Case Reports

A Case of Pneumomediastinum in a Patient With Acute Respiratory Distress Syndrome on Pressure Support Ventilation

Véronique Leray MD, Gael Bourdin MD, Ghislain Flandreau, Frédérique Bayle MD, Florent Wallet MD, Jean-Christophe Richard MD PhD, and Claude Guérin MD PhD

During mechanical ventilation for acute respiratory distress syndrome, tidal volume (V_T) must be reduced. Once switched to pressure-support ventilation, there is a risk that uncontrolled large V_T may be delivered. A 63-year-old man with community-acquired pneumonia required tracheal intubation and mechanical ventilation, with a V_T of 6 mL/kg predicted body weight, PEEP of 10 cm H_2O, a respiratory rate of 30 breaths/min, and F_{IO_2} of 0.60. Plateau pressure was 22 cm H_2O. He improved and received pressure-support. Twelve days later a chest radiograph showed suspected air leaks, confirmed via computed tomogram (CT), which showed anterior pneumomediastinum. V_T received over the previous 3 days had averaged 14 mL/kg predicted body weight. The patient was put back onto volume-controlled mode, and 2 days later there were no air leaks. In pressure-support ventilation, V_T must be closely monitored to ensure lung-protective mechanical ventilation. *Key words: acute respiratory distress syndrome; ARDS; ventilator-induced lung injury; mechanical ventilation; volutrauma.* [Respir Care 2010;55(6):770–773. © 2010 Daedalus Enterprises]

Introduction

The use of low tidal volume (V_T) and pressure limitation is highly recommended during mechanical ventilation for acute respiratory distress syndrome (ARDS), to prevent ventilator-induced lung injury and reduce mortality.[1] During pressure-support ventilation, V_T depends on the pressure, patient effort, and the mechanical properties of the respiratory system. It is therefore possible that a patient goes from a carefully controlled low-V_T strategy to uncontrolled V_T delivery.

Case Report

A 63-year-old man admitted to our intensive care unit on hour zero of day zero for community-acquired pneu-

Véronique Leray MD, Gael Bourdin MD, Ghislain Flandreau, Frédérique Bayle MD, Florent Wallet MD, Jean-Christophe Richard MD PhD, and Claude Guérin MD PhD are affiliated with Service de Réanimation Médicale, Hôpital de la Croix-Rousse, and University of Lyon, Lyon, France.

Correspondence: Claude Guérin MD PhD, Service de Réanimation Médicale, Hôpital de la Croix-Rousse, 103 Grande Rue de la Croix Rousse, 69004 Lyon, France. E-mail: claude.guerin@chu-lyon.fr.

monia with severe hypoxemia was intubated at hour 19 and ventilated with volume-controlled mode, with V_T 6 mL/kg predicted body weight, respiratory rate 30 breaths/min, inspiratory time 0.4 s, F_{IO_2} 0.60, and PEEP 10 cm H_2O. Continuous intravenous sedation (midazolam 4 mg/h), analgesia (morphine 3 mg/h), and neuromuscular blockade (cisatracurium 30 mg/h) were employed. With the above settings, total PEEP was 10 cm H_2O, end-inspiratory plateau pressure (P_{plat}) was 22 cm H_2O, P_{aO_2} was 65 mm Hg, P_{aCO_2} was 44 mm Hg, and pH was 7.42. The diagnosis of ARDS was made based on: (1) the acute onset of respira-

