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EDITORIAL

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# Respiratory mechanics in ARDS: a siren for physicians?

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Sirs: It is now established in patients with acute respiratory distress syndrome (ARDS) that mechanical ventilation per se may affect mortality [1]. Selecting the most appropriate ventilatory support is as urgent and important as the search for the cause and determining the consequent pathogenetic treatment. In "buying" time for the resolution of the disease, the possible iatrogenic effects of mechanical ventilation need to be minimized as much as possible. The iatrogenic costs of mechanical ventilation are thought to be: (a) high inspired oxygen fraction, (b) high pressures ("barotrauma") and large tidal volumes ("volutrauma"), and (c) intratidal collapse and reinflation ("atelectrauma"). There is no evidence that high inspired oxygen fraction is actually dangerous in ARDS. On the other hand, high inspiratory plateau pressures can cause various pulmonary lesions [2]. However, what it is important is not the pressure per se but the delivered volume, which, if excessive, may lead to high transpulmonary pressure with consequent stretching of the pulmonary tissues [3]. Moreover, the importance has recently been stressed of the interaction between inspiratory and expiratory phase. Even at low plateau pressures (20–25 $cmH_2O$) most of the dependent lung regions open up, and if positive end-expiratory pressure (PEEP) is not sufficient, they collapse at end-expiration [4]. Intratidal collapse and reinflation generate shear forces which can increase capillary permeability and induce activation of inflammatory factors leading to local and systemic inflammatory processes ("biotrauma") [5].

Thus at the moment two strategies of ventilatory support are suggested in severe ARDS: (a) small tidal volumes (< 6 ml/kg), low plateau pressures (< 35 $cmH_2O$), respiratory rate to control $PaCO_2$ and pH within as normal a range as possible, with PEEP titrated to achieve an "acceptable" oxygenation ($PaO_2$ between 80 and 100 mmHg) [1, 6, 7, 8]; (b) low tidal volumes and plateau pressures, with PEEP sufficiently high to assure the lung open at end-expiration ("open lung approach") [9]. In this case PEEP is titrated just above the inflection point of the inspiratory limb of the pressure-volume (PV) curve of the respiratory system [10, 11]. However, the use of high inspiratory oxygen fraction together with tidal volume so small as to lead to severe hypercapnia (i.e., low VA/Q ratios), even in presence of relatively high PEEP levels may facilitate the occurrence of reabsorption atelectasis. This can cause a deterioration in oxygenation and respiratory mechanics and increase "shear stress" trauma [12].

These considerations suggest that the following analysis of the mechanical properties of the respiratory system (compliance and resistance) may be useful in ARDS:

- To characterize the underlying pathology and determine the amount of lung parenchyma available for ventilation together with the potential for recruitment during the course of the disease [13]
- To set the mechanical ventilation within the "safe" zone of the PV curve region: (a) to avoid large tidal volumes with excessive overdistension (upper inflection point) and excessively low tidal volumes, (b) to

select PEEP necessary to adequately maintain open the lung at end-expiration (lower inflection point)
– To select appropriate respiratory rate and duty cycle times

Among the methods proposed for characterizing the pathology and selecting PEEP and tidal volume [14, 15], the PV curve of the respiratory system is gaining increasing popularity [16]. However, the application of respiratory mechanics measurements in clinical practice has been limited by the fact that these measurements are usually time consuming and cumbersome. On-line monitoring of respiratory mechanics makes it easier and more feasible for clinicians to perform these measurements [17]. The ease of measurement is in fact inseparable from its precision and accuracy. Nunes and Takala, in this issue of *Intensive Care Medicine*, show that measurements of respiratory mechanics and the PV curve may be easily obtained in a clinical setting using a dedicated device.

It has been suggested that the initial slope of the PV curve (starting compliance) estimates the amount of aerated tissue at atmospheric pressure. The lower inflection point (or more precisely the lower inflection zone) indicates the pressures around which most of the recruitment may occur. The slope of the PV curve after the lower inflection point (inflation compliance) related to starting compliance may give some estimate of the amount of recruitment (the higher the ratio, the greater the recruitment) [13]. The upper inflection point indicates the pressure at which the stretching of the pulmonary units becomes the prevalent phenomenon. Moreover, the PV curve may allow inferences on the underlying pathology: the presence of an inflection point suggests compression atelectasis, while its absence suggests consolidation [18]. Unfortunately, the PV curve is only a very simplistic mirror of the reality and has many pitfalls [19].

