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What's new in respiratory physiology? The expanding chest wall revisited!

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It is common opinion that the elastic lung is enclosed within another elastic structure, the chest wall, and that both require pressure to be expanded. However, this is not true. An isolated lung will collapse to airlessness, but a lung inside the chest will not. It will stay open at functional residual capacity, FRC, which is the volume at which the inward elastic forces of the lung are balanced by the outward elastic forces of the chest wall [1]. Thus, the chest wall acts to expand the lung. This balance between lung and chest wall results in a negative pleural pressure (negative in relation to atmospheric pressure) that is more negative in the upper, non-dependent region than in lower, dependent ones, because of the weight of the lung and possible shape differences between lung and rib cage [2]. Lung elastance decreases with increasing age because of loss of elastic tissue, and pleural pressure may become positive in the dependent part of the pleural space. Airways will close and alveoli may eventually be airless. During anesthesia, FRC is reduced by half a liter, presumably by reduced outward force by the rib cage because of loss of respiratory muscle tone [3]. This also promotes airway closure and atelectasis formation. However, if the lung or part of it is aerated at resting condition, i.e., at an alveolar pressure of 0 cm H_2O (FRC), then extra-luminal pressure, assumed to be reflected by pleural pressure at the level of the aerated lung, must be negative. And the other way around, with a higher extra-luminal than intra-luminal pressure, alveoli will collapse. Airways in the same region will also close and may delay the collapse but atelectasis will eventually occur [3]. This holds true whether the subject is breathing spontaneously or is connected to a ventilator, and irrespective of body position. In other words, to stay open, a lung region must be exposed to a positive transpulmonary pressure (airway pressure minus pleural pressure). Pleural pressure must be negative if the subject is breathing at atmospheric pressure, while it may be positive to a varying degree if the subject is breathing or ventilated at positive airway pressure.

Will the reasoning above also apply to a sick lung? Let us consider acute respiratory distress syndrome (ARDS), where lung volume is further reduced ("baby lung"). Lung elastance is increased and this should make pleural pressure more negative, but overall volume including tissue may instead increase or remain unaltered, and the net effect on pleural pressure will therefore be less predictable. The chest wall may also be affected in ARDS, becoming heavier, with potential impact on lung volume and pleural pressure ("extrapulmonary ARDS") [4]. Still, if part of the lung is aerated at FRC (resting condition with zero airway pressure), then pleural pressure at the level of the aerated lung must be negative. Similar reasoning has been presented earlier [5].

During spontaneous breathing, pleural pressure reflects the force applied to expand the lung, and during mechanical ventilation, the same pressure reflects any force applied to expand the chest wall. The chest wall is made

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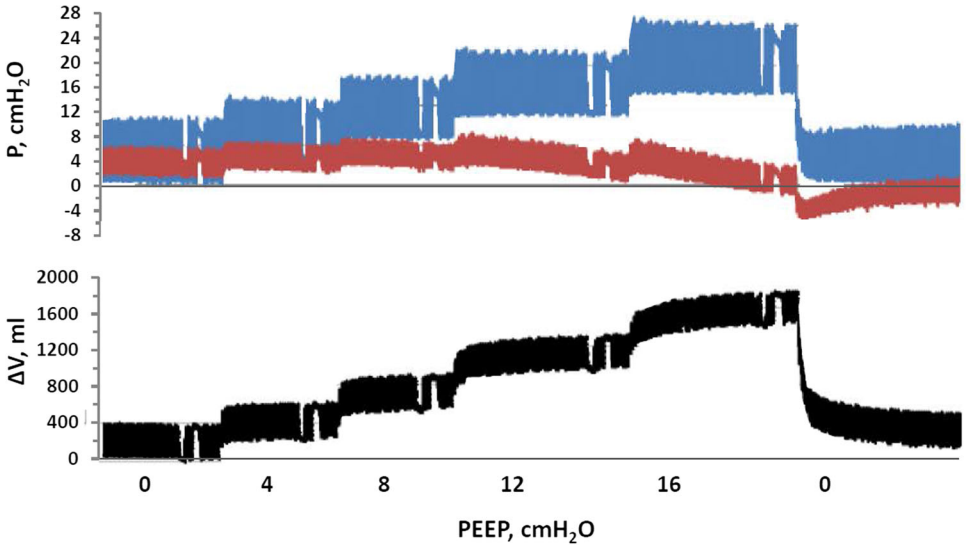

