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Short-term effects of the prone positioning manoeuver 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 ± 12 years in age with moderate to severe ARDS(1) intubated and mechanically ventilated during volume controlled ventilation, sedated and paralyzed were included once clinician indicated prone positioning ($\text{PaO}_2/\text{F}_i\text{O}_2 < 150$ mmHg under $\text{PEEP} \geq 5$ cm H_2O) after informed consent of the next of kin. Mean \pm SD tidal volume was 6 ± 0.6 ml/kg ideal body weight, PEEP 11 ± 3 cm H_2O , inspiratory flow 1 ± 0 L/s (constant shape) and F_iO_2 $73 \pm 15\%$. Airway pressure (Paw) was measured proximal to endotracheal tube and airflow by Fleisch II pneumotachograph inserted between Paw port and Y-piece. Oesophageal pressure (Pes) was measured by using air-filled catheter (Nutrivent, Sidam, Italy). Ventilator settings, except F_iO_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° for 5-10 minutes then in the transient 3-minute 90° lateral position (23 patients with leftlateral) and then during the first 5-10 minutes in prone. The patients remained in prone

in a 0°-15° angulation for the next consecutive 16 hours. The reverse manoeuvre from prone 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 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 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

Basically present results confirm that in ARDS patients E_{cw} increases in prone (2-4) . According to Pelosi et al(2) the increase in E_{cw} explains the redistribution of ventilation and

the improvement in oxygenation in prone. This effect would result from restriction in the 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. This is a mechanism by which 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 this increase was very small and at any rate below the upper safety limit suggested from theoretical considerations. 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 cmH₂O/L/s increase in R_L corresponds to a 1.2 cmH₂O 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 , E_{cw} , and E_L increased immediately in lateral position. R_L did not change further in prone and back to supine. E_{cw} and E_L returned to baseline values back to supine.

REFERENCES

1. Ranieri VM, Rubenfeld GD, Thompson BT, Ferguson ND, Cladwell E, Fan E, Camporota L, Slutsky AS. Acute respiratory distress syndrome. The berlin definition. *JAMA* 2012;307:E1-E8.
2. Pelosi P, Tubiolo D, Mascheroni D, Vicardi P, Crotti S, Valenza F, Gattinoni L. Effects of the prone position on respiratory mechanics and gas exchange during acute lung injury. *Am J Respir Crit Care Med* 1998;157:387-393.
3. Guerin C, Badet M, Rosselli S, Heyer L, Sab JM, Langevin B, Philit F, Fournier G, Robert D. Effects of prone position on alveolar recruitment and oxygenation in acute lung injury. *Intensive care medicine* 1999;25:1222-1230.
4. Mentzelopoulos SD, Roussos C, Zakynthinos SG. Prone position reduces lung stress and strain in severe acute respiratory distress syndrome. *Eur Respir J* 2005;25:534-544.
5. Mead J, Takishima T, Leith D. Stress distribution in lungs: A model of pulmonary elasticity. *J Appl Physiol* 1970;28:596-608.
6. Eissa NT, Ranieri VM, Corbeil C, Chasse M, Robatto FM, Braidy J, Milic-Emili J. Analysis of behavior of the respiratory system in ards patients: Effects of flow, volume, and time. *J Appl Physiol* 1991;70:2719-2729.
7. Thomas PJ, Paratz JD, Lipman J, Stanton WR. Lateral positioning of ventilated intensive care patients: A study of oxygenation, respiratory mechanics, hemodynamics, and adverse events. *Heart Lung* 2007;36:277-286.
8. Schellongowski P, Losert H, Locker GJ, Laczika K, Frass M, Holzinger U, Bojic A, Staudinger T. Prolonged lateral steep position impairs respiratory mechanics during continuous lateral rotation therapy in respiratory failure. *Intensive Care Med* 2007;33:625-631.

Table 1. Lung resistance and lung and chest wall elastance and their changes during the procedures of proning and back to supine position in 41 ARDS patients.