tory failure, (2) $P_{aO_2}/F_{IO_2} < 200$ mm Hg, (3) the presence of bilateral lung infiltrates on the frontal chest radiograph, and (4) the lack of evidence of elevated left atrial pressure on transthoracic echocardiography. At hour 32 he underwent a 16-hour prone-position session, which increased P_{aO_2}/F_{IO_2} from 108 mm Hg to 162 mm Hg at the end of the session, with corresponding P_{plat} of 23 cm H_2O and 22 cm H_2O. He was proned a second time for 16 hours the following day. As improved oxygenation was maintained in the supine position, the proning was interrupted and the sedation, analgesia, and neuromuscular blockade
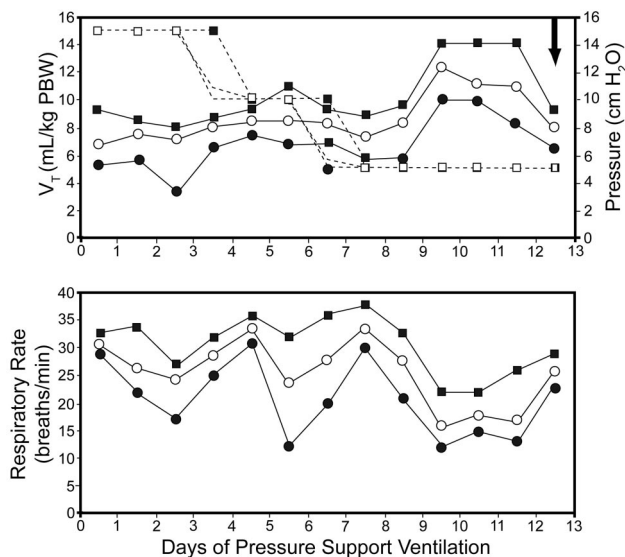
Fig. 1. Daily course of maximal (black squares), minimal (black circles), and mean (open circles) values of expired tidal volume (V_T) (continuous lines) and mean (open squares) level of pressure support (dashed lines) (upper panel) and of respiratory rate (lower panel). Observe the high V_T delivered in the few days before the discovery of air leaks. The final drop in V_T (arrow) might have resulted from reduction of lung and chest wall compliance during the pneumomediastinum.

were stopped on day 4. Over those 4 days the static compliance of the respiratory system, measured daily in the supine position, went from 37.5 mL/cm H_2O to 31.0 mL/cm H_2O.

Pressure-support ventilation was started on day 7, at 20 cm H_2O, and gradually lowered, by 5 cm H_2O at a time, as long as the respiratory rate was between 26 and 35 breaths/min and transcutaneous pulse oximetry was > 88%. PEEP was kept constant at 5 cm H_2O. As our weaning protocol states that volume-controlled mode must be resumed when 20 cm H_2O pressure support fails (ie, when the respiratory rate is > 35 breaths/min and/or pulse oximetry is < 88%, among other criteria), the patient was put back on volume-controlled ventilation, with the same settings as previously used. On day 11 the patient was put back onto pressure support for a further 12 consecutive days. The Glasgow coma score was 13–15, but a severe critical illness neuromyopathy with inability to cough delayed extubation. V_T increased progressively, despite a progressive reduction in the level of pressure support (Fig. 1). On day 20 a gastroscopy showed gastric ulcus, which was treated endoscopically. On day 23 systolic blood pressure dropped to 72 mm Hg. A chest radiograph showed suspected air leaks, a finding that was not present prior to that time. A CT showed anterior pneumomediastinum (Fig. 2). No further evidence of esophageal tear was obtained from either the CT or a second gastroscopy. The thorax was not drained. The patient returned to volume-controlled mode, on the previous ventilation settings. Blood pressure was maintained with inotropic support. Blood taken at the time of the hypotension contained *Candida albicans*, which was treated with fluconazole. Two days later there were no air leaks visible on the CT. Multiple organ failure occurred, which led to the patient's death 44 days after admission. No autopsy was carried out.

Discussion

We attributed the pneumomediastinum and air leaks to volutrauma induced by mechanical ventilation, in the absence of any other clear explanation. The role of gastroscopy could be discussed[2] but the delay after the procedure, the rapid reversal of the air leaks after lowering V_T, and the lack of direct evidence for esophageal tear weigh against that hypothesis.

