Risks and Complications of Mechanical Ventilation

Ewan C. Goligher MD PhD
Assistant Professor of Medicine, University of Toronto
Attending Physician, MSICU, Toronto General Hospital
Scientist, Toronto General Hospital Research Institute
Disclosures

• Conflicts of Interest
  – Equipment from Timpel
  – Equipment and personal fees from Getinge
Modern Mechanical Ventilation Saves Lives

**Table III—Mortality Rates**

<table>
<thead>
<tr>
<th>Group</th>
<th>Period of admission</th>
<th>No. of cases</th>
<th>Died</th>
<th>Died within three days</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>July 24—Aug. 25</td>
<td>31</td>
<td>27 (87%)</td>
<td>19 (70%)</td>
</tr>
<tr>
<td>II</td>
<td>Aug. 26—Sept. 8</td>
<td>50</td>
<td>26 (52%)</td>
<td>7 (27%)</td>
</tr>
<tr>
<td>III</td>
<td>Sept. 8—Sept. 23</td>
<td>50</td>
<td>24 (48%)</td>
<td>8 (33%)</td>
</tr>
<tr>
<td>IV</td>
<td>Sept. 23—Oct. 5</td>
<td>50</td>
<td>19 (38%)</td>
<td>10 (53%)</td>
</tr>
<tr>
<td>V</td>
<td>Oct. 6—Oct. 21</td>
<td>50</td>
<td>13 (26%)</td>
<td>7 (54%)</td>
</tr>
<tr>
<td>VI</td>
<td>Oct. 21—Nov. 6</td>
<td>50</td>
<td>18 (36%)</td>
<td>10 (55%)</td>
</tr>
<tr>
<td>Total II–VI</td>
<td>...</td>
<td>250</td>
<td>100 (40%)</td>
<td>42 (42%)</td>
</tr>
</tbody>
</table>

Lassen HCA *Lancet* 1953
West JB *JAP* 2005
Risks and Complications

- Hemodynamic effects
- Ventilation-induced lung injury
- Ventilator-induced diaphragm dysfunction
Hemodynamics: Heart-Lung Interactions

Venous return
Hemodynamics: Right Ventricle

Venous return
Hemodynamics: Right Ventricle


![Graph showing hemodynamics related to right ventricle pressure and mortality.](image)
Hemodynamics: Right Ventricle

**RECRUITMENT**
- Reverse hypoxic pulmonary vasoconstriction
- Unloads the RV
- Increases CO
- Improves hemodynamics

**OVERINFLATION**
- Overloads the RV
- Decreases CO
- Compromises hemodynamics

Pulmonary Vascular Resistance

Lung stress (TPP)

Repessé et al *Chest* 2015
Hemodynamics: Left Ventricle
Ventilation-Induced Lung Injury

Webb & Tierney<br>ARRD 1974
Ventilation-Induced Lung Injury

B  Ventilation at high lung volume

Normal

Hyperinflation

Air leaks

Overdistention

Slutsky & Ranieri *NEJM* 2013
Ventilation-Induced Lung Injury

A Ventilation at low lung volume

End expiration
End inspiration

Atelectrauma
Lung inhomogeneity

Slutsky & Ranieri NEJM 2013
Ventilation-Induced Lung Injury

Conventional ventilation involved a tidal volume of 12 ml per kilogram of body weight, a low PEEP, and a partial pressure of carbon dioxide of 35 to 38 mm Hg. Protective ventilation involved a tidal volume at or below 6 ml per kilogram, a high PEEP, and permissive hypercapnia. The mortality rate at 28 days was significantly lower with protective ventilation than with conventional ventilation (38% vs. 71%). There was also significantly less clinical barotrauma and a significantly higher rate of weaning from ventilation in the protective-ventilation group. Although some criticized this study for the high mortality rate in the conventional-ventilation group, the patients studied were extremely ill (with failure of a mean of 3.6 organs per patient).

In a subsequent, larger study by the Acute Respiratory Distress Syndrome Network (ARDSNet), 861 patients with acute lung injury or ARDS were randomly assigned to receive ventilatory support involving a tidal volume of either 12 or 6 ml per kilogram of predicted body weight. Although tidal volume was the manipulated variable, a major goal of the ventilatory strategy was to keep the plateau airway pressure below 30 cm of water; therefore, the group that underwent ventilation at 6 ml per kilogram of predicted body weight is often referred to as the low-stretch group. The low-stretch strategy was associated with a significantly lower mortality rate (31%, vs. 40% with ventilation at 12 ml per kilogram of predicted body weight). Therefore, the best available evidence is for a ventilation strategy using a tidal volume of 6 ml per kilogram of predicted body weight for patients with acute lung injury or ARDS.

Three other small, randomized trials, performed during the same period, failed to demonstrate a survival benefit with the low-tidal-volume strategy.

