Introduction

Acute respiratory distress syndrome (ARDS) is a major global public health problem. The mortality of ARDS is very high at approximately 40% (1). However, treatment measures are limited. Mechanical ventilation (MV) is one of the main treatment measures.

In recent years, sedation and paralysis have led to a greater understanding of the adverse effects of MV in patients, and assisted spontaneous ventilation has received attention (2,3). Spontaneous breathing can reduce neuromuscular damage, redistribute pulmonary blood flow, and improve oxygenation (4,5). In contrast, uncontrolled spontaneous breathing increased ventilation heterogeneity and redistributed, and the inspired tidal volume (VT) led to self-inflicted lung injury (6,7). Therefore, monitoring during spontaneous breathing is vital and indispensable. Recent studies have also highlighted how the driving pressure [i.e., ratio of VT to respiratory compliance (Crs), and when there is no spontaneous compliance, it is equal to plateau pressure (Pplat) minus positive end-expiratory pressure (PEEP)] affects the prognosis of mechanically ventilated ARDS patients (8). Driving pressure represents the strain of the lung and the target for limiting the inspiratory volume. Unfortunately, researchers generally believe that Pplat and driving pressure can only be accurately measured during paralysis. Moreover, if we obtain airway pressure without oesophageal manometry, the pressure is underestimated. Interestingly, with the development of MV, studies have shown that we could overcome these shortcomings (9).

In this review, we briefly describe the benefits and
damages of spontaneous breathing during MV, with a focus on using driving pressure for lung protection ventilation during spontaneous breathing. We present the following article in accordance with the Narrative Review Reporting Checklist (available at http://dx.doi.org/10.21037/apm-19-284).

Methods

Information used to write this paper was collected from the PubMed databases using the following key words: spontaneous breathing; monitor; driving pressure; ventilator-induced lung injury.

Discussion

Pros and cons of spontaneous breathing

It has been shown that spontaneous breathing improves ventilation-perfusion matching, reduces diaphragm damage, and improves oxygenation (2,10). Recently, Corral reported that diaphragm activity was closely related to the occurrence and development of ventilator-induced diaphragmatic dysfunction (VIDD) (3) and suggested that if a certain level of spontaneous breathing was maintained during MV, VILI could be reduced due to diaphragm unloading by reducing the harmful effects of controlled mechanical ventilation (CMV). However, spontaneous breathing also has adverse effects during MV, as evidenced by several studies. Due to the specificity of the lungs of patients with ARDS, VT can be out of control, and uneven distribution can be aggravated by negative pleural pressure. Additionally, pleural pressure combined with alveolar pressure may also aggravate pulmonary interstitial oedema (11,12). Moreover, these adverse effects might aggravate lung injury. Controlling VT and transpulmonary pressure can reduce barotrauma and volumetric injuries and thus reduce the damage caused by increased spontaneous effort (13). Interestingly, Yoshida et al. recently showed that only when the level of spontaneous breathing effort and local-dependent lung pressure are low could the risk of spontaneous breathing be eliminated by limiting VT and oesophageal pressure in the volume-controlled (VC) mode (11). In VC, spontaneous efforts to produce locally dependent pulmonary pressure increase, even at the same VT and oesophageal pressure settings, resulting in more than twice the amount of ventilation and tidal recruitment in the posterior region. Oesophageal pressure cannot accurately assess local pleural pressure when there is a spontaneous effort in patients with ARDS. More interestingly, spontaneous breathing does not contribute to severe lung injury but instead contributes to mild lung injury.

Indeed, Papazian et al. reported that early administration of neuromuscular blockers improves outcomes in patients with severe ARDS (14). However, there are still different perspectives on whether early continuous neuromuscular blockade is beneficial. More recently, in an RCT containing 1,006 patients with moderate-to-severe ARDS, there was no significant difference in mortality between the continuous neuromuscular block group and the mild sedation group (15).

In summary, spontaneous breathing is beneficial in mild ARDS, but the case of moderate-to-severe ARDS. One of the possible causes is that there is proper spontaneous breathing in mild ARDS, while spontaneous breathing is out of control in moderate to severe cases, which suggests that spontaneous breathing can only have a real protective effect if it has better monitoring indicators.

Spontaneous breathing monitoring

To better understand the forces generated by the patient, it is necessary to monitor respiratory muscle activity. There have been many studies on respiratory muscle monitoring, including the following methods. (I) Oesophageal manometry: oesophageal manometry can assess respiratory effort during MV, improve human-machine coordination, and serve as an indicator of weaning (16-18). It has considerable potential to improve clinical outcome in patients with ARDS as an early detector of the risk of lung injury from MV and spontaneous effort (18). (II) P0.1: the occlusion pressure, which is the negative pressure generated when the occlusion is 0.1 seconds, is also called P0.1. This pressure can respond well to respiratory drive and thus prevent P-SILI and serve as an indicator of weaning (19). (III) Monitoring of diaphragm function: in addition to P0.1, the inspiratory effort can be estimated noninvasively by diaphragm ultrasound. Ultrasound measures of diaphragm thickening in the zone of apposition may be useful for predicting extubation success or failure during spontaneous breathing trials (20). Moreover, the neutrally adjusted ventilation assist mode gives patients corresponding support according to diaphragm electrical activity (21). However, these monitoring methods have their own limitations; for example, oesophageal pressure is invasive.
and expensive, and due to the specificity of ARDS, oesophageal pressure only represents the average level of negative pleural pressure and cannot reflect the whole lung condition.

