Editorial

Closing, opening and reopening: the difficult coexistence

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Volume-controlled ventilation (VCV) has always been considered as protective ventilation, in particular during spontaneous breathing, being able to avoid the administration of injurious tidal volumes (V_t) to the patient. In fact, spontaneous efforts increase transpulmonary pressure (P_l) only during pressure-regulated ventilations (1). Despite this difference in terms of P_l, the conditions of lung parenchyma determine the damage to the lung tissue and the onset of ventilator induced lung injury (VILI).

In normal lung the pressures applied to a local region of the pleura are homogeneous and distributed over the whole lung (fluid-like behavior), whereas negative pleural pressures (P_{pl}) generated by diaphragmatic contraction are rather concentrated in dorsal parts (solid-like), once lung has been injured (2). Furthermore, when P_l is applied to the lung, a counterforce of equal intensity is developed (lung stress), whereas the associated lung deformation is called strain. These two forces are directly correlated by the specific elastance (El_{sp}), which reflects the intrinsic mechanical characteristics of the lung parenchyma (3). This concept is a recent introduction, but it has an important clinical relevance right away; in fact, El_{sp} is peculiar in each species: in pigs is about half that in human and consequently a plateau pressure (P_{plat}) of 30 cmH_2O in pigs would approximately correspond to 60 cmH_2O in humans. Therefore the differences on El_{sp} between species must be taken in account when considering the results of experimental studies (4,5).

In injured lung, dorsal zones are collapsed or cannot be inflated; therefore they can be stressed, but not strained. Consequently, in this inhomogeneous distribution of ventilation, the healthy regions have to sustain a greater stress that promotes the development of biotrauma. When the stress and strain from mechanical ventilation overcome the lung's ability to adapt, mechanical ventilation becomes injurious, thus ventilator settings must take into account both gas exchange and stress and strain. The latters can be modified by different interfaces and positions. Lopez-Aguilar and colleagues found that the combination of low dose partial liquid ventilation with perfluorocarbons and prone positioning improved lung function while inducing minimal stress in an animal model of acute lung injury (ALI) (6).

The effects of spontaneous breathing in different severities of lung injury were recently investigated. Patient efforts generate negative change in P_{pl}, with a large difference between mild and severe lung injury. In the first, spontaneous efforts reduce just before peak airway pressure was reached; in contrast, in severe lung injury the respiratory muscles continue to contract until the end of inspiration, and patient active expiration increase airway pressure above plateau. Consequently, the highest P_l was generated in severe lung injury. These findings were supported by histological samples: the alveolar damage was less pronounced in mild lung injury models. On the contrary, in severe lung injury more hyaline membrane formation and alveolar hemorrhaging with more severe neutrophil infiltration into the alveoli and interstitium were observed (7).

Another important effect of spontaneous breathing is that local negative P_{pl} generated by diaphragmatic contraction is not uniformly transmitted. Using electrical

impedance tomography during controlled ventilation, simultaneous inflation of the different lung regions was observed, whereas, when spontaneous efforts were present, they caused an early inflation of dependent lung regions. The latter was accompanied by simultaneous deflation of nondependent region, indicating movement of gas from nondependent to dependent lung regions; because this was not associated with alterations in tidal volume it indicates a pendelluft phenomenon. After induction of muscle paralysis a progressive reduction of inhomogeneity of ventilation was observed, confirming that extent of the pendelluft was proportional to the intensity of the respiratory effort (8).

Protti and colleagues introduced the difference between static and dynamic strain. Lung tissue deformation due to application of positive end-expiratory pressure (PEEP) is called static strain, whereas tidal ventilation is a dynamic process, as the energy is cyclically applied to the lungs; lung deformation due to tidal volume is called dynamic strain. In particular, if a large force is applied (dynamic strain), tension will concentrate and rupture will possibly occur. Conversely, if the force is applied on pre-stressed fibers (static strain), distension will be more uniform and rupture less common. Therefore PEEP-induced V_t may have had its own beneficial effect (9). Airway pressure release ventilation (APRV) is a ventilator mode characterized by an open-valve CPAP with a brief pressure release and that in previous animal studies was associated with higher V_t and P_{plat}. In an extrapulmonary lung injury model APRV was compared with low V_t ventilation and despite the greater P_{plat} and V_t in the APRV group, P_l was similar to that of protective ventilation group, demonstrating that the increases in P_{plat} in APRV reflects an increase in P_{pl}. The authors concluded that APRV represents a safe and effective ventilation mode in patients at risk for the development of extrapulmonary lung injury (10). The same group of authors recently studied the impact of dynamic strain on tissue injury in both normal and acutely injured lung tissue model ventilated in APRV. The normal tissue was not seriously injured as long as dynamic strain remained low. However, when high P_{plat} was combined with high dynamic strain, this caused significant damage to normal tissue and exacerbated damage to the injured tissue. This study reaffirmed that, even in the presence of high P_{plat}, the use of APRV promoted recruitment and stabilized the alveoli, reducing VILI (11).

Preserved spontaneous breathing during acute respiratory failure has been recently discussed. In ten patients equipped with esophageal catheters inspiratory muscle pressure (Pmus) was almost zero during controlled mechanical ventilation (CMV), although application of decreasing levels of pressure support led to a progressive increase of Pmus, under the assumption of identical mechanical properties of the respiratory system (compliance, resistance, flow and volume). As a result, P_l swings were similar overall between CMV and all the levels of pressure support ventilation (PSV) applied, although with a poor correlation. This difference was explained by the corresponding difference in the inspiratory flow rates between the different ventilator modalities, indicating that the resistive pressure drop caused this difference. If the analysis was focused only on values for which the inspiratory flow was similar, this resulted in a tight correlation. In the same way, the swings of esophageal pressure (P_{es}) were positive during CMV, but became negative during PSV and progressively lower for decreasing levels of support. Similarly, alveolar pressure (P_{alv}) progressively decreased from CMV through the different levels of PSV and P_{alv} was lower than the set PEEP, if a low level of support was applied. Finally, during spontaneous breathing, under identical mechanical properties of the respiratory system and for the same inspiratory flow, P_l will be the same during CMV and PSV. Similarly, if the lung is at the same volume, the pressure across the alveolar wall, which is due to the elastic recoil of the lung, will not differ between CMV and PSV. Consequently, a negative pressure will surround the alveoli and during PSV P_{alv} will become very negative to overcome the resistive pressure drop; negative P_{alv} values and their consequences on fluid shifts are potential mechanisms by which spontaneous breathing might induce lung injury (12).

Yoshida and colleagues reported that VCV prevented increases in V_t and in P_l calculated using P_{es}. The main findings were that the limitation of V_t and P_l by VCV could not eliminate harm from spontaneous breathing, unless the level of spontaneous effort was lowered and local dependent lung stress was reduced (13). We congratulate the authors and recognized the effort to provide more evidence in this field. In fact, these results confirmed that stress and strain are inadequately estimated by P_{plat} and V_t; therefore they must be taken in account when setting mechanical ventilation, especially in spontaneous breathing patients, to avoid further VILI.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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