Use of Hypothermia to Allow Low-Tidal-Volume Ventilation in a Patient With ARDS

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Low-tidal-volume ventilation reduces mortality in patients with ARDS, but there are often challenges in implementing lung-protective ventilation, such as acidosis from hypercapnia. In a patient with severe ARDS we achieved adequate ventilation with a very low tidal volume (4 mL/kg ideal body weight) by inducing mild hypothermia (body temperature 35–36°C). *Key words: acute respiratory distress syndrome; ARDS; mechanical ventilation; lung-protective ventilation; tidal volume; hypothermia; permissive hypercapnia.* [Respir Care 2011;56(12):1956–1958.

Introduction

ARDS is defined as an acute onset of severe respiratory distress with bilateral infiltrates on chest radiograph, an absence of left-atrial hypertension, a pulmonary capillary wedge pressure of ≤ 18 mm Hg, no clinical signs of left-heart failure, and severe hypoxemia ($P_{aO_2}/F_{IO_2} \leq 200$ mm Hg).[1] The current standard practice for the management of ARDS includes lung-protective ventilation, which entails low tidal volume (V_T) (6 mL/kg ideal body weight), plateau pressure ≤ 30 cm H_2O, and modest PEEP.

Low-V_T ventilation is the only ventilation strategy that has been shown to reduce mortality in patients with ARDS.[2] In the ARDS Network trial,[2] patients were randomized to either low V_T (≤ 6 mL/kg predicted body weight) and plateau pressure ≤ 30 cm H_2O or conventional ventilation with higher V_T. Low V_T had a 9% absolute reduction in mortality risk, more ventilator-free hospital days, and more days free from nonpulmonary-organ failure. However, we do not know if further lowering V_T or airway pressure would further improve survival.

In addition to lung-protective ventilation, many unproven strategies have been attempted to improve oxygenation in patients with severe ARDS, including alveolar recruitment, airway pressure-release ventilation, high-frequency oscillatory ventilation, and high-frequency percussive ventilation. Other strategies include paralysis (which recently was found to improve survival in early stages of ARDS, possibly by improving patient-ventilator synchrony and decreasing barotrauma and biotrauma),[3] inhaled nitric oxide, inhaled prostacyclin (which improves ventilation-perfusion matching), extracorporeal membrane oxygenation, and positioning maneuvers such as prone position.

Barriers to the use of low-V_T ventilation in patients with ARDS include hypercapnia and acidosis. We hypothesized that inducing hypothermia might allow very-low-V_T ventilation by decreasing carbon dioxide production and thus decreasing respiratory acidosis. There have been case reports on hypothermia as an adjunct in treating patients with ARDS. One patient with ARDS and sepsis had improved oxygenation with hypothermia.[4] Wetterberg and Steen reported the successful use of hypothermia and a buffer infusion in a patient with ARDS.[5] However, neither of those patients underwent hypothermia in order to allow low-V_T ventilation.

Case Report

A 29-year-old man was admitted to the intensive care unit after an aspiration event. He was intubated on arrival

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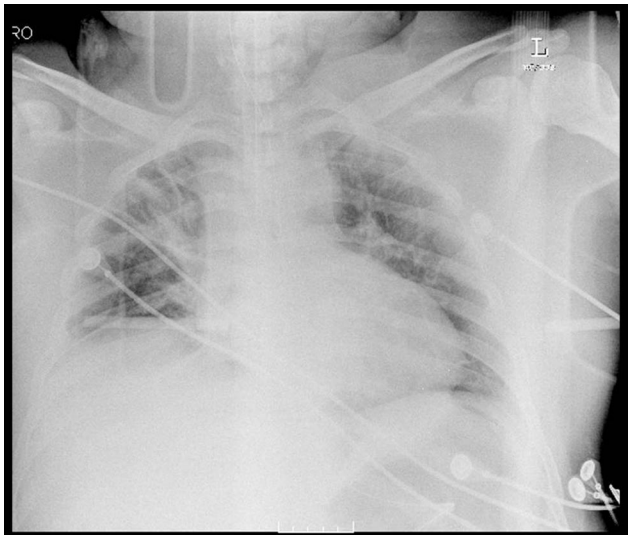

Fig. 1. Radiograph on admission day 3 is consistent with ARDS.