Fig. 1 Airway (*blue*) and esophageal pressure (*red*) and change in lung volume measured by electric impedance tomography (ΔV) (*black*) during an incremental PEEP trial with 0, 4, 8, 12, and 16 cm H_2O and back to ZEEP in an ARDS patient. Note that, at every increase of PEEP, there is an immediate increase in end-expiratory esophageal pressure which subsides slowly at the same time as the lung volume increases (c.f., [13]). When PEEP is lowered to 0 cm H_2O, there is an "undershoot" in esophageal pressure but it then slowly increases back towards the baseline. Any catheter leak was checked for, and can anyway not explain the rise in esophageal pressure at the end of the experiment (data from [7])

up by the rib cage and the diaphragm. At FRC, the rib cage acts as an outward force, and the resting position of the rib cage is well above FRC [1, 3]. This force generates the negative pleural pressure. Thus, at end-expiration, the diaphragm prevents the abdominal organs from encroaching on the thoracic space and influencing the lung in the supine/prone position. It is exposed to the pressure generated by the weight of the abdominal organs, a weight that does not change to any substantial degree when lung volume changes [6]. The abdomen can be considered a partly fluid-filled container, and its content creates a pressure that increases down the abdomen in the gravitational direction, from close to zero in the anterior part to 10–30 cm H_2O in the posterior part, depending on vertical distance and water content. A passive (mechanically controlled) inflation of the lung will move the thoracic wall outwards and the diaphragm caudally, changing the shape of the abdomen and its content. At end-inspiration, when abdominal movement has ceased, any additional pressure to maintain the new lung volume will depend (disregarding the lung) on abdominal tissue characteristics and vertical tissue displacement. There is no reason why any abdominal organ would behave as an elastic tissue; that would require that it is distended during passive lung inflation and causes an increased recoil pressure. The possibility remains that the abdominal wall is distended and recoils but increasing PEEP per se need not increase abdominal pressure [6]. Vertical displacement of abdominal tissue is more likely, but the amount and weight of the displaced tissue is difficult to predict since displacement will initially occur at an isogravitational level and even downwards without any increase in end-inspiratory pressure. We erroneously translate the additional pressure needed to store "energy" at a higher vertical level to reflect chest wall elastance. We thus hypothesize that, in the absence of a recoiling abdominal wall and vertical tissue displacement, pleural pressure need not increase with increasing static (no gas flow) airway pressure [7].

The statement of an unaltered pleural pressure may appear provocative and controversial, not least when it is a frequent finding that pleural pressure (or its substitute, esophageal pressure; see below) increases by a passive tidal inflation of the lung [8–10]. One possible answer to the differing observations is that the mechanical behavior of the abdomen (perhaps also the rib cage) is time-dependent. During passive tidal lung inflation, the chest wall is moving and this requires energy (pressure). This adds to the inspiratory airway pressure and to a calculated respiratory resistance. This resistive component remains for a second or more at end-inspiration, keeping pleural pressure elevated, and is traditionally interpreted as reflecting a chest wall elastance. Lu and co-workers found chest wall elastance to be lower the slower the inspiratory flow [11]. We elaborated on their observation by increasing the PEEP step by step in an ARDS patient, staying at each PEEP level for a couple of minutes until the lung volume stabilized at a new level. We measured airway and esophageal pressures and lung volume changes by electric impedance tomography (see Fig. 1). As can be seen, a step change in PEEP caused an immediate and persisting increase in airway pressure. Esophageal

