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Perspective

Mean airway pressure has the potential to become the core pressure indicator of mechanical ventilation: Raising to the front from behind the clinical scenes

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ABSTRACT

Mean airway pressure (Pmean) is a common pressure monitoring parameter of mechanical ventilators that is closely correlated with mean alveolar pressure and represents stresses applied to the lung parenchyma during ventilation. Pmean is determined by the peak inspiratory pressure, positive end-expiratory pressure (PEEP), and inspiratory-to-expiratory time ratio with dynamic and real-time characteristics, which represents mechanical power affected by the ventilator mode. Additionally, Pmean is an important parameter that affects hemodynamics. Tidal forces and PEEP increase pulmonary vascular resistance (PVR) in direct proportion to their effects on Pmean. Therefore, Pmean is increasingly considered to be related to the prognosis of patients on mechanical ventilation. We propose a 3P strategy (Pmean, central venous pressure [CVP], and perfusion index [PI]) which is indicated to achieve circulation protection mechanical ventilation with flow priority. Titrating the appropriate CVP and meeting PI to ensure tissue perfusion with a lower Pmean are the core purposes. Pmean links the circulatory and respiratory systems and is expected to become a potential parameter for intelligent ventilation.

Respiratory movement affects not only air flow but also blood flow. In spontaneous breathing, changes in air flow in the lungs are synchronized with blood flow. Blood flow changes periodically with respiratory movement that can affect changes in blood flow in the entire circulatory system and has different effects on left and right heart functions. At the same time, periodic changes in pulmonary blood flow also significantly affect the lung function [1]. In short, air flow and blood flow accompany each other. Blood flow depends on the existence of air flow, while air flow improves the distribution of blood flow. When positive pressure ventilation is used, normal respiratory physiology is disrupted. Tidal volume and an increased pleural pressure result in increased pulmonary circulation resistance, decreased right ventricular preload, increased afterload, and a decrease in both preload and afterload in the left heart. As a result, the blood flow of the pulmonary and systemic circulation is affected by clinical consequences such as decreased oxygenation, decreased blood pressure, and poor tissue perfusion [2]. This is a prominent occurrence in patients with severe acute respiratory distress syndrome (ARDS) who receive mechanical ventilation. Even if the principle of small tidal volume lung protection is applied, a sharp deterioration in circulation and perfusion will inevitably occur, especially in severe ARDS [3]. Finding a simple, objective, and dynamic real-time monitoring of airway pressure indicator is thus necessary for the clinical realization for both pulmonary and circulatory protection.

In the resting state, airway pressure is equivalent to alveolar pressure, which represents the pressure generated by overcoming the elastic retraction force of the respiratory system. Studies have shown that the measurement of alveolar pressure is very difficult; however, because of the linear relationship between airway pressure and alveolar pressure, we can indirectly obtain alveolar pressure through ventilator-mediated measurement of airway pressure [4]. At present, there are several indica-

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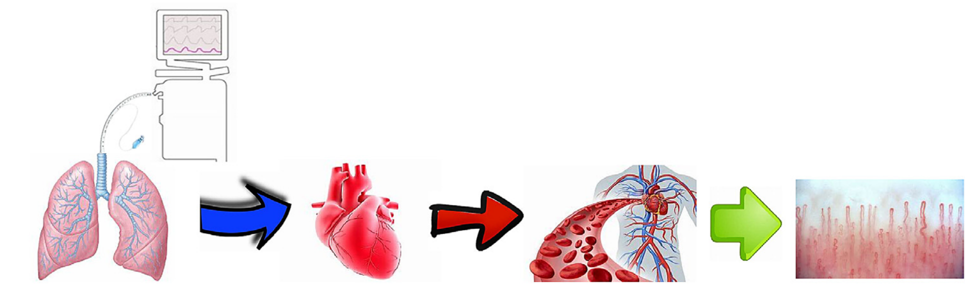

Fig. 1. Flow prioritized 3P (Pmean, CVP, and PI) Circulation Protective Ventilation Strategy. Gas accompanied by blood flow. If the Pmean increases >10 cmH_2O, the CVP may increase to >10 mmHg based on the cardiopulmonary interaction. Then, the blood flow will be disrupted, leading to perfusion injury based on PI < 1.4. ARDS: Acute respiratory distress syndrome; CVP: Central venous pressure; ICU: Intensive care unit; PI: Perfusion index.

tors that reflect airway pressure. For example, the plateau pressure (Pplat) reflects the pressure level in the alveoli at the end of inspiration, but it does not indicate changes in real lung volume. Moreover, Pplat is measured at the end of inspiration using an inspiratory hold maneuver under conditions of controlled breathing. Transpulmonary pressure is defined as the difference between alveolar pressure and pleural pressure, which reflects the degree of expansion of lung tissue. Therefore, it represents stress, which is related to the degree of lung injury. When using this indicator, it is essential to complete the measurement of esophageal pressure and to measure and titrate the transpulmonary pressure of the inspiratory and expiratory states, respectively. Driving pressure (Pdriv) is defined as the difference between Pplat and positive end-expiratory pressure (PEEP), which reflects the elasticity or compliance of the lung. Similarly, this value can only be obtained after manually measuring the platform pressure. The common feature of all the above parameters is that they only describe the respiratory mechanics of the inspiratory phase. Either different types of manual measurements are required or the measurements rely on other pressure measuring devices and methods.