to the hospital. His arterial blood gases (pH 7.33, P_{aCO_2} 44 mm Hg, and P_{aO_2} 109 mm Hg on 100% oxygen) and chest radiograph (Fig. 1) were consistent with ARDS. His oxygenation continued to worsen, and he was paralyzed to improve patient-ventilator synchrony and to achieve lung-protective ventilation. His temperature rose to 38.4°C after he was admitted to the intensive care unit. He was actively cooled to 35–36°C in an effort to decrease carbon dioxide production. By achieving a temperature of 35–36°C we were able to decrease V_T to 4 mL/kg ideal body weight (Table 1). The other ventilator settings were held constant (PEEP 12 cm H_2O, F_{IO_2} 0.60, respiratory rate 28 breaths/min). His hemodynamic variables were: blood pressure range 117–147/68–76 mm Hg, central venous pressure range 8–10 mm Hg, heart rate range 98–108 beats/min. His plateau pressure dropped from 23 cm H_2O to 18 cm H_2O with low-V_T ventilation. While there was an increase in his P_{aCO_2}, his pH remained stable despite the low V_T (see Table 1). His pH and P_{aCO_2} tolerated the 4 mL/kg V_T, and ventilator settings were maintained.

After 6 days of hypothermia we rewarmed the patient to evaluate for clinical improvement. Over the course of 8 hours his temperature increased from 35.9°C to 37.9°C, pH decreased from 7.41 to 7.24, and P_{aCO_2} increased from 48 mm Hg to 71 mm Hg. Over the next 24 hours we again cooled him to 35.7°C, and his pH increased to 7.43 and P_{aCO_2} decreased to 40 mm Hg. We made no ventilation changes during this period.

Because of worsening hypoxemia, despite paralysis and lung-protective ventilation, we initiated inhaled nitric oxide therapy and his oxygenation dramatically improved. We transitioned to inhaled prostaglandin, and then to an oral phosphodiesterase inhibitor. He was eventually extubated and discharged from the hospital.

Discussion

We hypothesize that hypothermia and paralysis allowed us to use a very low V_T and a low plateau pressure by decreasing the metabolic rate and carbon dioxide production and thus preventing a substantial P_{aCO_2} increase and acidosis. While it is likely that an even lower V_T could be achieved with a greater degree of hypothermia, our target was very mild hypothermia of 35–36°C, in order to avoid the complications of hypothermia, which include higher infection rate and coagulopathy. We believe that even very mild hypothermia reduces the metabolic rate enough to achieve low-V_T ventilation. Our patient's acidosis was relatively mild (pH 7.31) and was well tolerated on a V_T of 4 mL/kg ideal body weight. It would be interesting to further investigate mild hypothermia as a means of achieving low-V_T ventilation and lower plateau pressure in patients with severe ARDS.

While our approach in this patient was to minimize CO_2 production, other authors have proposed unconventional methods to improve CO_2 removal, such as decreasing airways dead space. In a laboratory study in sheep, Rossi et al developed a modified method of transtracheal gas insufflation that they called intratracheal pulmonary ventilation.[6] A continuous flow of fresh gas is delivered at the

Table 1. Body Temperature, Tidal Volume, Gas Exchange, and Airway Pressure in a Patient With ARDS

Temperature (°C)	V_T (mL)	V_T (mL/kg)	pH	P_{aCO_2} (mm Hg)	P_{aO_2} (mm Hg)	Peak Airway Pressure (cm H_2O)	Plateau Airway Pressure (cm H_2O)
38.0	394	6	7.36	58	64	25	23
38.4	351	6	7.41	51	80	23	21
36.0	354	6	7.42	50	74	23	21
36.0	340	6	7.36	62	68	23	21
35.5	296	5	7.37	57	61	24	22
35.5	238	4	7.31	68	72	20	18

V_T = tidal volume

carina, so the upper-airways dead space is bypassed. In healthy sheep, intratracheal pulmonary ventilation allowed lowering V_T to as low as 1 mL/kg while maintaining normocapnia. Similarly, during continuous apneic ventilation, oxygen is usually delivered at the carina while maintaining a stable level of hypercapnia.[7]

A different and fascinating method of removing CO_2 is artificial blood filtration. Gattinoni et al used venovenous extracorporeal membrane oxygenation to remove most of the metabolic carbon dioxide in ARDS patients.[8] More recently, Cressoni et al, in a sheep model, found that half of metabolic CO_2 production could be removed with a commercial hemofilter and a replacement solution containing sodium hydroxide.[9]

Both approaches (lowering CO_2 production and increasing CO_2 removal) may permit low-V_T ventilation and thus decrease the risk of lung injury. In a paralyzed patient, inducing hypothermia to 35–36°C is a safe and low-risk strategy that can be a useful adjunct to other strategies for managing ARDS.

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