First, the lack of a lower inflection point does not necessarily indicate lack of recruitment. If recruitment is proportional to the applied pressure, a linear PV curve results. Second, the presence of an inflection point may indicate the beginning of alveolar recruitment and not the region around which most of recruitment occurs [20]. Third, the PV curve of the respiratory system integrates the PV curves of the lung and chest wall. If the mechanical properties of the chest wall are altered, the PV curve of the respiratory system cannot be representative of lung behavior. The inflection points (lower and upper) of the respiratory system may be due to the inflection points of the chest wall and not of the lung, leading to erroneous interpretations of the PV curve of the respiratory system [21, 22]. It has been shown that mechanical alterations in the chest wall, due to an increased intra-abdominal pressure, are common in patients with ARDS stemming from abdominal disease [23]. Indeed, the meaningful assessment of mechanics should include the measurement of esophageal pressure to partition the respiratory system mechanics and understand the lung and chest wall interactions. Fourth, respiratory mechanics investigates the lung as whole, while in reality the same lung presents regional mechanical differences, and one lung may differ from the other both in underlying pathology (prevalent consolidation vs. atelectasis) and in mechanical characteristics. Fifth, the mechanical measurements, as usually performed, assume that the only variations in the respiratory system are due to changes in volumes and pressures, while intrathoracic blood volume is considered unchanged. However, when high intrathoracic pressures are reached during the PV curve, part of the blood is squeezed out from the thorax. Optoelectronic plethysmography suggests that part of the changes in lung volume usually interpreted as recruitment or derecruitment are due to pressure-dependent blood shift [24]. Sixth, clinical relevant data of the PV curve are usually derived from the inspiratory limb. While this procedure may be appropriate to studying the process of recruitment, it is important to realize that derecruitment follows a different pattern and is related to the expiratory limb of the PV curve. This may be extremely relevant for PEEP selection, as PEEP is an expiratory and not an inspiratory phenomenon. Unfortunately, the expiratory limb is usually ignored, while major efforts are currently focused only on the inspiratory phase.

Another important issue regarding respiratory mechanics is the measurement of resistances. Inspiratory resistance is the result of the sum of airway resistances and "additional" resistances due to viscoelastic properties and gas distribution inhomogeneities within the respiratory system. In ARDS both airway "additional" and expiratory resistances are increased [25]. Respiratory resistance may be increased as the result of reduced lung volume, reduced caliber (due to local edema and/or increased smooth muscle tone), or both. Thus it is important to identify the pathophysiological causes of increased airway resistance and to select the appropriate respiratory rate and an effective bronchodilator treatment [26]. However, the automatic measurement of resistance as proposed by Nunes and Takala presents some limitations. First, the computation of total resistances does not distinguish between airway and "additional" resistance. Second, the resistance includes that of the tube, which can change markedly with time, giving inappropriate measurement of the actual airway resistance. Third, the inspiratory and expiratory resistances are computed as averages. This measurement may be misleading since expiratory resistance is generally higher than inspiratory resistance. Fourth, the difficulty in relating airway resistance to absolute lung volume makes this measurement extremely poor from a clinical point of view.

Thus, for all these reasons, measurements of respiratory mechanics and PV curve cannot be easily used as a surrogate measure of the degree of alveolar inflation and recruitment. Consequently, they need to be considered with extreme caution when used to select the ventilatory setting.

In conclusion, recent efforts to develop and test new devices for providing easy, accurate, and precise respiratory mechanics data at the bedside must be encouraged. However, we need more information of the physiological meaning of information derived from respiratory mechanics and it possible limitations. The incorrect use of respiratory mechanics information could produce more negative than positive effects on treatment.

The sirens are alluring and sexy, but they can be dangerous if listened to without precautions!

Sirens sing a nice song, attracting the traveler: but near by a heap of human putrefied bones and putrid bodies lie. Go beyond fast and stop your ears, so that you don't listen their voice." (Homer, *Odyssey*, Chap. XII, 59–66)