In ARDS patients receiving invasive mechanical ventilation, the occurrence of barotrauma (ie, macroscopic air leaks, including pneumothorax) is consistently reported at around 10%.[1,3-5] Interestingly, the incidence of barotrauma was not significantly different between patients receiving high or low V_T,[1] or high or low PEEP at fixed V_T in large prospective randomized controlled trials.[4,6,7] Retrospective analysis of the determinants of barotrauma during mechanical ventilation in ARDS patients found slightly different associations with airway pressure. Barotrauma was found to be associated with increasing PEEP in one study,[8] and with P_{plat} > 35 cm H_2O in another.[9]

In the spontaneously breathing patient receiving mechanical ventilation, the total applied pressure to the respiratory system at any time is the sum of the pressure generated by the respiratory muscles (P_{mus}) and the pressure generated by the ventilator (P_{aw}). The equation of motion of the respiratory system predicts that during inspiration P_{mus} and P_{aw} dissipate against the resistance to gas flow and the elastic recoil of the respiratory system above the end-expiratory elastic equilibrium position:

$$P_{mus} + P_{aw} = \text{total PEEP} + (R \times \dot{V}) + (E \times V_T) \quad (1)$$

where R is the resistance of the airways, $\dot{V}$ is the air flow rate, and E is the elastance of the respiratory system (1/static compliance). Let's look at what happens when the left side of Equation 1 changes. When the patient pulls harder, P_{mus} increases. If we assume that P_{mus} is high enough to open the inspiratory valve of the respirator and is prolonged during the mechanical insufflation time, the impact on the total applied pressure depends on which mode of mechanical ventilation the patient is receiving. In volume-controlled mode, P_{aw} should decrease and the total applied pressure should remain constant, with no change in delivered V_T. In volume-controlled mode, V_T is the
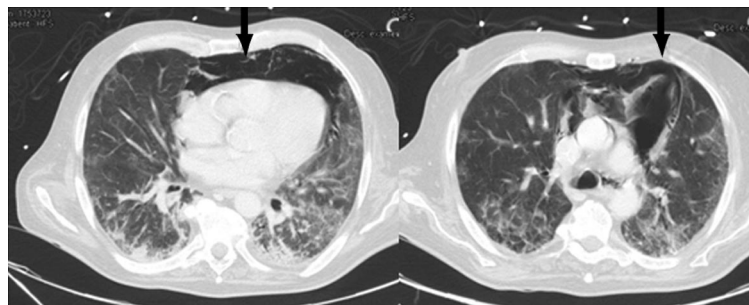
Fig. 2. Computed tomogram shows anterior pneumomediastinum (arrows).

independent variable and is unaffected by the patient's inspiratory effort. In contrast, in pressure-support ventilation, P_{aw} should not decrease in response to patient effort, because P_{aw} is the independent variable. As a result of the combination of P_{mus} and P_{aw}, transpulmonary pressure (P_L) increases. As predicted by the right side of Equation 1, the patient receives increased $\dot{V}$ and V_T. The distribution of this high V_T throughout the lungs depends on their regional pressure-volume relationships. In the heterogeneous ARDS lungs the stress on alveoli varies in different parts of the lung. Some parts of the lungs, presumably those that are more compliant and the most anterior, will receive the highest V_T and will operate on the upper part of their pressure-volume relationship, increasing the risk of hyperinflation/over-distention and barotrauma. It should be noted that these considerations also apply to other pressure-controlled modes, including airway-pressure-release ventilation, bi-level positive airway pressure ventilation, and pressure-controlled ventilation, in which V_T is poorly controlled by the physician or respiratory therapist.

