Managing Acute Lung Injury

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KEYWORDS

• Mechanical ventilation • Acute respiratory distress syndrome • Lung-protective ventilation
• Acute lung injury • Prone ventilation • Respiratory failure

KEY POINTS

- Limiting tidal volume to roughly 6 mL/kg predicted body weight protects the lung against ventilator-induced injury.
- PEEP should be sufficiently high to prevent cyclic recruitment and derecruitment and allow adequate oxygenation, but not so high as to overdistend the lung.
- Choosing a ventilator mode depends, in part, on whether to facilitate or attempt to suppress patient effort.
- For patients with early, severe ARDS, prone ventilation and neuromuscular blockade may improve survival.
- Mechanical ventilation for ARDS should be guided by severity, the potential for recruitment, and the response to initial ventilator settings.

The fact that mechanical ventilation can support the failing lung and amplify its injury (see Jeremy R. Beitler, Atul Malhotra, B. Taylor Thompson's article "Ventilator-Induced Lung Injury," in this issue) has profound implications for the details of ventilator management. As a consequence, the last two decades have witnessed a paradigm shift in the management of the acute respiratory distress syndrome (ARDS), as attention has turned from normalizing blood gas values to protecting the injured lung. The biophysical determinants of ventilator-induced lung injury (VILI) remain incompletely delineated and how to translate what is known into "safe" ventilator settings is controversial (**Table 1**). Nevertheless, this article provides concrete clinical recommendations by addressing ventilation modes and volumes, how to set positive end-expiratory pressure (PEEP) optimally, the role of spontaneous breathing effort, prone ventilation, the impact of ventilator settings on the heart and circulation, and rescue strategies for the most critically ill patients.

BALANCING LIFE SUPPORT AND LUNG PROTECTION

Data from animal studies conducted in the 1970s and 1980s suggested that reducing ventilator tidal volumes (V_T) could protect against VILI. At the same time, reducing V_T worsened oxygenation and the likelihood of hypercapnic acidosis. This kind of apparent tradeoff (higher PEEP might reduce atelectrauma but produce overstretch; higher respiratory rate might dampen acidemia but deliver more harmful energy to the lung[1]; high-frequency oscillatory ventilation [HFOV] might keep the lung open but compromise right ventricular [RV] function) makes consensus around ventilator strategies difficult to achieve (**Table 2**). The ARMA Trial provided clear evidence

Disclosure Statement: The author has nothing to disclose.
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Clin Chest Med ■ (2016) ■–■
http://dx.doi.org/10.1016/j.ccm.2016.07.005

Table 1
Common ventilator settings for patients with ARDS

Mode	Settings
V-ACV (ARMA)	Tidal volume: 6 mL/kg PBW Pplat $\leq$30 cm H_2O Mean RR: 30/min I:E = 1:1–1:3 PEEP: low PEEP table
V-ACV (ExPress)	Tidal volume: 6 mL/kg PBW Pplat $\leq$30 cm H_2O Mean RR: 28/min PEEP: raised until Pplat 28–30
P-ACV (LOVS)	Pressure adjusted to yield: 6 mL/kg PBW Mean RR: 25/min I:E = 1:1–1:3 PEEP: high PEEP table
PRVC	Tidal volume target: 6 mL/kg PBW Mean RR: 25–30 I:E = 1:1–1:3

Abbreviations: ARMA, acute respiratory management in ARDS; ExPress, expiratory pressure study group; I:E, inspiratory to expiratory ratio; LOVS, lung open ventilation study; P-ACV, pressure assist-control ventilation; PBW, predicted body weight; Pplat, plateau airway pressure; PRVC, pressure-regulated volume control; RR, respiratory rate; V-ACV, volume assist-control ventilation.

that 6, rather than 12 mL/kg predicted body weight (PBW) improved survival despite detrimental effects on gas exchange, but such high-quality evidence surrounding patient-centered outcomes is simply unavailable for most questions.

Lacking outcome data, clinicians often rely on surrogate measures, such as blood gas values, to judge ventilator settings, but this approach is fraught. For example, when an intervention raises Pa_{O_2}, it is commonplace to infer that the change was "beneficial." Although some treatments that raise Pa_{O_2} do improve outcomes (eg, prone positioning), many do not (large V_T, HFOV, airway pressure-release ventilation [APRV], inhaled nitric oxide). Thus approaches to mechanical ventilation in ARDS should be viewed skeptically[2] until they are tested in high-quality clinical trials or the basic science is more clearly delineated.

LIMITING LUNG OVERSTRETCH

Conventional Modes

As described in Jeremy R. Beitler, Atul Malhotra, B. Taylor Thompson's article "Ventilator-Induced Lung Injury," in this issue, stretching the lung beyond a threshold produces injury. The simplest way to limit end-inspiratory lung volume is to keep V_T small. PEEP has an impact, too, and this is discussed further later. A V_T of 6 mL/kg, based

Table 2
Tradeoffs in balancing life support and lung protection

	Potential Benefit	Possible Harm
Lower V_T (6 mL/kg PBW)	Less overstretch	More acidemia, sedation
Higher PEEP	Avoid atelectrauma	Greater overstretch
High RR	Less acidemia	More power dissipation to lung
HFOV	Enhanced recruitment, low ΔP	Higher Ptp
Cis-atracurium	Limit overstretch, reduced Ptx	Myopathy, more sedation
V-ACV	Control of V_T	Reduced comfort
ΔP <15 cm H_2O	Limit overstretch	More acidemia
Ultralow V_T with $ECCO_2R$	Much less overstretch	Cost, risks of extracorporeal circuit
ECMO	Much less power dissipation	Cost, risks of extracorporeal circuit
Prone position	More homogenous ventilation, PEEP	Pressure necrosis
Measure Ptp	Avoid inadequate PEEP	Somewhat invasive, technically demanding
Recruitment maneuver	Recruit lung, raise Pa_{O_2}	Hemodynamic effects, Ptx
Inhaled vasodilators	Higher Pa_{O_2}, less PEEP	Cost, renal impairment

Abbreviations: $ECCO_2R$, extracorporeal CO_2 removal; ECMO, extracorporeal membrane oxygenation; PBW, predicted body weight; Ptp, transpulmonary pressure; Ptx, pneumothorax; RR, respiratory rate; V-ACV, volume assist-control ventilation.