	Supine (reference)	Lateral	Prone	Prone (reference)	Lateral	Supine
R_L (cmH ₂ O/L/s)	13.8 [12.7;14.9]	+ 1.9 [1.1;2.8]*	+ 1.2 [0.3;2.1]*	15.2 [13.7;16.7]	- 0.1 [-1.3;1.2]	- 0.2 [-1.4;1.0]
E_L (cmH ₂ O/L)	32.4 [27.8;36.9]	+ 3.8 [1.6;5.9]*	+ 1.2 [-0.4;2.8]	34.2 [28.5;39.9]	- 2.9 [-5.7;-0.1] *	- 3.0 [-5.8;-0.2] *
E_{cw} (cmH ₂ O/L)	9.8 [8.5;11.2]	+ 2.5 [1.2;3.7]*	+ 1.6 [0.8;2.3]*	12.5 [10.6;14.5]	- 0.7 [-2.0;0.6]	- 4.8 [-6.2;-3.3]*

R_L lung airflow resistance, E_L lung elastance, E_{cw} chest wall elastance. Supine (reference) and Prone (reference) give absolute values while the other positions give change with the reference position.

*P<0.05 versus corresponding reference

Values of reference are the intercept of the linear mixed model and the other values pertain to the corresponding changes from the reference. Values into brackets are 95% confidence intervals.

Table 2. Airway, absolute esophageal and trans-pulmonary pressures and their changes during the procedure of proning and back to supine position in 41 ARDS patients.

	Supine (reference)	Lateral	Prone	Prone (reference)	Lateral	Supine
Maximal Paw	35.3 [33.7;37.0]	3.1 [2.0;4.3] *	1.8 [0.7;2.8] *	38.1 [36.2;40.1]	-2.4 [-4.0;-0.8] *	-1.6 [-3.1;0.0]
Plateau Paw	23.9 [22.4;25.4]	1.6 [0.6;2.7] *	1.6 [0.4;2.7] *	26.2 [24.8;27.7]	-2.5 [-3.9;-1.0] *	-1.2 [-2.3;-0.0] *
PEEP,aw	10.6 [9.6;11.6]	-0.3 [-1.1;0.5]	0.3 [0.0;0.7]*	11.1 [9.8;12.3]	0.0 [-1.1;1.1]	0.1 [-0.8;1.0]
Driving pressure of the respiratory system	13.5 [12.3;14.6]	2.0 [1.3;2.6]*	1.1 [0.3;1.9]*	15.0 [13.6;16.4]	-2.4 [-3.4;-1.5]*	-1.2 [-2.0;-0.4]*
Maximal Pes	14.5 [13.1;15.8]	-0.7 [-1.8;0.4]	-1.3 [-2.3;-0.3] *	14.6 [13.0;16.2]	0.1 [-1.4;1.5]	0.1 [-1.0;1.3]
Plateau Pes	13.9 [12.6;15.2]	-0.8 [-1.9;0.2]	-1.2 [-2.2;-0.2]	14.0 [12.4;15.6]	0.3 [-1.2;1.7]	0.3 [-0.8;1.5]
PEEP,es	10.7 [9.6;11.8]	-1.7 [-2.6;-0.7]*	-1.7 [-2.7;-0.8]*	3.8 [3.0;4.7]	2.5 [1.9;3.1]*	2.2 [1.8;2.8]
Driving pressure of the chest wall	3.3 [2.9;3.7]	0.8 [0.4;1.1]*	0.5 [0.3;0.8]*	4.2[3.6;4.8]	-1.6 [-2.1;-1.2]*	-0.2 [-0.6;0.2]*

Maximal trans-pulmonary pressure	20.9 [19.1;22.7]	3.8 [2.6;5.1]*	3.0 [1.7;4.3]*	23.8 [21.8;25.8]	-2.5 [-4.2;-0.8]*	-1.7 [-3.4;0.1]
Trans-pulmonary plateau pressure	10.0 [8.6;11.4]	2.6 [1.4;3.7]*	2.8 [1.5;4.0]*	12.4 [11.0;13.9]	-3.1 [-4.5;1.6]*	-1.4 [-2.7;-0.0]*
Trans-pulmonary PEEP	-0.0 [-1.1;1.0]	1.4 [0.4;2.4]*	2.0 [1.1;2.9]*	1.6 [0.4;2.7]	-1.9 [-3.1;-0.7]*	-0.6 [-1.5;0.4]
Trans-pulmonary driving pressure	10.5 [9.4;11.6]	1.1[0.5;1.7]*	0.3 [-0.2;0.7]	10.9 [9.5;12.3]	-0.9 [-1.7;-0.1]*	-1.1 [-1.9;-0.3]*

Paw airway pressure, Pes esophageal pressure. Driving pressures of respiratory system and chest wall are obtained by subtracting the corresponding Paw and absolute Pes plateau pressures from the corresponding PEEP, respectively. Trans-pulmonary pressures are obtained by the difference between the corresponding Paw and absolute Pes. Supine (reference) and Prone (reference) give absolute values while the other positions give change with the reference position.

