Intensive Care Med (1992) 18:383–385

Intensive Care Medicine

Editorials

PEEP, ARDS, and alveolar recruitment

J. Mancebo

Servei de Medicina Intensiva, Hospital de la Santa Creu i Sant Pau, Barcelona, Universitat Autònoma de Barcelona, Spain

Adult respiratory distress syndrome (ARDS) is a particular form of permeability pulmonary edema which, from a pathophysiological point of view, is characterized by refractory hypoxemia, principally due to intrapulmonary right to left shunting of blood and a low functional residual capacity (FRC). Another key feature is the low thoraco-pulmonary compliance [1, 2]. It has been said that compliance is low because lung volume is also low, which has led to the concept that ARDS lungs are similar to "baby lungs" with a normal specific compliance [3]. Additionally, ARDS is characterized not only by a low compliance, but also by high airway and tissue resistance [4–6], although this is beyond the scope of this paper.

Since its original description by Ashbaugh et al. [7] in the late 60s, when the authors suggested that positive end-expiratory pressure (PEEP) may be useful in improving arterial oxygenation of these patients, PEEP has been widely used in ARDS as standard treatment to reverse severe arterial hypoxemia. Much of the work on PEEP, lung volume and gas exchange in patients with ARDS has been done by European investigators. We have since learnt a lot about PEEP and ARDS, but further intriguing questions have arisen.

Shortly after the first report on ARDS, two landmark papers by Falke et al. [8] and Suter et al. [9] were published on the effects of PEEP in severe acute respiratory failure and methods to titrate the PEEP level in these patients. The most important findings were that improvement of oxygenation by PEEP is basically due to the increase in FRC and redistribution of intrapulmonary blood flow, and also that the effects of PEEP on lung volumes and gas exchange depend principally on pulmonary mechanics.

With regard to pulmonary mechanics, patients with ARDS present with different patterns of compliance depending on their evolution [10, 11]. According to this concept, in the initial phases of ARDS lungs are edematous and wet and their mechanical characteristic is a somewhat reduced compliance with an inflection point in the thoraco-pulmonary pressure-volume (PV) curve, which is in fact the initial zone of the PV curve rather than a definite pressure. In this phase the specific compliance is still normal [12]. In late stages of ARDS fibrosis develops, the lungs become stiff, there is a very low thoraco-pulmonary compliance without an inflection point in the PV curve and specific compliance is probably low. However, our data indicate that in late stages of ARDS, the specific compliance is low, that is to say, compliance is lower than expected in relation to the reduced FRC [13]. Additionally, the data reported by Matamis and colleagues [11] show that, during ARDS evolution, while compliance is decreasing, FRC does not change further giving as a result a specific compliance significantly lower in the late phase than in the early phase of ARDS.

There is considerable clinical evidence that the inflection point, or better the inflection zone, seen in the thoraco-pulmonary PV curves, represents the recruitment of collapsed alveoli, and when this inflection point is used as PEEP there is a dramatic improvement in oxygenation [10–12, 14–16]. In fact, this is the approach that we are still using to titrate PEEP levels in "early" ARDS patients. When evaluating the effects of PEEP on alveolar recruitment it is important to keep in mind that changes in arterial oxygenation should be viewed together with changes in cardiac output, which may or may not be depressed by PEEP, and therefore may influence intrapulmonary shunt and arterial partial pressure of oxygen. Additionally, according to the PV curves of the respiratory system, when PEEP recruits alveoli there is an increase both in FRC and in compliance [17].

However, when there is no inflection and lung complicance is low, as occurs in the late phases, it is not sufficiently clear whether the use of PEEP is beneficial: while some clinical data suggest that PEEP still helps to improve arterial oxygenation in these cases [14, 18], other data do not [15]. Furthermore, in this situation, PEEP may cause alveolar overdistension. We all know that patients in late phases of ARDS are not easy to ventilate, and an important question at this point is that related with ventilator-induced lung injury, namely barotrauma and pulmonary edema. Barotrauma results from the rupture of overdistended alveoli, and several risk factors may contribute to its development such as high tidal volume (V_T), high inspiratory pressures or PEEP [19, 20]. Pulmonary edema and alveolar cell injury have been documented in several animal experimental models when ventilating with high peak airway pressures, about 45 cmH_2O [21, 22]. Additionally, what seems to be more dangerous than the pressure itself, is the overexpansion caused by high V_T: it has been demonstrated that it is the effect of volume more than the effect of pressure that causes permeability pulmonary edema and diffuse alveolar damage [23–25]. Interestingly, PEEP appears to decrease the amount of edema induced by high volume-high pressure mechanical ventilation [23]. Another important aspect is that not only high V_T are dangerous but that low V_T are as well: in ARDS patients the lowest compliance is found at high and low V_T, whereas it improves at intermediate V_T [6]; on the other hand, ventilation of isolated non-perfused injured lungs below the inflection zone of the PV curves at low airway pressure and low V_T, produces severe alveolar lesions in animal models [26]. Finally, the clinical data reported by Hickling and colleagues [27] suggest a significantly reduced mortality in ARDS patients ventilated with a low peak airway pressures (usually below 30 cmH_2O) by reducing V_T and allowing hypercapnia.

