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WHAT'S NEW IN INTENSIVE CARE

The ten pressures of the respiratory system during assisted breathing

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The mechanics of breathing are defined by the pressures which act on different parts of the respiratory system. Since, among them, only airway opening pressure (Pao) is typically monitored during assisted mechanical ventilation, their interplay and interpretation may confound clinicians. The aim of this paper is to review ten pressures during assisted breathing, focusing on pressure support ventilation (Fig. 1 ESM) in acute respiratory distress syndrome (ARDS). In addition to Pao, we identified the pressures reported in Table 1 ESM as the most relevant.

Since mechanical ventilator and respiratory muscles are pressure generators arranged in series, their sum at any time t defines the total pressure across the respiratory system during assisted mechanical ventilation (Fig. 1), according to the following equation:

$$\begin{aligned} \text{Pao}(t) + \text{Pmus}(t) = \text{PEEP} + (\text{Ers} \times V(t)) \\ + (\text{Rrs} \times \text{Flow}(t)), \end{aligned} \quad (1)$$

whereby PEEP represents Pao at end-expiration, Ers is the respiratory system elastance, V is the tidal volume, Rrs is the resistance of the respiratory system and Flow is the airflow. In equation [1], $(\text{Ers} \times V(t))$ and $(\text{Rrs} \times \text{Flow}(t))$ represent the elastic and resistive components of the pressure generated across the respiratory system by tidal volume inspired and exhaled during the respiratory cycle. During unassisted spontaneous breathing, the maximal value of Pao is equal to PEEP (if present) or to the atmospheric pressure, and, hence, PEEP (or zero) + Pmus is the corresponding total pressure across the respiratory system. During inspiration, Pmus decreases the pressure within the pleural cavity (Ppl), so that Pmus is the difference between the pressure of the relaxed chest wall and the change in Ppl at a given gas volume. In clinical practice, bedside assessment of Pes [1] can be used as surrogate to Ppl, and chest wall elastance (Ew) may be measured by switching the patient to controlled ventilation and dividing the change in Pes by the tidal volume. Thus, Pmus can be calculated at any time t as:

$$\text{Pmus} = V(t) \times E - \Delta\text{Pes}(t), \quad (2)$$

whereby V is the tidal volume, Ew is the chest wall elastance and ΔPes is the esophageal pressure change from baseline end-expiratory level. This allows measuring patient's total inspiratory muscular pressure and work of breathing at the bedside. Negative Ppl/Pes swing is then transmitted from visceral pleura to lung structures and to the alveolar space. This, in the presence of Rrs >0 $cmH_2O/l/s$, decompresses alveolar gas, reducing its pressure and decreasing Palv. When Palv becomes lower than Pao, which corresponds to atmospheric pressure or to PEEP, the Pao–Palv gradient generates an airflow inversely proportional to Rrs and the tidal volume enters the alveolar space. This is true for aerated alveolar units and for those which cross the opening pressure along inspiration. Instead, if an alveolar unit is collapsed at end inspiration, no airflow is generated despite a positive Pao–Palv value. When the tidal volume enters the alveolar space, increasing the mass of gas per unit of lung volume, Palv increases. Thus, during inspiration, the Pao–Palv gradient progressively increases and then decreases (depending on the temporal pattern of Pmus), generating a decelerated inspiratory flow. We must also notice that, during spontaneous breathing, the difference between intravascular capillary pressure and Palv increases, posing diseased alveoli with large surface tension at risk for transmural vascular fluid exudation and collapse. At the end of inspiration (zero flow), the difference between Pao and the absolute Pes/Ppl value is driving P_L (i.e., the pressure across lung structures due to