*P<0.05 versus corresponding reference

Values of reference are the intercept of the linear mixed model and the other values pertain to the corresponding changes from the reference. Values into brackets are 95% confidence intervals. Values are in cmH2O

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

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

Legends for figure

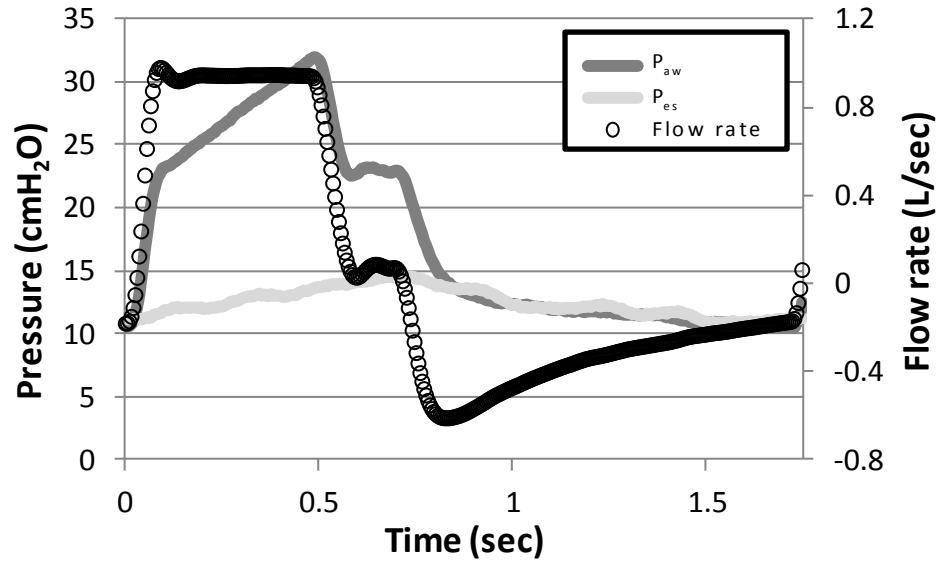
Figure 1. A. Superimposed tracings over time of airway pressure (Paw, dark grey line), esophageal pressure (Pes, white grey line) and flow rate (open circles) during a single breath of mechanical mechanical ventilation in volume-controlled mode at constant flow inflation. B. Trans pulmonary pressure (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 R_L is lung flow resistance, E_L lung elastance, E_{cw} chest wall elastance and ΔV tidal volume

A



B

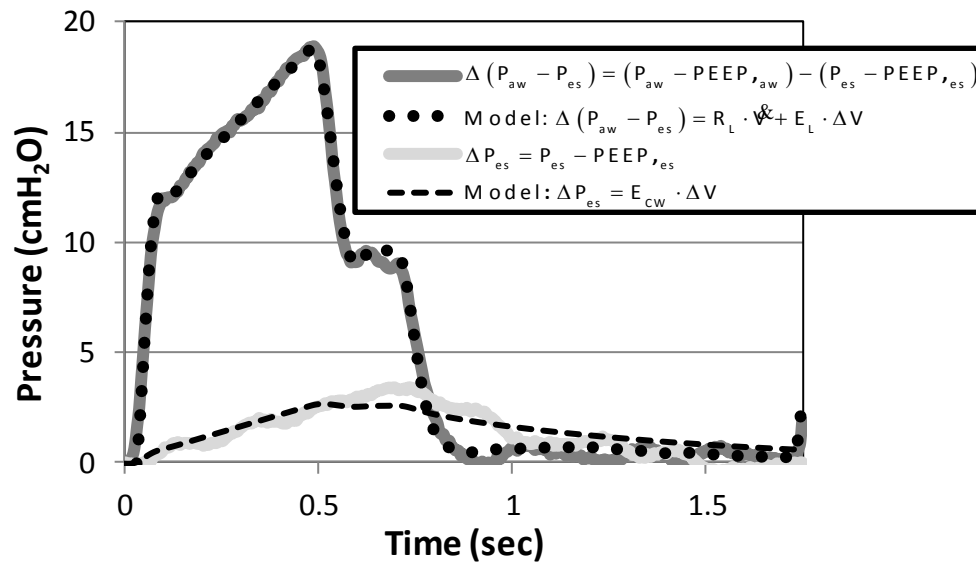


Figure 1