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

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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 FIO_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 pneumonia 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, FIO_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-

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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 H2O to 31.0 mL/cm H2O.

Pressure-support ventilation was started on day 7, at 20 cm H2O, and gradually lowered, by 5 cm H2O 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 H2O. As our weaning protocol states that volume-controlled mode must be resumed when 20 cm H2O 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 on 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. VT increased progressively, despite a progressive reduction in the level of pressure support (Fig. 1). On day 20 a gastroscopy showed gastric ulcer, 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 but the delay after the procedure, the rapid reversal of the air leaks after lowering VT, 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 VT,1 or high or low PEEP at fixed VT 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 $P_{\text{plat}} > 35$ cm H2O 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 ($P_{\text{mus}}$) and the pressure generated by the ventilator ($P_{\text{aw}}$). The equation of motion of the respiratory system predicts that during inspiration $P_{\text{mus}}$ and $P_{\text{aw}}$ dissipate against the resistance to gas flow and the elastic recoil of the respiratory system above the end-expiratory elastic equilibrium position:

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

where R is the resistance of the airways, $V$ 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, $P_{\text{mus}}$ increases. If we assume that $P_{\text{mus}}$ 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, $P_{\text{aw}}$ should decrease and the total applied pressure should remain constant, with no change in delivered $V_{\text{T}}$. In volume-controlled mode, $V_{\text{T}}$ is the
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 (\( PL \)) increases. As predicted by the right side of Equation 1, the patient receives increased \( V \) and \( VT \). The distribution of this high \( VT \) 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 \( VT \) 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 \( VT \) is poorly controlled by the physician or respiratory therapist.

The key points that must be emphasized are that: (1) \( PL \) and thus \( VT \) 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 \( VT \) can be delivered for several hours per day because neither medical nor nursing staff pay close attention to \( VT \) in pressure-support ventilation, and (4) it is often thought that lung protection is achieved by setting a pressure level below 30 cm H\(_2\)O, but this is not the case. It is important to emphasize here that \( P_{plat} \) could have been very high if the same \( VT \) (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, the central venous pressure via a catheter inserted into the superior vena cava. In routine clinical practice, the measurement of esophageal pressure is the method of choice to estimate pleural pressure, even though it has clear constraints. 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.

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. Similar findings were also obtained in humans with ARDS. 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. However, an experimental study with normal sheep lungs found that prolonged hyperventilation resulted in acute lung injury. Finally, several reports have strongly suggested that large \( VT \) delivered by mechanical ventilation to normal lungs may predict the development of ARDS in humans. 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 \( VT \). High compliance and \( P_{mus} \) would combine to further increase \( VT \). The fact that our patient exhibited high \( VT \) 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

![Computed tomogram shows anterior pneumomediastinum (arrows).](image-url)
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