The key points that must be emphasized are that: (1) P_L and thus V_T may reach excessive levels despite very low airway pressure, (2) P_{mus} is not measured, and therefore the airway pressure recorded by the ventilator can be misleading when considering the lung injury, (3) high V_T can be delivered for several hours per day because neither medical nor nursing staff pay close attention to V_T in pressure-support ventilation, and (4) it is often thought that lung protection is achieved by setting a pressure level below 30 cm H_2O, but this is not the case. It is important to emphasize here that P_{plat} could have been very high if the same V_T (14 mL/kg) was given to the patient in volume-controlled mode. It comes from the above considerations that the monitoring of P_L should be important in the management of ARDS patients receiving mechanical ventilation. Determining P_L requires P_{aw} and pleural pressure measurements. Pleural pressure can be obtained at the bedside by the measurement of either the pressure into the pleural space via a catheter or a drain in place, the esophageal pressure after insertion of an esophageal balloon,[10] the central venous pressure via a catheter inserted into the superior vena cava. In routine clinical practice, the mea-

surement of esophageal pressure is the method of choice to estimate pleural pressure, even though it has clear constraints.[11] Some intensive-care ventilators offer an input for external pressure, so bedside monitoring of P_L should be easy. Note that a recent randomized controlled trial showed that guiding mechanical ventilation with esophageal-pressure measurements may benefit patients.[12]

The issue of spontaneous breathing in ARDS is a widely debated topic. On the one hand, spontaneous breathing during airway-pressure-release ventilation in experimental ARDS was associated with reduced intrapulmonary shunt, which was explained both by the increased ventilation of aerated dependent lung tissue and by opening up non-aerated tissue so that ventilation is distributed to more of the lung.[13] Similar findings were also obtained in humans with ARDS.[14] A small randomized controlled trial with 30 patients, including only 5 ARDS patients, showed that airway-pressure-release ventilation required less sedation and improved cardiopulmonary function, as compared to conventional volume-controlled ventilation.[15] However, an experimental study with normal sheep lungs found that prolonged hyperventilation resulted in acute lung injury.[16] Finally, several reports have strongly suggested that large V_T delivered by mechanical ventilation to normal lungs may predict the development of ARDS in humans.[17-20] On balance, it is helpful to have some diaphragm function to help recruit dependent, atelectatic lung, but it is also important to limit P_L when breathing spontaneously. Large multicenter randomized controlled trials might help to resolve this issue.

Equation 1 also predicts that in pressure-support ventilation at any given P_{aw}, the improvement of respiratory mechanics, shown as a reduction in elastance of the respiratory system (ie, an increase in compliance) would increase V_T. High compliance and P_{mus} would combine to further increase V_T. The fact that our patient exhibited high V_T at a low pressure-support level would suggest that lung compliance was normal. However, this assumption is not supported by the findings of initially decreasing static compliance and the persistence of bilateral lung infiltrates on CT. Therefore, it is likely that the high ventilatory demand increased the patient's effort, which resulted in

high V_T, leading to barotrauma in a still heterogeneous and diseased lung. The origin of the increased ventilatory demand sustained over time was, however, not clear in the present case. It is interesting to observe that between day 9 and day 10, V_T increased by 50% despite no change in pressure support (see Fig. 1). It could be that at this time the sedation, which was interrupted several days before, was completely washed-out, leading to a marked further increase in the patient's inspiratory effort. We have, however, no evidence to support that hypothesis, as we did not directly measure the patient's effort.

In short, this case highlights that in pressure-support ventilation, V_T must be carefully maintained, and the same rules for lung protection as in the early stage of ARDS should be applied.