Mean airway pressure (Pmean) is defined over a given interval of time using the area under the curve of the pressure time curve for one breath divided by the total cycle time. Because airway pressure changes in real time with alveolar pressure, it is particularly important to monitor changes in airway pressure in real time, under different ventilation conditions and during different breathing phases, to clarify changes in lung and thorax elastic resistance and compliance. Pmean is a more objective indicator that is directly measured by the ventilators themselves [5]. In addition, it is related to the oxygenation index [6]. More importantly, according to the formula of mechanical power, Pmean is an important parameter involved in constituting mechanical power [7]. High Pmean indicates that the patient has higher mechanical energy power. Taken together, Pmean is a potential indicator of lung stress. A recent multicenter study showed that in a group of mechanically ventilated ARDS patients, Pmean was independently associated with 90-day mortality and correlated with Pplat or Pdriv, which had predicted mortality like the implication of Pplat or Pdriv [8]. From this point of view, Pmean also has potential prompt significance when spontaneous breathing is too strong or breathing drive is too high.

More importantly, Pmean is an important pressure parameter that affects the hemodynamics of a patient. It has been shown that elevated Pmean leads to decreased cardiac output in the infant population during both normal frequency and high frequency mechanical ventilation [9,10]. Interventions to reduce Pmean during cardiopulmonary resuscitation (CPR) can reduce pleural pressure and improve hemodynamics [11]. Our previous study showed that Pmean affects CVP and then cardiac output (CO) through mechanical ventilation and cardiopulmonary interactions; at the same time, Pmean and CVP are proven risk factors for poor prognosis on mechanical ventilation [12,13]. In addition, CO determines the perfusion level, but measuring CO is often tedious. There is a positive correlation between perfusion index (PI) and left ventricular output [14]. At the same time, it is positively related to superior vena cava flow, which indirectly provides left ventricular flow changes or may lack volume capacity status in real time [15]. This opens the possibility for skipping complex CO measurements and using PI directly to indicate the perfusion level. These theories coincide with our finding that PI is a protective factor for mechanical ventilation [16]. Therefore, we propose a 3P strategy of circulation protection mechanical ventilation with flow priority [Fig. 1]. The strategy we proposed should reverse this process to open the lung, match the flow, and ensure perfusion. If someone meets these 3P criteria, the prognosis is always better. The Pmean, CVP, and PI cut-off values are based on our previous publication, which came from all patients who received mechanical ventilation in the ICU during a period of observational time [12,13]. The cut-off value needs further development and validation, especially in patients with severe ARDS with unstable hemodynamics. However, patients with high CVP and Pmean accompanied by low PI deserve clinical attention with respect to their hemodynamic status.

At present, the era of big data and intelligent intensive care units (ICUs) has arrived. Determining whether it is possible to use a simple real-time parameter for respiratory mechanics measurement and monitoring has become an urgent need. Using Pmean as an indicator of daily clinical management, our latest research showed that the 90-day mortality rate of patients who reached the Pmean target was lower than that of patients who did not, again illustrating the vital role of Pmean in the daily management of patients in the ICU [17]. Additionally, we proposed that three quality control targets ($SpO_2 \neq 100\%$, $PaCO_2 \geq 40$ mmHg, Pmean ≤ 10 cm H_2O) may be used for ICU treatment, which may be a highly operable method to achieve enhanced recovery after surgery (ERAS) in the ICU. Pmean measurement is conducive to the development of related studies, avoids tedious measurement of respiratory mechanics, and is not restricted by ventilation mode. It is of great significance for real-time monitoring and dynamic adjustment of respiratory mechanics to achieve intelligent ventilation.

In summary, we believe that Pmean has many advantages compared with other traditional pressure indicators and has the potential to become a bridge parameter connecting respiratory mechanics and hemodynamics in the future. In the process of clinical treatment, in cases of increased Pmean, strict low tidal volume lung protection, more suitable PEEP, more suitable volume state, and the lowest load of the right heart are factors to be considered. Intensive care clinicians and nurses are supposed to use this index widely in daily clinical work to validate our theory of "3P strategy of circulation protection mechanical ventilation with flow priority." More research and data are needed to verify and validate these findings in the future.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

[1] Kinnear W. Pulmonary physiology and pathophysiology. Thorax 2002;57:752. doi:10.1136/thorax.57.8.752-a.

