

# CHEST<sup>®</sup>

Official publication of the American College of Chest Physicians



## Prone Position in ARDS

Alain F. Broccard

*Chest* 2003;123:1334-1336  
DOI 10.1378/chest.123.5.1334

The online version of this article, along with updated information and services can be found online on the World Wide Web at:  
<http://www.chestjournal.org/content/123/5/1334.full.html>

CHEST is the official journal of the American College of Chest Physicians. It has been published monthly since 1935. Copyright 2007 by the American College of Chest Physicians, 3300 Dundee Road, Northbrook IL 60062. All rights reserved. No part of this article or PDF may be reproduced or distributed without the prior written permission of the copyright holder.  
(<http://www.chestjournal.org/site/misc/reprints.xhtml>) ISSN:0012-3692

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S<sup>®</sup>

## REFERENCES

- 1 Leung RS, Bradley DT. Sleep apnea and cardiovascular disease. *Am J Respir Crit Care Med* 2001; 164:2147–2165
- 2 Shahar E, Whitney CW, Redline S, et al. Sleep-disordered breathing and cardiovascular diseases: cross sectional results of the Sleep Heart Health Study. *Am J Respir Crit Care Med* 2001; 163:19–25
- 3 Javaheri S, Parker TJ, Liming JD, et al. Sleep apnea in 81 ambulatory male patients with stable heart failure: types and their prevalences, consequences and presentations. *Circulation* 1998; 97:2154–2159
- 4 Sin DD, Fitzgerald F, Parker JD, et al. Risk factors for central and obstructive sleep apnea in 450 women and men with congestive heart failure. *Am J Respir Crit Care Med* 1999; 160:1101–1106
- 5 Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study: Sleep Heart Health Study. *JAMA* 2000; 283:1829–1836
- 6 Young T, Peppard P, Palta M, et al. Population based study of sleep disordered breathing as a risk factor for hypertension. *Arch Intern Med* 1997; 157:1746–1752
- 7 Peppard P, Young T, Palta M, et al. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med* 2000; 359:204–210
- 8 Levy D, Larson MG, Vasani RS, et al. The progression from hypertension to congestive heart failure. *JAMA* 1996; 275:1557–1563
- 9 Fletcher EC, Proctor M, Yu L et al. Pulmonary edema develops after recurrent obstructive apneas. *Am J Respir Crit Care Med* 1999; 160:1688–1696
- 10 Parker JD, Brooks D, Kozar LF, et al. Acute and chronic effects of airway obstruction on canine left ventricular performance. *Am J Respir Crit Care Med* 1999; 160:1888–1896
- 11 Hedner J, Ejnell H, Caidahl K. Left ventricular hypertrophy independent of hypertension in patients with obstructive sleep apnea. *J Hypertens* 1990; 8:244–264
- 12 Niroumand M, Kuperstein R, Sasson Z, et al. Impact of obstructive sleep apnea on left ventricular mass and diastolic dysfunction. *Am J Respir Crit Care Med* 2001; 163:1632–1636
- 13 Mehra MR, Ventura HO. Heart failure. Civetta JM, Taylor RW, Kirby RR, eds. *Critical care*. 3rd ed. Philadelphia, PA: JB Lippincott, 1997; 1887–1903
- 14 Tkacova R, Rankin F, Fitzgerald FS, et al. Effects of continuous positive airway pressure on obstructive sleep apnea and left ventricular afterload in patients with heart failure. *Circulation* 1998; 98:2269–2275
- 15 Logan AG, Tkacova R, Tisler A, et al. High prevalence of obstructive sleep apnea in refractory hypertension. *J Hypertens* 2001; 19:2271–2277

## Prone Position in ARDS

### Are We Looking at a Half-Empty or Half-Full Glass?

In 1976, Piehl and Brown<sup>1</sup> reported for the first time that ventilation in the prone position improves “oxygenation” in patients with acute hypox-

emic respiratory failure. Since then, numerous clinical series have been published regarding the use of prone positioning in patients with ARDS, but its impact on outcome remains uncertain.