The monitoring of diaphragm function is easily interfered with by many factors and is prone to errors. More importantly, these monitoring methods only reflect respiratory muscle strength, and the patient’s lung expansion is also under pressure provided by the ventilator. Excessive lung expansion is the most critical cause of VILI. Therefore, there is an urgent need for a reliable and straightforward indicator to comprehensively reflect the expansion of the lungs to prevent the occurrence of lung injury. The emergence of the driving pressure makes up for this deficiency.

**Development of driving pressure**

CT brings us closer to the real face of ARDS. According to the results of the CT examination, the concept of “baby lung” has been proposed (22). More importantly, the compliance and functional residual capacity of the baby lung are proportional (23), and VILI is related to its excessive stress and strain. To prevent VILI, we must limit VT based on the functional residual capacity of the baby’s lungs rather than the ideal weight (24-26). Recently, Amato et al. reported that the Crs-standardized VT was a better predictor of prognosis in patients with ARDS than VT alone. The ratio of the two in the absence of the effect of spontaneous effort was defined as the driving pressure (8).

Subsequently, the LUNG SAFE study is a prospective study of ICUs in 50 countries. As a result, when the driving pressure is higher than 10 cmH₂O, the mortality rate increases linearly (27). Similar results to these studies, a meta-analysis reported by Hiroko Aoyama that ARDS patients who underwent MV at lower driving pressure had lower mortality (28).

In recent years, many pieces of evidence (8,27-31) have shown that driving pressure might be one of the essential indicators of lung protection during MV. However, it still has many disadvantages; for example, driving pressure should be used during paralysis without spontaneous breathing, and it does not consider the effects of the chest wall (32,33). However, Amato et al. found that in severe ARDS patients, the pressure to inflate the lungs mainly derives from the driving pressure, and the driving pressure might replace the transpulmonary driving pressure. Moreover, the cutoff of driving pressure was not explicit, and no RCT had indicated the feasibility of driving pressure. In any case, the concept of driving pressure means that we have a better understanding of protective ventilation.

**Application of driving pressure in spontaneous breathing**

MV combined with spontaneous breathing is a combination of positive pressure ventilation and negative pressure ventilation, but the essence is the same for lung expansion, so we believe that driving pressure can be used as a predictor for such patients. P0.1, the diaphragm point or the oesophageal pressure reaction are all related to muscle strength, and the driving pressure response is the result of the final force of the lung, that is, the strain of the lung. Therefore, monitoring the driving pressure may be more intuitive. However, a reliable driving pressure could be obtained correctly without spontaneous breathing, and spontaneous breathing efforts to produce pleural pressure could not be ignored. How could we accurately assess the driving pressure of patients with spontaneous breathing?

Zhou et al. measured the platform pressure by switching to the capacity control mode and then subtracted the previously monitored positive end-expiratory pressure (PEEP) from the platform pressure as the driving pressure in the airway pressure release ventilation (APRV) group. They found that early application of APRV was similar to driving pressure compared to low VT ventilation but improved oxygenation and Crs. Additionally, the platform pressure of the APRV group is lower, and the MV time and ICU residence time are shortened (34). Additionally, Tomas et al. also evaluated the driving pressure in the ARPV mode. They compared the effects of PEEP on lung inflammation in mild-to-moderate ARDS pig models with or without spontaneous breathing, with a limit of driving pressure less than 15 cmH₂O (35). However, Taylor et al. thought that the real driving pressure should be calculated as P_{high}−(intrinsic PEEP+P_{low}). Otherwise, the DRIVING PRESSURE is overestimated in APRV mode because the effect of intrinsic PEEP is ignored (36).

Recently, Bellani et al. reported that a brief inspiratory hold during pressure-supported ventilation can achieve a relatively accurate Pplat and that the inspiratory effort does not significantly affect the airway pressure waveform, which is only noticeable when the inspiratory is interrupted. Moreover, inspiratory pauses during positive pressure ventilation with spontaneous breathing efforts result in
increased pressure in the airway and increased platform pressure. Therefore, they suggest that in the case of spontaneous breathing, inspiratory hold can also be used to measure Pplat during pressure support ventilation (PSV), thereby deriving lung compliance and platform pressure (9). Whether we could obtain a relatively accurate platform pressure, it is essential to know which is the actual platform pressure, which reflects the extraordinary impact of VT and spontaneous breathing.

In addition to PSV, proportional assist ventilation with load-adjustable gain factors (PAV+) is a new mode of ventilation in which the ventilator provides proportion assistance to the muscle strength by calculating the muscle strength required to overcome elastic and airway resistance during spontaneous breathing; these are updated several times per minute during PAV ventilation (37). We used the driving pressure formula (equal to Vt/Crs) to obtain the dynamic driving pressure directly. Recently, Vaporidi et al. calculated driving pressure as the Vt-to-Crs ratio when treating PAV+ patients. The results show that high driving pressure is closely related to low compliance. More importantly, when the compliance is less than 30 mL/cmH\textsubscript{2}O, a driving pressure greater than 15 cmH\textsubscript{2}O will occur. Unfortunately, similar to other research studies, those authors did not evaluate chest wall mechanics and intrinsic PEEP yet (38).

The above studies have shown that people have begun to evaluate the driving pressure during MV in the presence of spontaneous breathing, and it seems that the driving pressure can be conveniently and accurately assessed, although there is still much to be discussed.

Conclusions

In summary, this discussion of the advantages and disadvantages of spontaneous breathing reflects a deeper understanding of respiratory physiology, and the debate regarding driving pressure also reflects people’s more in-depth knowledge of VILI.

Driving pressure is a useful and valid measure of lung strain in MV patients with spontaneous breathing. Monitoring the driving pressure may be more conducive to lung protection ventilation. Since this parameter is now readily available, we can apply it to clinical practice.

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Footnote

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