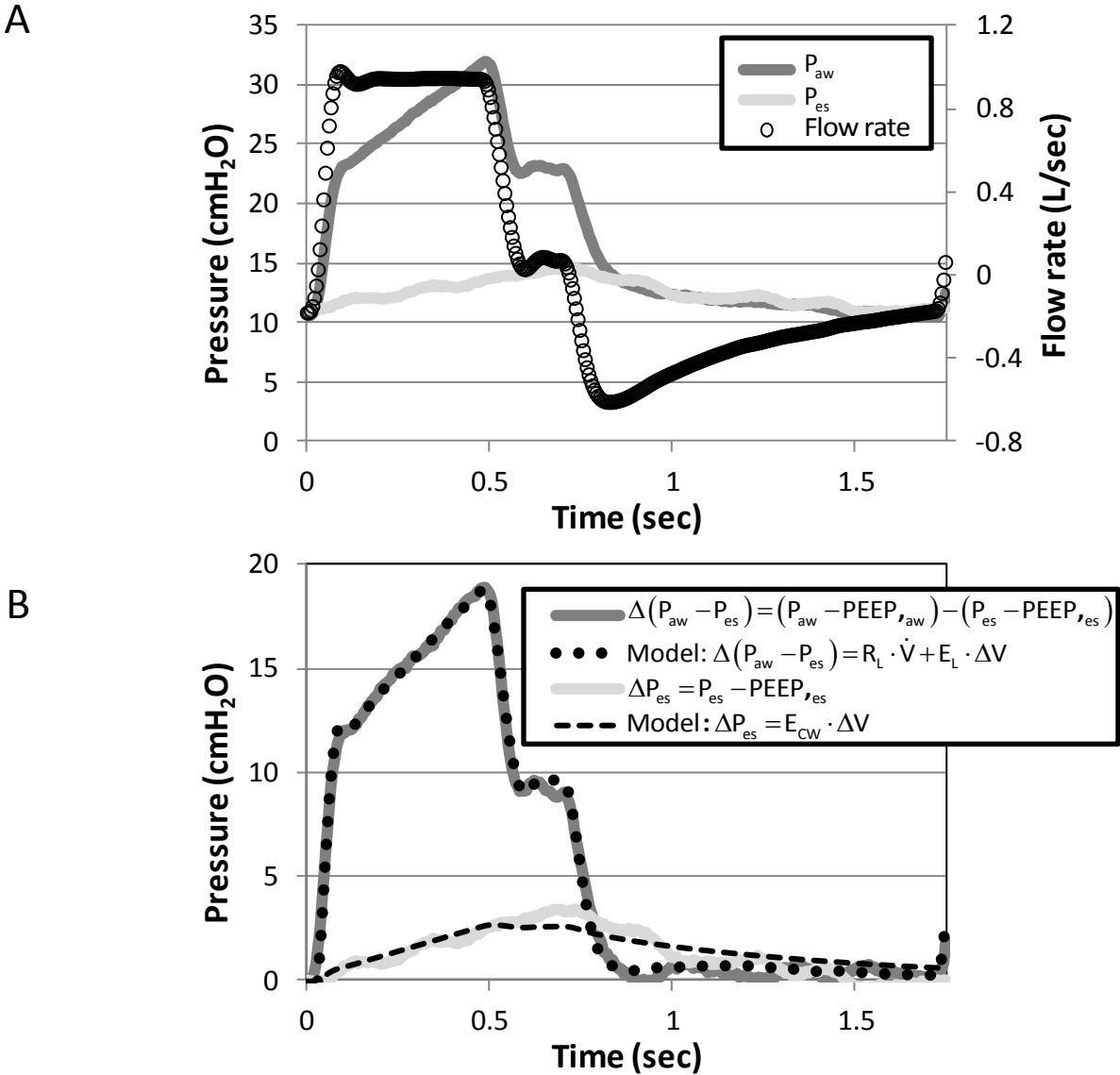
Table 1. Respiratory, lung and chest wall predicted values obtained from the mixed model during the procedure of proning and back to supine position in 41 ARDS patients.

	Supine  (reference)	Lateral	Prone	Prone  (reference)	Lateral	Supine
R <sub>L</sub> (cmH <sub>2</sub> O/L/s)	14 [13;15]	16 [14;17] *	15 [14;16] *	15 [14;17]	16 [15;17]	14 [13;15]
E <sub>L</sub> (cmH <sub>2</sub> O/L)	32 [28;37]	35 [30;40] *	32 [28;37]	33 [27;38]	35 [30;41] *	32 [26;37] *
Ecw (cmH <sub>2</sub> O/L)	10 [9;11]	12 [11;14] *	11 [10;13] *	11 [9;13]	12 [11;14]	9 [7;11] *
Maximal Paw (cmH <sub>2</sub> O)	35 [34;37]	38 [37;40] *	37 [35;39] *	37 [35;39]	39 [37;40] *	36 [34;37]
Plateau Paw (cmH <sub>2</sub> O)	24 [22;25]	26 [24;27] *	25 [24;27] *	25 [24;27]	26 [25;27] *	24 [22;25] *
PEEP,aw (cmH <sub>2</sub> O)	11 [10;12]	11 [10;12]	11 [10;12] *	11 [10;12]	11 [10;11]	11 [10;12]
Driving pressure of the respiratory system (cmH <sub>2</sub> O)	14 [12;15]	15 [14;17] *	14 [13;16] *	14 [13;16]	15 [14;17] *	13 [12;14] *
Maximal Pes (cmH <sub>2</sub> O)	15 [13;16]	14 [12;15]	14 [12;15]	14 [12;15]	14 [13;15]	14 [12;16]

Plateau Pes (cmH2O)	14 [13;15]	13 [12;15]	13 [12;15]	13 [12;15]	13 [12;15]	14 [12;16]
PEEP,es (cmH2O)	11 [10;12]	9 [8;10] *	9 [8;11] *	9 [8;11]	9 [8;10] *	11 [10;12]
Driving pressure of the chest wall (cmH2O)	3 [3;4]	5 [4;6] *	5 [4;5] *	4 [3;4]	4 [4 ;5]*	3 [2;4] *
Maximal trans-pulmonary pressure (cmH2O)	21 [19;23]	25 [23;27] *	23 [21;26]*	23 [21;25]	24 [23;26] *	21 [19;23]
Trans-pulmonary plateau pressure (cmH2O)	10 [9;11]	13 [11;14] *	12 [10;14] *	12 [11;14]	13 [11;14] *	10 [8;11] *
Trans-pulmonary PEEP (cmH2O)	0 [-1;+1]	2 [0;3] *	2 [0;3] *	2 [1;3]	1 [0;2] *	-1 [-1;+1]
Trans-pulmonary driving pressure (cmH2O)	11 [9;12]	11 [10;13] *	10 [9;12]	10 [9;12]	11 [10;13] *	10 [9;12] *

Values are mean [95% confidence interval]

\*P<0.05 when comparing the mean value in the position to the mean value of the reference position in the sequence



**Figure 1**  
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