Overall, prone positioning helps to improve gas exchange in approximately two thirds of the patients with ARDS.<sup>2</sup> The mechanisms that account for the rise in the arterial blood oxygen content have essentially been investigated in animal models of lung injury.<sup>3</sup> In these models,<sup>4</sup> as well as in many patients with ARDS,<sup>2</sup> the poorly and/or nonaerated lung units appear to be mainly localized in the dependent regions both in the supine and prone position. The time constant of the dependent collapsed/flooded lung units is such that tidal ventilation distributes preferentially to the “open” nondependent lung units.<sup>4</sup> Since the distribution of perfusion is largely gravity-independent, at least under West zonal 3 conditions, the largest proportion of the perfusion goes through the dorsal lung regions, with patients in both the supine and prone positions.<sup>5</sup> As a result, perfusion is largest in the dependent regions with the patient in the supine position and is largest in the nondependent region when the patient is in the prone position, and this remains true in the setting of lung injury.<sup>6</sup> Regardless of position, positive-pressure ventilation (*ie*, the creation of West zonal conditions 2 or 1) alters the vertical distribution of perfusion, and blood flow tends to be redistributed from the nondependent region to the dependent regions. Positive airway pressure thus tends to reduce the vertical perfusion gradient with the patient in the prone position and tends to amplify the gradient with the patient in the supine position.<sup>7</sup> It follows that with the patient in the supine position the vertical ventilation and perfusion gradients of mechanically ventilated injured lungs vary in opposite direction, promoting ventilation-perfusion mismatch and shunting. In contrast, a larger proportion of perfusion distributes to the well-ventilated nondependent regions (dorsal) and, everything else being equal, a smaller amount of desaturated blood perfuses the poorly and/or nonaerated lung regions with the patient in the prone position. This helps to explain the fact that the ventilation-perfusion relationship is more favorable with patients in the prone position than in the supine position.<sup>3,8</sup>

Additionally and/or alternatively, other factors may contribute to the improved gas exchange afforded by prone positioning. Along the vertical axis, the pleural pressure gradient is smaller with the patient in the prone position than in the supine position.<sup>9</sup> In the dependent regions, the pleural pressure is also comparatively less positive (more negative) with the patient in the prone position rather than in the supine position,<sup>10</sup> in large part because in the prone position the heart rests almost entirely on the ster-

num and exerts significantly less pressure on the lungs and pleural space than in the supine position.<sup>11</sup> These physiologic characteristics of prone positioning help to explain the more uniform vertical distribution of regional lung volume at relaxation and the rise in functional residual capacity that may be observed after turning from the supine position to the prone position.<sup>12</sup> Although a rise in functional residual capacity or recruitment can contribute to the improvement in gas exchange, PaO<sub>2</sub> may clearly rise without any demonstrable change in lung volume.<sup>13</sup> Together, these suggest that the improved gas exchange that is observed with the patient in the prone position may be due to the relatively larger perfusion in the dorsal aerating lung regions and/or to recruitment and better aeration of the dependent lung unit. In patients with ARDS, the effect of prone positioning on gas exchange apparently also depends on chest wall compliance,<sup>14</sup> suggesting that prone positioning is an important determinant of the regional ventilation/perfusion relationship in paralyzed mechanically ventilated subjects. Which one of the above mechanisms prevails and best explains the effect on gas exchange of prone positioning in a given patient is unknown but could be important in regard to its potential protective effects against ventilator-induced lung injury, and thus to its possible impact on outcome.

The improved physiologic understanding of the effect of prone positioning on the respiratory system and the encouraging preliminary clinical data have made it very tempting to ventilate patients with severe ARDS in the prone position in the recent years. The only randomized prospective study<sup>2</sup> that is available today has confirmed its safety and efficacy in improving gas exchange but has failed to show an overall reduction in mortality. For those among us who rely heavily if not exclusively on clinical outcome studies to decide what does and what does not work, prone positioning may thus appear as just another ARDS treatment that did not live up to its promise.

There are, however, good reasons not to regard the recent negative prone positioning study as indicating that the prone position is of no interest or only marginal interest. After all, it took not less than five randomized clinical trials to confirm the experimental data and to demonstrate that ventilation with excessive tidal volume detrimentally affects patients' outcomes. Given what we already know regarding prone positioning, it would be a mistake, I believe, to dismiss its potential benefit. The good practice of evidence-based medicine requires the assessment of all evidence, not just that of the results of outcome studies (which are often more apt to test the efficacy of a given protocol than the concept behind it) and

not the drawing of final conclusions based on a single outcome study. Apart from the now established fact that prone positioning improves arterial blood oxygen content in the majority of patients with ARDS, other encouraging facts should be kept in perspective.