## References


1. The Acute Respiratory Distress Syndrome Network (2000) Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung, injury and the acute respiratory distress syndrome. N Engl J Med 342: 1301–1308
2. Kolobow T, Moretti MP, Fumagalli R, Mascheroni D, Prato P, Chen V, Joris M (1987) Severe impairment in lung function induced by high peak airway pressure during mechanical ventilation: an experimental study. Am Rev Respir Dis 135: 312–315
3. Dreyfuss D, Soler P, Basset G, Saumon G (1988) High inflation pressure pulmonary edema. Respective effects of high airway pressure, high tidal volume and positive end-expiratory pressure. Am Rev Respir Dis 137: 1159–1164
4. Gattinoni L, Pelosi P, Crotti S, Valenza F (1995) Effects of positive end-expiratory pressure on regional distribution of tidal volume and recruitment in adult respiratory distress syndrome. Am J Respir Crit Care Med 151: 1807–1814
5. Slutsky AS, Tremblay LN (1998) Multiple system organ failure: is mechanical ventilation a contributing factor? Am J Respir Crit Care Med 157: 1721–1725
6. Stewart TE, Meade MO, Cook DJ, Granton JT, Hodder RV, Lapinsky SE, Mazer CD, McLean RF, Rogovein TS, Shouten BD, Todd TRJ, Slutsky AS and the Pressure-and Volume Limited Ventilation Strategy Group (1998) Evaluation of a ventilation strategy to prevent barotrauma in patients at high risk for acute respiratory distress syndrome. N Engl J Med 338: 355–361
7. Brochard L, Roudot-Thoraval F, Roupie E, Delclaux C, Chastre J, Fernandez-Mondejar E, Clementi E, Mancebo J, Factor P, Matamis D, Ranieri VM, Blanch L, Rodi G, Mentec H, Dreyfuss D, Ferrer M, Brun-Buisson C, Tobin M, Lemaire F, and the Multicenter Trial Group on Tidal Volume Reduction in ARDS (1998) Tidal volume reduction for prevention of ventilator-induced lung injury in acute respiratory distress syndrome. Am J Respir Crit Care Med 158: 1831–1838
8. Brower RG, Shanholtz CB, Fessler HE, Shade DM, White P Jr, Wiener CM, Teeter JG, Dodd JM, Almong Y, Piantadosi S (1999) Prospective, randomized, controlled trial comparing traditional versus reduced tidal volume ventilation in acute respiratory distress syndrome patients. Crit Care Med 27: 1492–1498
9. Lachmann B (1992) Open up the lung and keep the lung open. Intensive Care Med 18: 319–321
10. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, De Paula Pinto Schettino G, Lorenzi-Filho G, Kairalla R, Deheinzelin D, Munoz C, Oliveira R, Takagaki TY, Carvalho CRR (1998) Effect of a protective ventilation strategy on mortality in the acute respiratory distress syndrome. N Engl J Med 338: 347–354
11. Ranieri VM, Suter PM, Tortorella C, De Tullio R, Dayer JM, Brienza A, Bruno F, Slutsky AS (1999) Effect of mechanical ventilation on inflammatory mediators in patients with acute respiratory distress syndrome: a randomized controlled trial. JAMA 282: 54–61
12. Pelosi P, Cadringher P, Bottino N, Panigada M, Carrieri F, Riva E, Lissoni A, Gattinoni L (1999) Sigh in acute respiratory distress syndrome. Am J Respir Crit Care Med 159: 872–880
13. Gattinoni L, Pesenti A, Avalli L, Rossi F, Bombino M (1987) Pressure-volume curve of total respiratory system in acute respiratory distress failure: computed tomographic scan study. Am Rev Respir Dis 136: 730–736
14. Suter PM, Fairley HB, Isenberg MD (1975) Optimum end-expiratory airway pressure in patients with ARF. N Engl J Med 292: 284–289
15. Harf A, Lemaire F, Lorino H, Atlan G (1975) Etude de mecanique ventilatoire: application a la ventilation artificielle. Bull Physiol Pathol Resp 11: 709–729
16. Jonson B, Svantesson C (1999) Elastic pressure volume curves: what information do they convey? Thorax 54: 82–87
17. Servillo G, De Robertis E, Coppola M, Blasi F, Rossano F, Tufano R (2000) Application of a computerized methods to measure static pressure volume curve in acute respiratory distress syndrome. Intensive Care Med 26: 11–14
18. Vieira SR, Puybasset L, Lu Q, Richecoreur J, Cluzel P, Coriat P, Rouby JJ (1999) A scanographic assessment of pulmonary morphology in acute lung injury. Significance of the lower inflection point detected on the lung pressure-volume curve. Am J Respir Crit Care Med 159: 1612–1623
19. Gattinoni L, Pelosi P, Brazzi L, Valenza F (1999) Acute respiratory distress syndrome. In: Albert RK, Spiro SG, Jelt JR (eds) Comprehensive respiratory medicine. Mosby, London, pp 69.1–69.16
20. Hickling KG (1998) The pressure-volume curve is greatly modified by recruitment. A mathematical model of ARDS lungs. Am J Respir Crit Care Med 158: 194–202

21. Ranieri VM, Brienza N, Santostasi S, Puntillo F, Mascia L, Vitale N, Giuliana R, Memeo V, Bruno F, Fiore T, Brienza A, Slutsky AS (1997) Impairment of lung and chest wall mechanics in patients with acute respiratory distress syndrome: role of abdominal distension. Am J Respir Crit Care Med 156: 1082–1091
22. Mergoni M, Martelli A, Volpi A, Primavera S, Zuccoli P, Rossi A (1997) Impact of positive end-expiratory pressure on chest wall and lung pressure volume curve in acute respiratory failure. Am J Respir Crit Care Med 156: 846–854
23. Gattinoni L, Pelosi P, Suter PM, Pedoto A, Vercesi P, Lissoni A (1998) Acute respiratory distress syndrome caused by pulmonary and extrapulmonary disease. Different syndromes? Am J Respir Crit Care Med 158: 3–11
24. Aliverti A, Dellaca R, Pelosi P, Chiumello D, Pedotti A, Gattinoni L (2000) Optoelectronic plethysmography in intensive care patients. Am J Respir Crit Care Med (in press)
25. Pelosi P, Cereda M, Foti G, Giacomini M, Pesenti A (1995) Alterations of lung and chest wall mechanics in patients with acute lung injury; effect of positive end-expiratory pressure. Am J Respir Crit Care Med 152: 531–537
26. Pesenti A, Pelosi P, Rossi N, Aprigliano M, Brazzi L, Fumagalli R (1993) Respiratory mechanics and bronchodilator responsiveness in patients with the adult respiratory distress syndrome. Crit Care Med 21: 78–83