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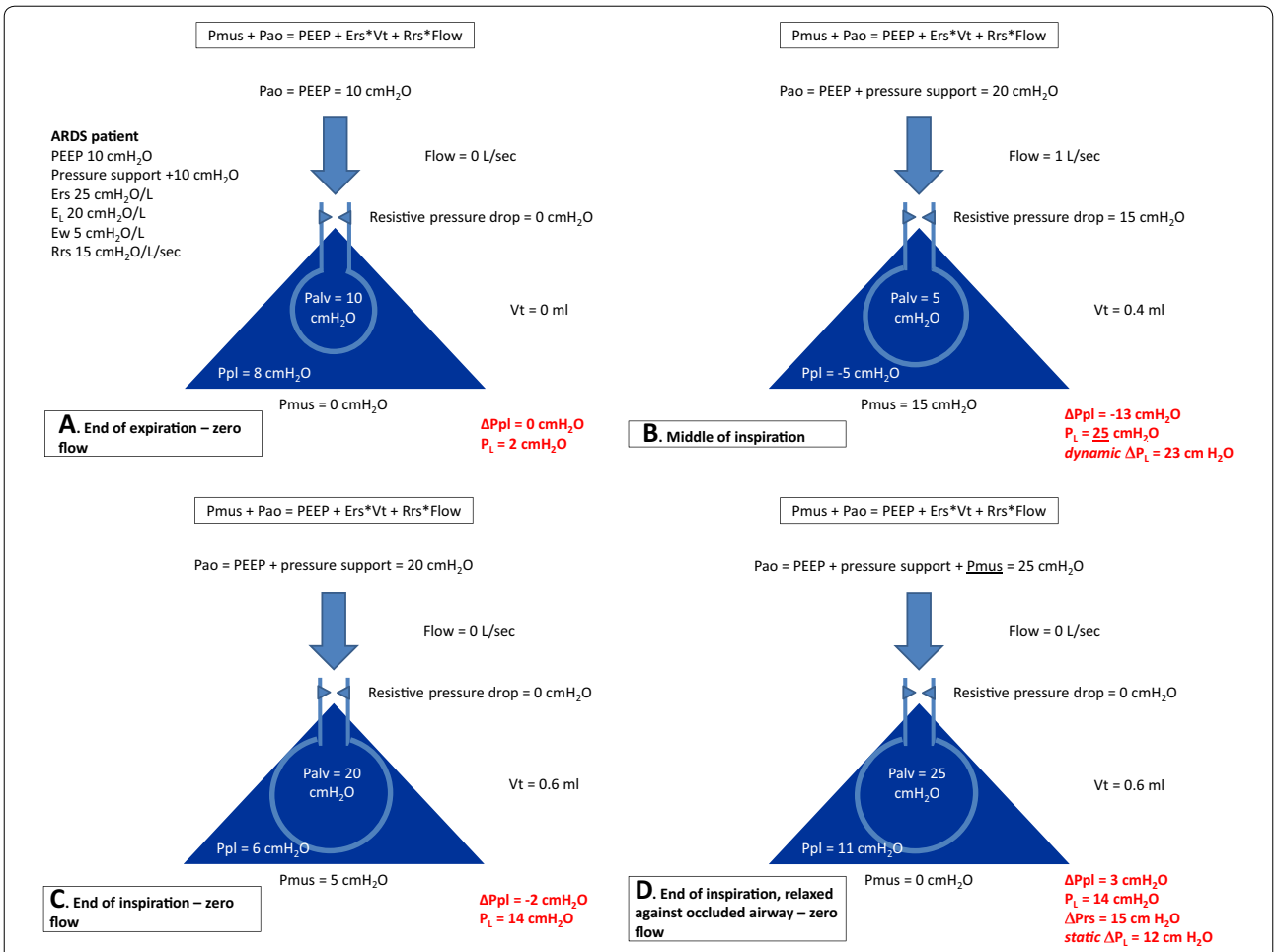

Fig. 1 Pressure interplay during spontaneous assisted breathing in acute respiratory distress syndrome (*ARDS*). Schematic representation of the most relevant pressures within and across the respiratory system structures in an active ARDS patient undergoing pressure support ventilation with the following settings: positive end expiratory pressure (*PEEP*) = 10 cmH_2O; support level = 10 cmH_2O. At the end of expiration (zero flow, **a**), the patient is relaxed (Pmus = 0 cmH_2O), and alveolar pressure (*Palv*), airway pressure (*Pao*) and PEEP are equal. Then (**b**), active inspiration triggers the ventilator and pressure support is delivered, raising Paw to the PEEP + support level = 20 cmH_2O, while Palv increases at a slower rate, inversely proportional to the respiratory system resistance (*Rrs*). **b** Patient's maximum effort and pleural pressure deflection (Δ*Ppl*), Pmus and dynamic change of transpulmonary pressure (ΔP_L) reach their highest values. At end inspiration (i.e., when inspiratory flow crosses the zero value, **c**), the tidal volume (*Vt* = 0.6 l) is fully delivered but the patient muscles are not yet completely relaxed (note negative ΔPpl and positive Pmus) and Pao still corresponds to the sum of PEEP and pressure support = 20 cmH_2O. Finally (**d**), when the patient relaxes against an occluded airway at end inspiration, end inspiratory Pmus is transmitted back to the inner structures, increasing Ppl, Palv and Pao despite zero flow and unchanged *Vt*. *Ers* respiratory system elastance, E_L lung elastance, *Ew* chest wall elastance, Δ*Prs* static driving respiratory system pressure