Prone positioning has been established as safe in adults.<sup>2</sup> When close attention is paid particularly to the lines, the endotracheal tube, and the secretions that may occasionally enter the endotracheal tube during or shortly after repositioning, the rate of complications is not different in the prone position than in the supine position. The study by Haefner and colleagues in the current issue of *CHEST* (see page 1589) confirms the notion that prone positioning is safe in a challenging pediatric population receiving extracorporeal support, and thus at increased risk of bleeding complications. Their study also reinforces the idea that prone positioning does not require special beds or devices, which makes it both cheap and universally available. The study by Haefner et al is also unique in that prone positioning was not used primarily for short-term gas exchange improvements since no such effects would immediately be apparent during extracorporeal membrane oxygenation. Along these lines, the remarkably low mortality observed in a very sick pediatric population is also encouraging, but, given the limitations inherent to a retrospective study, no premature conclusion should be drawn in that regard.

The data of Haefner et al are, however, consistent with the *post hoc* analysis of the multicentric prospective study of Gattinoni and colleagues,<sup>2</sup> which suggested a reduction in mortality in the patients with the severest cases of ARDS or in patients who were at high risk of volutrauma (*ie*, patients ventilated with the largest tidal volume), and with the experimental data demonstrating that prone positioning helps to limit the incidence of ventilator-induced lung injury.<sup>15,16</sup> Compared to supine positioning, the lesser positive pleural pressure in the dependent thorax regions, the more uniform regional pleural pressure, lung volume, and perfusion observed in the prone position as the potential to limit lung volume loss/atelectrauma trauma and to distribute the tidal mechanical stress imposed on the vessels and airways more evenly between all lung regions.

In summary, none of the present studies on prone positioning, including the one by Gattinoni and colleagues,<sup>2</sup> should be considered definitive. All should, however, be looked at carefully to help design future studies. Clearly, more studies are needed before drawing any final conclusion regarding the effect (*eg*, mortality) of prone positioning on outcome. We need particularly to determine the target population that might benefit the most from prone positioning, and the optimal timing for its use

(*ie*, at what stage of the natural course of ARDS, and how often and for how long should we turn patients to the prone position and keep patients prone). It is also likely that the best prone ventilatory strategy differs from the best supine ventilatory strategy and that the intensity of abdominal compression that is allowed for the patient in the prone position also may be important. These issues have not been comprehensively addressed so far. As improved gas exchange *per se* is not the ultimate goal, we also need to determine how to best manage responders (*ie*, shall we aim at reducing airway pressure or the fraction of inspired oxygen?), and we should pay closer attention to how prone positioning affects the lungs rather than to how much it impacts PaO<sub>2</sub> in future studies. Although clinical outcome studies are very important, the premature or suboptimally designed study carries the risk of inappropriately changing the way we look at reality. Given the established safety, the frequent gas exchange improvement, and the potential beneficial effects on ventilator-induced lung injury that are associated with prone positioning, I do not see any compelling reason not to turn my next patient with severe ARDS to the prone position. I still believe the glass is half-full, not half-empty.

Alain F. Broccard, MD, FCCP  
St. Paul, MN

Dr. Broccard is Associate Professor of Medicine, University of Minnesota, and Medical Director of the Medical Intensive Care Unit, Regions Hospital.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (e-mail: [permissions@chestnet.org](mailto:permissions@chestnet.org)).

Correspondence to: Alain F. Broccard, MD, FCCP, University of Minnesota, Medical Director of the Medical Intensive Care Unit, Pulmonary and Critical Care Division, Regions Hospital, 640 Jackson St, St Paul, MN 55101-2595; e-mail: [brocc001@umn.edu](mailto:brocc001@umn.edu)

## REFERENCES

- 1 Piehl MA, Brown RS. Use of extreme position changes in acute respiratory failure. *Crit Care Med* 1976; 4:13-14
- 2 Gattinoni L, Tognoni G, Pesenti A, et al. Effect of prone positioning on the survival of patients with acute respiratory failure. *N Engl J Med* 2001; 345:568-573
- 3 Lamm WJ, Graham MM, Albert RK. Mechanism by which the prone position improves oxygenation in acute lung injury. *Am J Respir Crit Care Med* 1994; 150:184-193
- 4 Martynowicz MA, Minor TA, Walters BJ, et al. Regional expansion of oleic acid-injured lungs. *Am J Respir Crit Care Med* 1999; 160:250-258
- 5 Glenny RW, Lamm WJ, Albert RK, et al. Gravity is a minor determinant of pulmonary blood flow distribution. *J Appl Physiol* 1991; 71:620-629
- 6 Wiener CM, Kirk W, Albert RK. Prone position reverses gravitational distribution of perfusion in dog lungs with oleic acid-induced injury. *J Appl Physiol* 1990; 68:1386-1392
- 7 Nyren S, Mure M, Jacobsson H, et al. Pulmonary perfusion is more uniform in the prone than in the supine position:

- scintigraphy in healthy humans. *J Appl Physiol* 1999; 86: 1135-1141
- 8 Pappert D, Rossaint R, Slama K, et al. Influence of positioning on ventilation-perfusion relationships in severe adult respiratory distress syndrome. *Chest* 1994; 106:1511-1516
- 9 Mutoh T, Lamm WJ, Embree LJ, et al. Abdominal distension alters regional pleural pressures and chest wall mechanics in pigs *in vivo*. *J Appl Physiol* 1991; 70:2611-2618
- 10 Mutoh T, Guest RJ, Lamm WJ, et al. Prone position alters the effect of volume overload on regional pleural pressures and improves hypoxemia in pigs *in vivo*. *Am Rev Respir Dis* 1992; 146:300-306
- 11 Albert RK, Hubmayr RD. The prone position eliminates compression of the lungs by the heart. *Am J Respir Crit Care Med* 2000; 161:1660-1665
- 12 Pelosi P, Croci M, Calappi E, et al. The prone positioning during general anesthesia minimally affects respiratory mechanics while improving functional residual capacity and increasing oxygen tension. *Anesth Analg* 1995; 80:955-960
- 13 Albert RK, Leasa D, Sanderson M, et al. The prone position improves arterial oxygenation and reduces shunt in oleic-acid-induced acute lung injury. *Am Rev Respir Dis* 1987; 135: 628-633
- 14 Pelosi P, Tubiolo D, Mascheroni D, et al. Effects of the prone position on respiratory mechanics and gas exchange during acute lung injury. *Am J Respir Crit Care Med* 1998; 157:387-393
- 15 Broccard AF, Shapiro RS, Schmitz LL, et al. Influence of prone position on the extent and distribution of lung injury in a high tidal volume oleic acid model of acute respiratory distress syndrome. *Crit Care Med* 1997; 25:16-27
- 16 Broccard A, Shapiro RS, Schmitz LL, et al. Prone positioning attenuates and redistributes ventilator-induced lung injury in dogs. *Crit Care Med* 2000; 28:295-303

## Percutaneous Dilatational Tracheostomy

### We Live in a Twisted World

**I**n 175 AD, Galen inflated the lungs of a dead animal with a bellows, similar to that used to keep a fire alive. This was the beginning of mechanical ventilation. Marco Aurelio Severino (1580-1656) was one of the first to use the tracheostomy as an operation for obstructed air passages. With a trocar-like instrument, he was able to save innumerable lives during the diphtheria epidemic in Naples in 1610. Lorenz Heister (1683-1758) established the term *tracheotomy* for a windpipe incision.<sup>1</sup> From then on, many changes have occurred. With improvements in critical care, we now have patients receiving mechanical ventilation for years or even decades. For this reason, tracheotomies have become one of the most common surgeries performed in critically ill patients.

Traditionally, surgeons have performed tracheotomies in the operating room. Ciaglia et al<sup>2</sup> in 1985 demonstrated the feasibility and utility of the percu-

**Prone Position in ARDS**  
Alain F. Broccard  
*Chest* 2003;123; 1334-1336  
DOI 10.1378/chest.123.5.1334

**This information is current as of July 30, 2009**

<b>Updated Information &amp; Services</b>	Updated Information and services, including high-resolution figures, can be found at: <a href="http://www.chestjournal.org/content/123/5/1334.full.html">http://www.chestjournal.org/content/123/5/1334.full.html</a>
<b>References</b>	This article cites 15 articles, 10 of which can be accessed free at: <a href="http://www.chestjournal.org/content/123/5/1334.full.html#ref-list-1">http://www.chestjournal.org/content/123/5/1334.full.html#ref-list-1</a>
<b>Open Access</b>	Freely available online through CHEST open access option
<b>Permissions &amp; Licensing</b>	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: <a href="http://www.chestjournal.org/site/misc/reprints.xhtml">http://www.chestjournal.org/site/misc/reprints.xhtml</a>
<b>Reprints</b>	Information about ordering reprints can be found online: <a href="http://www.chestjournal.org/site/misc/reprints.xhtml">http://www.chestjournal.org/site/misc/reprints.xhtml</a>
<b>Email alerting service</b>	Receive free email alerts when new articles cite this article. sign up in the box at the top right corner of the online article.
<b>Images in PowerPoint format</b>	Figures that appear in CHEST articles can be downloaded for teaching purposes in PowerPoint slide format. See any online article figure for directions.