REFERENCES

1. The Acute Respiratory Distress Syndrome Network. 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 2000;342(18):1301-1308.
2. Pasquet F, Pavic M, Karkowski L, Galoo E, Vitry T, Debourdeau P. A rare complication of gastroscopy. Rev Med Interne 2008;29(10):823-824.
3. Weg JG, Anzueto A, Balk RA, Wiedemann HP, Pattishall EN, Schork MA, et al. The relation of pneumothorax and other air leaks to mortality in the acute respiratory distress syndrome. N Engl J Med 1998;338(6):341-346.
4. Brower RG, Lanken PN, MacIntyre N, Matthay MA, Morris A, Ancukiewicz M, et al. Higher versus lower positive end-expiratory pressures in patients with the acute respiratory distress syndrome. N Engl J Med 2004;351(4):327-336.
5. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, et al. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. N Engl J Med 1998;338(6):347-354.
6. Mercat A, Richard JC, Vielle B, Jaber S, Osman D, Diehl JL, et al. Positive end-expiratory pressure setting in adults with acute lung injury and acute respiratory distress syndrome: a randomized controlled trial. JAMA 2008;299(6):646-655.
7. Meade MO, Cook DJ, Guyatt GH, Slutsky AS, Arabi YM, Cooper DJ, et al. Ventilation strategy using low tidal volumes, recruitment maneuvers, and high positive end-expiratory pressure for acute lung injury and acute respiratory distress syndrome: a randomized controlled trial. JAMA 2008;299(6):637-645.
8. Eisner MD, Thompson BT, Schoenfeld D, Anzueto A, Matthay MA. Airway pressures and early barotrauma in patients with acute lung injury and acute respiratory distress syndrome. Am J Respir Crit Care Med 2002;165(7):978-982.
9. Boussarsar M, Thierry G, Jaber S, Roudot-Thoraval F, Lemaire F, Brochard L. Relationship between ventilatory settings and barotrauma in the acute respiratory distress syndrome. Intensive Care Med 2002;28(4):406-413.
10. Milic-Emili J, Mead J, Turner JM. Topography of esophageal pressure as a function of posture in man. J Appl Physiol 1964;19:212-216.
11. Zin W, Milic Emili J. Esophageal pressure measurement. In: Tobin MJ, editor. Principles and practice of intensive care monitoring. New York: McGraw-Hill; 1998:545-552.
12. Talmor D, Sarge T, Malhotra A, O'Donnell CR, Ritz R, Lisbon A, et al. Mechanical ventilation guided by esophageal pressure in acute lung injury. N Engl J Med 2008;359(20):2095-2104.
13. Neumann P, Wrigge H, Zinserling J, Hinz J, Maripuu E, Andersson LG, et al. Spontaneous breathing affects the spatial ventilation and perfusion distribution during mechanical ventilatory support. Crit Care Med 2005;33(5):1090-1095.
14. Putensen C, Mutz NJ, Putensen-Himmer G, Zinserling J. Spontaneous breathing during ventilatory support improves ventilation-perfusion distributions in patients with acute respiratory distress syndrome. Am J Respir Crit Care Med 1999;159(4 Pt 1):1241-1248.
15. Putensen C, Zech S, Wrigge H, Zinserling J, Stuber F, Von Spiegel T, et al. Long-term effects of spontaneous breathing during ventilatory support in patients with acute lung injury. Am J Respir Crit Care Med 2001;164(1):43-49.
16. Mascheroni D, Kolobow T, Fumagalli R, Moretti MP, Chen V, Buckhold D. Acute respiratory failure following pharmacologically induced hyperventilation: an experimental animal study. Intensive Care Med 1988;15(1):8-14.
17. Schultz MJ, Haitsma JJ, Slutsky AS, Gajic O. What tidal volumes should be used in patients without acute lung injury?. Anesthesiology 2007;106(6):1226-1231.
18. Gajic O, Dara SI, Mendez JL, Adesanya AO, Festic E, Caples SM, et al. Ventilator-associated lung injury in patients without acute lung injury at the onset of mechanical ventilation. Crit Care Med 2004;32(9):1817-1824.
19. Gajic O, Frutos-Vivar F, Esteban A, Hubmayr RD, Anzueto A. Ventilator settings as a risk factor for acute respiratory distress syndrome in mechanically ventilated patients. Intensive Care Med 2005;31(7):922-926.
20. Jia X, Malhotra A, Saeed M, Mark RG, Talmor D. Risk factors for ARDS in patients receiving mechanical ventilation for > 48 h. Chest 2008;133(4):853-861.