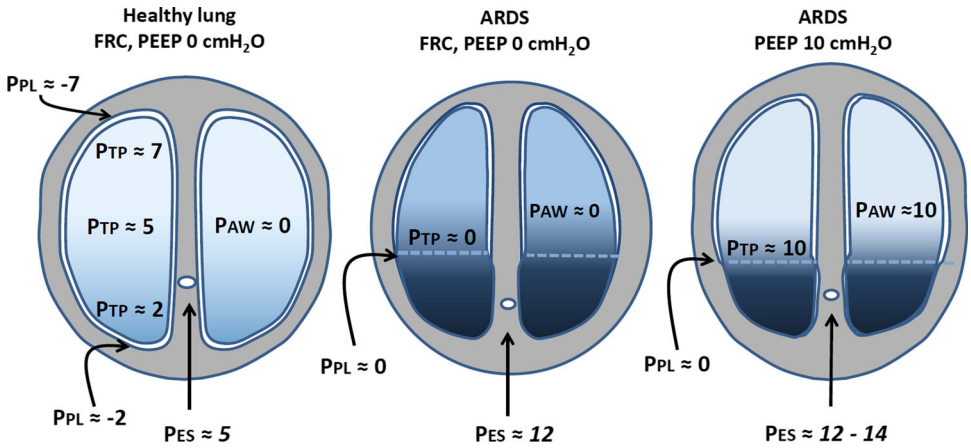

Fig. 2 Schematic illustrations of the gravitational effect on pleural pressure (P_{PL}) during mechanical ventilation in supine position. Pleural pressure is increasing from non-dependent to dependent lung regions and at the zone where collapse starts the pleural pressure can be anticipated to be zero. In more dependent regions with lung collapse and negative transpulmonary pressure (P_{TP}), pleural pressure is positive. Also, note that there is an offset between pleural pressure and esophageal pressure (P_{ES}), which is caused by extrapulmonary and extrapleural effects, where one of many such factors is the weight on the esophageal balloon [18] and also, obviously, that the esophageal pressure is related to one single gravitational level. Airway pressure (P_{AW}) is uniform in gravitational direction in the open lung. *Left panel* Healthy lung (FRC/ZEEP): *white* pleural space indicates that the recoil of the lung and the expanding force of the rib cage create a negative pressure in the pleura, near zero at the base and increasingly negative in more non-dependent regions. Absolute esophageal pressure (in relation to atmospheric pressure) is positive, GŠõ5-Ãcm H_2O [18]. *Middle panel* ARDS, FRC/ZEEP: *dashed line* indicates transitional zone between open and collapsed lung, where pleural pressure is zero. Esophageal pressure is increased, GŠõ12-Ãcm H_2O [17], possibly related to increased mediastinal tissue-Ãand lung weight. *Right panel* ARDS, PEEP 10-Ãcm H_2O, EELV increased above FRC. The rib cage strives outwards and a negative pleural pressure is maintained [7]

pressure, on the other hand, rose initially but then decreased partly or completely to the initial level, while the lung volume increased, somewhat gradually. On the decrease of PEEP, all changes were reversed. Thus, the response by the chest wall to a pressure change is time-dependent. Such an influence by time with decreasing end-inspiratory pressure until a plateau is reached after seconds was noticed long ago and nick-named "creeping" [12]. The time course of PEEP inflation has also been described by Katz et al. [13] and shown to last for minutes. Putensen and co-workers even suggested that it may take up to an hour to obtain a new steady state after increasing PEEP [14].

For practical and safety reasons, it has become common practice to use esophageal pressure as a substitute for pleural pressure to separate lung and chest wall elastance (or its inverse, "compliance"). However, it has been developed and refined in upright subjects and is less reliable in supine subjects [15] with the balloon exposed to many forces that generate pressure [16]. Thus, esophageal pressure is frequently reported as positive at FRC in supine intensive care patients [17, 18]. However, to be representative of pleural pressure at mid-thoracic level, where the lung is normally aerated in most subjects, it should be negative if the subject is breathing at atmospheric pressure, while it may be positive during positive pressure breathing. What always matters is the transpulmonary pressure. Correction for the influence of gravity by subtracting 5 cm H_2O has been suggested [18] (see Fig. 2), but it may well be that additional forces on the esophageal balloon raise the baseline level [16]. It may also be that the slope of the pressure–volume curve is correct so that the calculated lung compliance is also correct [19]. This conclusion does not preclude any of our previous discussion regarding pleural pressure and lung volume.

To summarize, in the mechanically ventilated patient:

1. The lung is not enclosed by an elastic chest wall except when the abdomen is severely expanded; vertical shift of abdominal organs may affect pleural pressure but in the absence of such shift, static pleural pressure need not increase with increase in lung volume;
2. Opposing observations on chest wall elastance may be explained by time-dependent behavior of chest wall mechanics;
3. Alveolar pressure must be higher than extra-alveolar pressure to keep the alveoli open; thus at zero alveolar pressure, relative to atmospheric pressure (FRC), extra-alveolar pressure, assumed to be reflected by pleural and esophageal pressure, must be negative at the same vertical level as there is aerated lung, otherwise the lung would be collapsed.

Conflicts of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

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