Transpulmonary Pressure-Guided Lung-Protective Ventilation Improves Pulmonary Mechanics and Oxygenation Among Obese Subjects on Mechanical Ventilation

Dear Editor,

In their retrospective study on pulmonary mechanics in mechanically ventilated patients with obesity, Rowley and his group pointed out some specific features of the pathophysiology of ventilatory failure in obesity.[1] First, the authors confirmed prior literature showing that, when measured, esophageal manometry and transpulmonary pressure reveal the necessity of a higher PEEP to avoid pulmonary collapse in the context of a high pleural pressure.[2,3]

Pirrone et al[2] showed that without the measurement of a static transpulmonary pressure (ie, the difference between airway pressure and esophageal pressure during an expiratory/inspiratory pause) the tendency is to underestimate the level of PEEP needed to keep the alveoli open at the end of expiration. Pirrone et al[2] selected subjects without respiratory failure, whereas Rowley[1] included subjects with ARDS.

The mechanics of lung recruitment may differ between a healthy lung and a lung with inflammatory edema, due to the presence of a gravitational gradient of pleural pressure in the latter.[4] This concept is illustrated in the picture (Fig 1) where, in the presence of pure ARDS, the benefits of high pressures delivered by the ventilator (recruiting the mid-lower part of the lung where the pleural pressure is high) may be counterbalanced by the risk of overdistention in the ventral parts of the thorax, where pleural pressure is low and transpulmonary pressure high, leading to volutrauma (even when ventilation is set at low tidal volume). Of note, some part of the ARDS lung will be inflamed and consolidated and will not be recruited even at high airway pressures. However, 60% of subjects recruited by Rowley et al[1] had primary acute respiratory failure.

More recently, in a subsequent study from our group, Fumagalli and Santiago[5] demonstrated that an approach based on transpulmonary pressure (in the context of a decremental maneuver to set PEEP at the best global compliance) was effective in improving oxygenation and lung compliance in obese patients with ARDS. The study confirmed that PEEP selected with the ARDSnet table was low and resulted in a negative transpulmonary pressure at end expiration, with consequent lung collapse.

As of today, it is unclear if the same concepts valid for lean patients with ARDS (gravitational gradient of pleural pressure, overdistention of ventral lungs, risk of volutrauma and barotrauma at high level of PEEP) are applicable to morbidly obese patients. These physiological results, although based on a small number of patients, seem to suggest otherwise.

In a recent study, Florio et al[6] demonstrated severe derangement of respiratory physiology even in the spontaneously breathing otherwise healthy obese individual. It was found that even at rest subjects with obesity needed substantial swings of negative pleural pressure to initiate inspiratory flow, probably due to persistent tidal collapse of small airways. From a respiratory point of view, these patients would benefit from permanent positive end-respiratory pressure in the form of CPAP during their daily life, even in the absence of a formal diagnosis of any disease.

A speculation may be that even in the presence of ARDS lung atelectasis induced by elevated pleural pressure is predominant in the patient with obesity, and the gravitational gradient of pleural pressure is less relevant, hence the positive response to high levels of PEEP. However, more studies are needed to confirm this hypothesis.

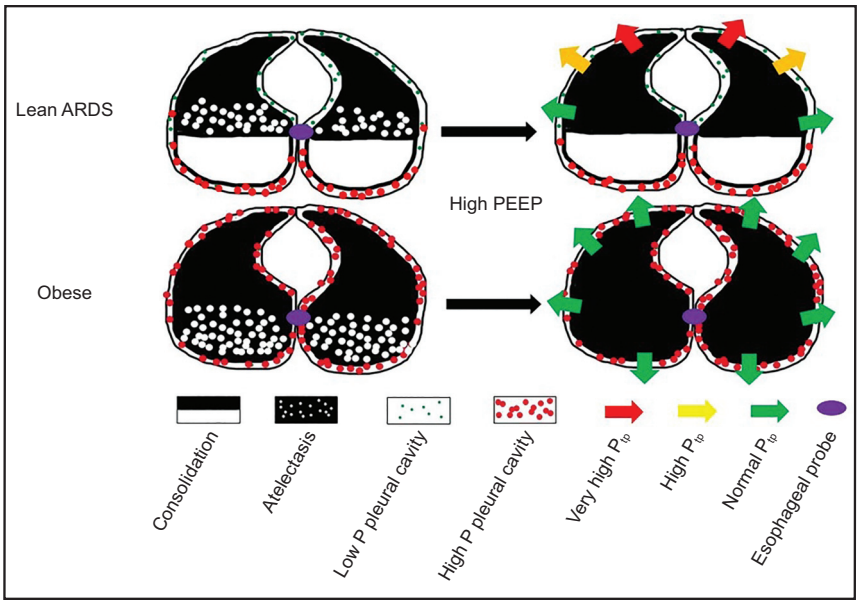

Fig. 1. Different effect of high PEEP in a model of ARDS (above) versus respiratory failure in the obese patient (below). In the model above, the gravitational gradient of pleural pressure (high pleural pressure in the dorsal lung, high density red dots, vs low pleural pressure in the ventral lung, green dots) leads to overdistention in the dorsal parts of the lung due to very high transpulmonary pressure (the difference between airway pressure and pleural pressure). Moreover, the uniform white part of the lung is consolidated and will not be recruited even at high pressures. In the obese model, very high pleural pressure is uniformly distributed and causes atelectasis, which is reversed by high PEEP. Since there is less gravitational gradient, pressures from the ventilator are evenly distributed, with less risk of overdistention. P_{tp} = transpulmonary pressure.

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