This example of ventilation of a 70-kg patient with ARDS shows that conventional ventilation at a tidal volume of 12 ml per kilogram of body weight and an end-expiratory pressure of 0 cm of water (Panel A) can lead to alveolar overdistention (at peak inflation) and collapse (at the end of exhalation). Protective ventilation at a tidal volume of 6 ml per kilogram (Panel B) limits overinflation and end-expiratory collapse by providing a low tidal volume and an adequate positive end-expiratory pressure. Adapted from Tobin.
Ventilation-Induced Lung Injury

\[ P_L = P_{airway} - P_{pleural} \]
Monitoring

Flow (L/min)

-50
-25
0
25
50

Time (sec)

Paw (cmH₂O)

-10
0
10
20
30

Time (sec)

Inspiratory hold

Expiratory hold

ΔPₐw

Pₖₚ₇₅

P₀.₁

P₀.₅
Lung-Protective Ventilation

• Aim for lower tidal volumes in patients with ARDS
  – Vt ≤6-8 ml/kg predicted body weight

• Minimize the pressure applied to the lung
  – Driving pressure ≤15 cm H$_2$O
  – Plateau pressure ≤30 cm H$_2$O

• Avoid excessive respiratory efforts
  – Pocc <15 H$_2$O
  – $P_{0.1}$ <3.5 cm H$_2$O
Ventilator-Induced Diaphragm Dysfunction

Patients are generally provided with positive-pressure mechanical ventilation when their own ventilatory capabilities are outstripped by the demands imposed by various disease states (Fig. 1). Positive-pressure mechanical ventilation is also needed when the respiratory drive is reduced by disease or drugs and the patient is incapable of initiating ventilatory activity. As these reasons for providing mechanical ventilatory support stabilize and begin to resolve, the clinical focus must be directed toward strategies that remove the ventilator as quickly as possible. Unnecessary delays in this withdrawal process increase the complication rate of mechanical ventilation (eg, pneumonia, discomfort) and drive up cost. Aggressiveness in removing ventilatory support, however, must be balanced against the risks of prematurely withdrawing that support, including difficulty in re-establishing the artificial airway, ventilatory muscle fatigue, and compromised gas exchange.

There are 2 fundamental issues involved in the management of mechanically ventilated patients whose disease process has begun to stabilize and/or reverse. First, appropriate assessment techniques are needed to identify patients capable of ventilator withdrawal. Once identified, these patients should have the device removed promptly. Second, in patients judged to still require mechanical ventilatory support, appropriate management strategies are needed, which should include regular withdrawal reassessments. This article focuses on these 2 issues and emphasizes applied respiratory physiology as an adjunct in the decision-making processes. As much as possible, my comments will be evidence-based. Indeed, this discussion will rely heavily on the comprehensive evidence-based review, by the McMaster University evidence-based-research group, of the ventilator withdrawal process and the sub-

Neil R MacIntyre MD FAARC is affiliated with Respiratory Care Services, Duke University Medical Center, Durham, North Carolina.

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Correspondence: Neil R MacIntyre MD FAARC, Respiratory Care Services, PO Box 3911, Duke University Medical Center, Durham NC 27710.

E-mail: neil.macintyre@duke.edu.
Ventilator-Induced Diaphragm Dysfunction

Figure 1. A comparison of histologic sections of case and control specimens. Panels A and B (hematoxylin and eosin) show that neither inflammatory infiltrate nor necrosis is present in case or control specimens. The sections in Panels C and D were preincubated with NOQ7.5.4D antibody, which reacts with all myosin light chains. The antibody appears black. In Panels C, D, E, and F, a representative slow-twitch fiber is indicated by an open circle and a fast-twitch fiber by an open square. The proportion of slow- and fast-twitch fibers (Panel B), and control specimens do not differ with respect to these transcripts noted in the diaphragm-biopsy specimens from case subjects are consistent with the idea that weaning patients from ventilators is closely linked to diaphragm force generation, which reacts with all myosin light chains. The antibody appears black. In Panels C, D, E, and F, a representative slow-twitch fiber is indicated by an open circle and a fast-twitch fiber by an open square. The proportion of slow- and fast-twitch fibers (Panel B), and control specimens do not differ with respect to these transcripts noted in the diaphragm-biopsy specimens from case subjects are consistent with the idea that weaning patients from ventilators is closely linked to diaphragm force generation.
Ventilator-Induced Diaphragm Dysfunction

Control specimen

Resistive loading specimen

Reid et al. *J Appl Phys* 1994
Ventilator-Induced Diaphragm Dysfunction

Goligher et al. AJRCCM 2015
Monitoring

- Inspiratory hold
- Expiratory hold

Flow (L/min)

Paw (cmH₂O)

Time (sec)
Diaphragm-Protective Ventilation

• Minimize duration of passive ventilation
• Aim for resting effort level
  – Clinical trials awaited
• Lung protection > diaphragm protection
Summary: Risks and Complications

- Hemodynamic effects
- Ventilation-induced lung injury
- Ventilator-induced diaphragm dysfunction
Questions?

EWAN.GOLIGHER@UTORONTO.CA