In recent years there has been a considerable increase in the use of microcomputers to study the mechanical behaviour of the respiratory system in clinical practice. This has also been the case for the modern artificial ventilators. These technologic advances have permitted accurate study of the mechanical characteristics of the respiratory system [28, 29]. The PV curve performed with the super-syringe will probably be replaced by the computerized systems built into the ventilators, which in turn allow non-invasive determination of the physiologic variables of interest without disconnecting patients from ventilators, and probably avoiding paralysis.

A stimulating paper on this topic was recently published by Ranieri and colleagues [30] who studied eight ARDS patients and showed that a tidal volume of 10 ml/kg of body weight and a PEEP of 15 cmH_2O was probably responsible for barotrauma, since the mean end inspiratory plateau pressure reached 42 cmH_2O. These data of Ranieri and colleagues confirm the importance of taking lung mechanics into account when dealing with PEEP in ARDS lungs. In the context of a heterogeneous and continuously evolving disease such as ARDS, ventilatory management should be adjusted to the mechanical status of the lung. From the dâta presently available, this may be accomplished in the usual clinical setting if various points are taken into account: firstly, by using moderate PEEP levels (8–12 cmH_2O), secondly V_T should be lower than that previously used (5–10 ml/kg) in order to maintain tidal ventilation below the flat upper part of the PV curve [31] and also to allow hypercapnia if it ensues, thirdly it may be useful to maintain a high airflow rate in order to minimize lung tissue resistance, and finally the end-inspiratory plateau pressure should be monitored by keeping its maximum level at 35 cmH_2O. Accordingly, what seems very important nowadays and probably should be a direction for the future is to ventilate safely, that is to say, to follow the old premise of primum non nocere.