V_T), while Pplat and ΔPao are negligible. When pressure support is added to spontaneous breathing, the same principles apply but total pressure across the respiratory system and P_L increase, generating additional flow and volume. During pressure support, if inspiratory airflow exists after the end of Pmus, Pes/Ppl can turn to positive swings along inspiration, the ventilation being a hybrid of active (during the first part) and passive (towards the end) phenomena.

A post hoc analysis of data from 3562 ARDS patients enrolled in nine randomized clinical trials suggested that, during controlled mechanical ventilation, respiratory system Δ*P* (ΔPrs) was the one ventilation variable that best stratified VILI risk [2]. For the purpose of this analysis, ΔPrs was defined as the difference between measured end-inspiratory airway occlusion pressure and extrinsic PEEP. Because ΔPrs varies with the number of open and recruited lung units, it is a surrogate measure

of parenchymal stress as well as a marker of the severity and extent of the underlying disease process. Respiratory physiologists generally equate lung parenchymal stress with the elastic recoil pressure of the lung, which under no flow conditions equals P_L, defined by the difference between Pao and Ppl. While it is possible to estimate Ppl with esophageal manometry, there is some controversy about measurement bias in supine recumbent patients with edematous lungs, because the esophagus may be compressed by the weight of the heart and mediastinal contents. This uncertainty raises questions if Pes-derived estimates of end-expired P_L at zero flow should guide PEEP management in supine patients with ARDS. A clinical trial, which is to address this concern, is nearing completion. Nevertheless, in the absence of lung and chest wall distortion by the respiratory muscles, ΔPes provides a reasonable estimate of chest wall ΔP. At present, it is unclear if esophageal manometry-guided estimates of lung ΔP (ΔP_L) would provide a more sensitive and specific estimate of VILI risk than ΔPrs.

Besides Pao and PEEP, which are set by the clinicians and displayed on the screen of any ICU ventilator, other more specific pressures can be monitored. Proportional assisted ventilation (PAV) provides the patient with a direct feed-back to her/his effort. By using Eq. 1, the ventilator continuously computes (and delivers) the pressure assistance in proportion to patient effort. Furthermore, in PAV, brief end-inspiratory occlusions are performed and hence ΔPrs can be monitored in this unique SB mode. Once switched from lung protective mechanical ventilation to PAV, lung compliance is higher, which results in higher V_T while ΔPrs remains in the safe range, suggesting that patients could control ΔPrs [3]. Assessing patient effort is important to accommodate the ventilator assistance during pressure support. Pmus can be monitored in different ways. It can be computed breath by breath by fitting the equation of motion of the respiratory system to flow and Pao tracings. Maximal Pmus can be measured breath by breath as the difference between maximal Pes deflection and elastic recoil of the chest wall [4]. The Pmus index has been proposed to estimate Pmus at end inspiration from Pao during pressure support, as the difference between the sum of set Pao and PEEP and elastic recoil of the respiratory system during end-inspiratory occlusion [5]. Its measurement requires full relaxation of the patient. P0.1, which is Pao measured within the first 100 ms after airway occlusion, and is implemented automatically in many ICU ventilators, can be used as a surrogate of the work of breathing [6] and to titrate the level of pressure support ventilation during weaning [7].

In conclusion, during SB, monitoring specific pressures that are not routinely displayed by commercially available ICU ventilators might be key to avoiding excessive injury and to fully exploit clinical benefits.

Electronic supplementary material
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Compliance with ethical standards

Conflicts of interest
The authors declare no conflict of interest.

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