[2] Pinsky MR. Cardiopulmonary interactions: physiologic basis and clinical applications. Ann Am Thorac Soc 2018;15:S45–8. doi:10.1513/AnnalsATS.201704-339FR.

[3] Vieillard-Baron A, Matthay M, Teboul JL, Bein T, Schultz M, Magder S, et al. 'Experts' opinion on management of hemodynamics in ARDS patients: focus on the effects of mechanical ventilation. Intensive Care Med 2016;42:739–49. doi:10.1007/s00134-016-4326-3.

[4] Fuhrman BP, Smith-Wright DL, Venkataraman S, Orr RA, Howland DF. Proximal mean airway pressure: a good estimator of mean alveolar pressure during continuous positive-pressure breathing. Crit Care Med 1989;17:666–70.

[5] Marini JJ, Ravenscraft SA. Mean airway pressure: physiologic determinants and clinical importance – Part 1: physiologic determinants and measurements. Crit Care Med 1992;20:1461–72. doi:10.1097/00003246-199210000-00017.

[6] Trachsel D, McCrindle BW, Nakagawa S, Bohn D. Oxygenation index predicts outcome in children with acute hypoxemic respiratory failure. Am J Respir Crit Care Med 2005;172:206–11. doi:10.1164/rccm.200405-625OC.

[7] Gattinoni L, Tonetti T, Cressoni M, Cadringher P, Herrmann P, Moerer O, et al. Ventilator-related causes of lung injury: the mechanical power. Intensive Care Med 2016;42:1567–75. doi:10.1007/s00134-016-4505-2.

[8] Sahetya SK, Wu TD, Morgan B, Herrera P, Roldan R, Paz E, et al. Mean airway pressure As a predictor of 90-day mortality in mechanically ventilated patients. Crit Care Med 2020;48:688–95. doi:10.1097/CCM.0000000000004268.

[9] Gullberg N, Winberg P, Selldén H. Changes in stroke volume cause change in cardiac output in neonates and infants when mean airway pressure is altered. Acta Anaesthesiol Scand 1999;43:999–1004. doi:10.1034/j.1399-6576.1999.431005.x.

[10] Gullberg N, Winberg P, Selldén H. Changes in mean airway pressure during HFOV influences cardiac output in neonates and infants. Acta Anaesthesiol Scand 2004;48:218–23. doi:10.1111/j.1399-6576.2004.00299.x.

[11] Kwon Y, Debaty G, Puertas L, Metzger A, Rees J, McKnite S, et al. Effect of regulating airway pressure on intrathoracic pressure and vital organ perfusion pressure during cardiopulmonary resuscitation: a non-randomized interventional cross-over study. Scand J Trauma Resusc Emerg Med 2015;23:83. doi:10.1186/s13049-015-0164-5.

[12] Long Y, Su L, Zhang Q, Zhou X, Wang H, Cui N, et al. Elevated mean airway pressure and central venous pressure in the first day of mechanical ventilation indicated poor outcome. Crit Care Med 2017;45:e485–92. doi:10.1097/CCM.0000000000002290.

[13] Long Y, Su L, Liu D. The authors reply. Crit Care Med 2017;45:e739–40. doi:10.1097/CCM.0000000000002472.

[14] Corsini I, Cecchi A, Coviello C, Dani C. Perfusion index and left ventricular output correlation in healthy term infants. Eur J Pediatr 2017;176:1013–18. doi:10.1007/s00431-017-2920-1.

[15] Takahashi S, Kakiuchi S, Nanba Y, Tsukamoto K, Nakamura T, Ito Y. The perfusion index derived from a pulse oximeter for predicting low superior vena cava flow in very low birth weight infants. J Perinatol 2010;30:265–9. doi:10.1038/jp.2009.159.

[16] Su L, Zhang R, Zhang Q, Xu Q, Zhou X, Cui N, et al. The effect of mechanical ventilation on peripheral perfusion index and its association with the prognosis of critically Ill patients. Crit Care Med 2019;47:685–90. doi:10.1097/CCM.0000000000003661.

[17] Pan P, Su L, Zhang Q, Long Y, Wang X, Liu D. Effects of quality control targets (SpO2≠100%, $PaCO_2$/<40 mmHg, Pmean/ > 10 cmH_2O) on outcomes in patients in the ICU. Front Med (Lausanne) 2020;7:111. doi:10.3389/fmed.2020.00111.