References

1. Murray JF, Matthay MA, Luce JM, Flick MR (1988) An expanded definition of the adult respiratory distress syndrome. Am Rev Respir Dis 138:720–723
2. Petty TL (1988) ARDS: refinement of concept and redefinition. Am Rev Respir Dis 138:724
3. Gattinoni L, Pesenti A (1991) Computed tomography scanning in acute respiratory failure. In: Zapol WM, Lemaire F (eds) Adult respiratory distress syndrome. Dekker, New York, pp 199–221
4. Broseghini C, Brandolese R, Poggi R et al (1988) Respiratory mechanics during the first day of mechanical ventilation in patients with pulmonary edema and chronic airway obstruction. Am Rev Respir Dis 138:355–361
5. Wright PE, Bernard GR (1989) The role of airflow resistance in patients with the adult respiratory distress syndrome. Am Rev Respir Dis 139:1169–1174
6. Eissa NT, Ranieri VM, Corbeil C et al (1991) Analysis of behavior of the respiratory system in ARDS patients: effects of flow, volume, and time. J Appl Physiol 70:2719–2729
7. Ashbaugh DG, Bigelow DB, Petty TL, Levine BE (1967) Acute respiratory distress in adults. Lancet II:319–323
8. Falke KJ, Pontoppidan H, Kumar A, Leith DE, Geffin B, Laver MB (1972) Ventilation with end-expiratory pressure in acute lung disease. J Clin Invest 51:2315–2323
9. Suter PM, Fairley HB, Isenberg MD (1975) Optimum end-expiratory pressure in patients with acute pulmonary failure. N Engl J Med 292:284–289
10. Lemaire F, Harf A, Simonneau G, Matamis D, Rivara D, Atlan G (1981) Echanges gazeux, courbe statique pression-volume et ventilation en pression positive de fin d'expiration. Etude dans seize cas d'insuffisance respiratoire aiguë de l'adulte. Ann Anesth Fr 5:435–441
11. Matamis D, Lemaire F, Harf A, Brun-Buisson C, Ansquer JC, Atlan G (1984) Total respiratory pressure-volume curves in the adult respiratory distress syndrome. Chest 86:58–66
12. Gattinoni L, Pesenti A, Avalli L, Rossi F, Bombino M (1987) Pressure-volume curve of total respiratory system in acute respiratory failure. Computed tomographic scan study. Am Rev Respir Dis 136:730–736
13. Mancebo J, Benito S (1992) Mécanique respiratoire dans les oedèmes pulmonaires. In: Lemaire F, Zelter M (eds) Oedèmes pulmonaires. Masson, Paris, pp 133–143
14. Blanch L, Fernández R, Benito S, Mancebo J, Net A (1987) Effect of PEEP on the arterial minus end-tidal carbon dioxide gradient. Chest 92:451–454
15. Holzapfel L, Robert D, Perrin F, Blanc PL, Palmier B, Guerin C (1983) Static pressure-volume curves and effect of positive end-expiratory pressure on gas exchange in adult respiratory distress syndrome. Crit Care Med 11:591–597
16. Gattinoni L, Pesenti A, Bombino M et al (1988) Relationships between lung computed tomographic density, gas exchange, and PEEP in acute respiratory failure. Anesthesiology 69:824–832
17. Benito S, Lemaire F (1990) Pulmonary pressure-volume relationship in acute respiratory distress syndrome in adults: role of positive end expiratory pressure. J Crit Care 5:27–34
18. Falke KJ (1980) Do changes in lung compliance allow the determination of "optimal PEEP"? Anaesthesist 29:165–168
19. Haake R, Schlichtig R, Ulstad DR, Henschen RR (1987) Barotrauma. Pathophysiology, risk factors, and prevention. Chest 91:608–613
20. Pingleton SK (1988) Complications of acute respiratory failure. Am Rev Respir Dis 137:1463–1493
21. Dreyfuss D, Basset G, Soler P, Saumon G (1985) Intermittent positive-pressure hyperventilation with high inflation pressures pro-

duces pulmonary microvascular injury in rats. Am Rev Respir Dis 132:880–884
22. Kolobow T, Moretti MP, Fumigalli R et al (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
23. 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
24. Corbridge TC, Wood LDH, Crawdord GR, Chudoba MJ, Yanos J, Sznajder I (1990) Adverse effects of large tidal volume and low PEEP in canine acid aspiration. Am Rev Respir Dis 142:311–315
25. Carlton DP, Cummings JJ, Scheerer RG, Poulain FR, Bland RD (1990) Lung overexpansion increases pulmonary microvascular protein permeability in young lambs. J Appl Physiol 69:577–583
26. Muscedere JG, Muller JBM, Gan K, Bryan AC, Slutsky AS (1992) Tidal ventilaton at low airway pressures can cause pulmonary barotrauma. Am Rev Respir Dis 145 (Part 2):A454
27. Hickling KG, Henderson SJ, Jackson R (1990) Low mortality associated with low volume pressure limited ventilation with permissive hypercapnia in severe adult respiratory distress syndrome. Intensive Care Med 16:372–377
28. Levy P, Similowsky T, Corbeil C et al (1989) A method for studying the static volume-pressure curves of the respiratory system during mechanical ventilation. J Crit Care 4:83–89
29. Sydow M, Burchardi H, Zinserling J, Ische H, Crozier ThA, Weyland W (1991) Improved determination of static compliance by automated single volume steps in ventilated patients. Intensive Care Med 17:108–114
30. Ranieri VM, Eissa NT, Corbeil C et al (1991) Effects of positive end-expiratory pressure on alveolar recruitment and gas exchange in patients with the adult respiratory distress syndrome. Am Rev Respir Dis 144:544–551
31. Dall'Ava-Santucci J, Armaganidis A, Brunet F et al (1990) Mechanical effects of PEEP in patients with adult respiratory distress syndrome. J Appl Physiol 68:843–848

Dr. J. Mancebo
Servei de Medicina Intensiva
Hospital de la Santa Creu i Sant Pau
Universitat Autònoma de Barcelona
Sant Antoni Ma. Claret 167
E-08025 Barcelona
Spain