in patients with acute respiratory distress syndrome (ARDS). Implicit in his comment is that, in the context of the results of the ARDS Network trial,2 which showed that a V_T of 6 mL/kg was associated with better survival in patients with ARDS, decreasing the V_T in response to decreasing compliance is beneficial and recommended. We believe that that contention, though broadly correct, overlooks the essential finding of the ARDS Network (in which our institution participated), that a specific V_T of 6 mL/kg is recommended, not a varying V_T , and not a V_T that exceeds 6 mL/kg, even if V_T decreases as the lung stiffens. Furthermore, in endorsing a maximum inspiratory pressure up to 50 cm H₂O, the "clarification" overlooks the target of a plateau pressure of $< 30 \text{ cm H}_2\text{O}$ in the ARDS Network trial,² which would probably be exceeded by a maximum inspiratory pressure of 50 cm H₂O in the absence of increased airway resistance (depending, of course, on the inspiratory flow rate). Furthermore, Mr Piper's comment about the device's automatic adjustment seems to imply that there is some intelligence in the adjustment process, which, of course, there is not. The adjustment is simply a mechanical response to a changing respiratory-system time constant. The only ventilators that make "intelligent" changes to the delivered V_T are much more sophisticated devices.³

The most important issue regarding "automatic" changes in ventilatory parameters is that, unlike any other ventilatory device, setting a "rate" on the Vortran device does not guarantee a preset number of mandatory breaths per minute, because the breaths are not time-triggered independent of the patient's respiratory-system mechanics (ie, resistance, compliance, and muscle activity). On the contrary, spontaneous breaths are pressure-triggered according to the interaction of the Vortran's internal leak flow (set by the "rate" knob) and the patient's inspired V_T and expiratory time constant. Indeed, the "rate" knob should be thought of not as a frequency control but rather as a trigger-sensitivity control. What the operator is really doing (with a passive patient) is setting the device to auto-trigger, much like a standard ventilator will do when there is a leak in the system.

The second contention is that our choosing a compliance of 14 mL/cm H₂O as a working condition in the study¹ was imprudent and cast the device's performance in an unfavorable light. As we stated, our

goal was to examine the device's performance under 2 mass-casualty conditions that would simulate those in which a portable, inexpensive device might be considered desirable, such as poisoning causing neuromuscular paralysis (in which the lung compliance would be expected to be normal) and acute lung injury/ARDS (in which the lung compliance would be decreased). Still, patients with ARDS have been reported to have average compliances as low as 37 mL/cm H₂O, with a standard deviation of 23 mL/cm H₂O, so compliance values in the teens would be expected in perhaps 30% of patients.4 In that context our choice of compliance values under which to simulate the use of the device seems defensible and appropriate.

Finally, Mr Piper found our statement about calculated $P_{\rm CO_2}$ misleading. We absolutely agree that only actual blood gas data from patients will settle the issue and allay concerns, but our experience in this study makes us reluctant to undertake actual clinical testing to resolve this.

Overall, we stand by our suggestions that, "The variable performance under changing load along with the lack of alarms should prompt caution in using the Vortran Automatic Resuscitator for emergency ventilatory support in situations where patients cannot be constantly monitored by trained and experienced operators." As evidence that truth in science is replication of findings, we point out that conclusions from other groups echo our concerns about the device.⁵

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More Environmental Prevention of Gram-Negative Infections Needed

I appreciated the excellent review article¹ by Robert Siegel on emerging antibiotic resistance of Gram-negative bacteria. Gram-negative bacteria account for a large percentage of the estimated 99,000 annual United States deaths due to hospital-acquired infections. Better antibiotic management and the development of new antibiotics are important for controlling Gram-negative bacteria. However, many environmental interventions exist that can prevent Gramnegative infections, but are often overlooked in hospital practice.

Hand-washing is the most important single step in preventing the spread of Gramnegative infections. Various studies have reported that viable bacteria are commonly found on the hands of health care providers; these include *Pseudomonas* (found on 1.3–25% of provider hands), *Acinetobacter* (3–15%), *Klebsiella* (17%), and vancomycinresistant enterococcae (41%).² An intervention to increase the use of alcoholbased hand rub and gloves reduced Gramnegative infections by 60% and Gram-positive infections by 60% (p < 0.001 for each comparison) in a neonatal intensive care unit.³

An intervention that involved education of hospital cleaning staff was associated with a 64% reduction in vancomycin-resistant enterococcae infection (95% confidence interval 0.19–0.68).⁴ Portable high-efficiency-particulate-air (HEPA) filters significantly reduce hospital airborne *Pseudomonas*.⁵ Siegel¹ cited several sources that reported that better disinfection and man-

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agement of respiratory equipment can reduce the spread of Gram-negative bacteria.¹ Meta-analyses have found that many non-pharmacologic interventions can significantly reduce the rate of ventilator-associated pneumonia; these include kinetic bed therapy, subglottic secretion drainage, heat-and-moisture exchangers (rather than heated humidifiers), and oral decontamination with chlorhexidine.⁶

Environmental controls can prevent a large percentage of hospital-acquired bacterial infections. Fewer nosocomial infections will reduce overall antibiotic use, which should reduce the risk of creating bacterial antibiotic resistance and improve the efficacy of antibiotics given to patients who do acquire infections. Much more research is needed on the prevention and antibiotic treatment of Gram-negative infections.

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The author responds:

Dr Curtis emphasizes some very important environmental issues that are frequently overlooked and some measures that can decrease the incidence of nosocomial infections. Organisms, such as *Acinetobacter*, that can persist for long periods on surfaces easily spread from patient to patient via hospital personnel (eg, radiology technicians and respiratory therapists) and equipment (eg, electrocardiographs, ultrasound machines, thermometers).

Improvements in intensive care unit design may reduce the risk from environmental surfaces that become colonized with pathogenic organisms. Curtains used for patient privacy may harbor organisms such as *Clostridium difficile* spores, and should be replaced with alternatives, such as E-glass or double glass plates with embedded shades

or blinds. Those surfaces are easy to clean and might help prevent infections by preventing colonization. Seamless intensive care unit floors and fabrication of environmental surfaces from materials that inhibit bacterial growth, such as copper, might also be effective. Research on those subjects is underway and may lead to new concepts and designs that provide a hospital milieu hostile to bacteria (personal communication, Neil A Halpern MD, Memorial Sloan-Kettering Cancer Center, New York, New York, April 28, 2008).

As Dr Curtis pointed out, subglottic suctioning decreases ventilator-associated pneumonia. Another strategy is silvercoated endotracheal tubes, which get less bacterial colonization. We clearly need to look at innovations that can prevent infections, because they are not only cost-effective but will reduce the development of resistant microorganisms, decrease the need for antibiotics, and save lives.

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