TB transmission to a health-care worker is an age old problem, but when it occurs, it should not be considered the “end of the world” but an opportunity for a highly effective intervention. A conversion to a positive Mantoux test merely means that one has recently been infected with the tubercle bacillus, a situation which is, in the overwhelming majority of cases, amenable with a simple, well-characterized, benign treatment. TB infection is a state which is shared with one-third of the world’s population. However with any documented new TB infection, an individual is, by definition, at presentable high risk for advancement to active TB.

The CDC has presented guidelines designed to foment administrative, engineering, and respiratory protection control for prevention of TB transmission. However, institutional resolve to constructively deal with any employee who has been exposed and/or infected has been seriously lacking.

Our colleagues, health-care workers, have presumably joined our profession because of concern for patients and clients. Furthermore, one might suspect that they might be somewhat more sophisticated than the population at large in matters of their own health. We should think that they would deserve, expect, and demand “state-of-the-art” medical care! The only unambiguously current “state-of-the-art” of TB prevention for documented new TB infection is isoniazid preventive therapy. Schemas like those of Cohen et al may be effective as far as they go but no schema can completely eliminate new TB infection in health-care workers.

Recently Sorresso et al and Gonzalez-Rothi depicted the shocking lack of commitment of the medical profession to isoniazid preventive therapy in high risk new infections or even in themselves.

Until we all accept and promote the fact that the control of TB consists of not only identifying, isolating, and treating the infectious case, but also very specific behavior relative to any newly exposed and infected individual (agreeing to take and then taking a full course of preventive therapy), we cannot ever hope to make a dent in TB, and TB will continue to strike fear and hysteria in the health-care worker and his or her community.

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REFERENCES

Tidal Volume, PEEP, and Barotrauma

An Open and Shut Case?

A remarkably consistent scientific literature indicates the potential for certain patterns of mechanical ventilation used in clinical practice to induce or extend lung injury. In managing ARDS, attention has appropriately been concentrated on avoiding inflation pressures and tidal volumes that overdistend the reduced number of fragile alveoli that remain functional. Although we are gradually approaching consensus regarding the need to limit peak alveolar pressure, there is less agreement on how positive end-expiratory pressure (PEEP) should be adjusted. When selecting combinations of PEEP and tidal volume (VT), a growing number of physicians are now shifting priority from optimizing oxygen exchange to assuring adequate lung protection. The machine settings that achieve these goals may not invariably coincide.

Our current focus on avoiding high alveolar pressure, while clearly well directed, may be too narrow; in the first stages of ARDS both end-expiratory and plateau pressures appear to need close regulation. Ventilation at low volumes may extend lung injury, even when high peak alveolar pressures and large tidal volumes are avoided. The exact cause for this “low end” damage remains uncertain, but persistent collapse and/or tidal opening and reclosure of injured tissue are thought to contribute.

In the heterogeneous microenvironment of acute lung injury, regional mechanics differ widely. Because maximal shearing (tangential) forces experienced by an individual alveolus are a nonlinear function of the disparity between its own volume and those of contiguous alveoli, high tidal volumes amplify stresses at boundaries of closed and open lung units. Theoretically, fully
open, flooded, or debris-filled alveoli should experience much less structural tension than atelectatic ones juxtaposed to units inflated by high pressure. Simple geometric arguments suggest that collapsed alveoli surrounded by others distended by only 25 to 35 cm H2O might experience forces known to disrupt capillaries in rabbits and dogs.6-8 Similar stresses could also damage the airway and alveolar epithelium or evoke mediator release. In addition, repeated alveolar opening and closure could deplete functional surfactant, further increasing mechanical stresses and encouraging alveolar edema. Persistent atelectasis per se might also prove damaging, for similar reasons. This would help explain the failure of high frequency oscillation at modest PEEP to prevent barotrauma in neonates with respiratory distress.7

In the setting of ARDS, even limited collapse may be worrisome, judging from the relatively small volumes that correspond to PEEP levels that avert widespread ventilator-induced lung damage in the animal laboratory.8-10 Near-total reversal of atelectasis may partially account for the reported success of a large Vt/high PEEP approach in adult trauma victims.10 It follows that minimizing alveolar collapse in the earliest stage of ARDS (when proteinaceous edema and atelectasis are most prevalent), avoiding high transalveolar pressures, and preventing recurrent tidal opening and reclosure of terminal lung units are all essential elements of a lung protective strategy.

Given the marked differences in respiratory mechanics among patients with ARDS and the wide variation and dynamism of this syndrome, the machine settings best aligned with lung protection cannot be prespecified. Reflecting these concerns, many investigators—but relatively few clinicians—inscribe the entire static pressure volume curve of the respiratory system, so as to determine empirically the tidal pressure range that avoids the low compliance regions associated with collapse and overdistension. The best technique for constructing pressure volume curves, and indeed their precise relevance to tidal ventilation cycles, are not agreed upon. A lower infection zone cannot always be identified, and when found, “Fxpl” to some degree may overestimate the least PEEP level actually required to maintain alveolar stability. Any such difference parallels the size of the Vt and the temporal proximity to a sustained recruitment maneuver. As a practical compromise, many practitioners rely on tidal compliance to guide PEEP and Vt selections. Yet, as reemphasized by the data of Cereda and colleagues appearing in this issue (see page 490), standard calculations of tidal compliance do not provide quite enough information.

More than 20 years ago, Suter and colleagues11 demonstrated that tidal (or chord) compliance, the quotient of Vt and the difference between static pressures at the extremes of the tidal cycle, is jointly determined by PEEP and Vt. High tidal volumes used in conjunction with low end-expiratory pressures can yield tidal compliance values that exceed those of a lower Vt/higher PEEP combination. Judging from acute animal experiments, however, the former pattern may inflict greater damage.9 Under low PEEP conditions, applying a higher Vt undoubtedly recruits lung units during the initial inflation cycles (tidal compliance improves). But a key question concerns the fate of these newly recruited and marginally stable units: continued patency, tidal recollapse, or eventual derecruitment? The answer, as implied indirectly by Cereda’s data showing time-dependent deterioration of compliance, may depend in part on the level of coexisting PEEP and the period of observation.

Despite some suggestive recent evidence12, the extent to which repeated opening and closure actually occurs within each tidal cycle is still controversial. That tidal recruitment might occur in at least a portion of the lung is highly plausible; dependent alveoli are exposed to lower end-expiratory transmural forces, owing to the increased vertical gradients of pleural and interstitial pressure that characterize the edematous lung. Therefore, in the setting of ARDS, alveoli at the top of the recumbent lung tend to remain patent whatever the ventilatory pattern may be, whereas those at the bottom may require terminal airway pressures of 15 to 20 cm H2O to open and only modestly lower end-expiratory pressures to stabilize.12,13 Consequently, as transmural pressures build during inflation, alveolar recruitment may occur in the most dependent sectors. In large animal models of ventilator-induced lung injury, histologic damage concentrates in dependent regions, where the tendency for persistent collapse or phasic tidal opening and reclosure is greatest.14 The prone position lessens the gradient of pleural pressure, reduces the tendency for dependent atelectasis, and attenuates regional damage.15

End-inspiratory plateau pressures of only 25 cm H2O often infringe upon the upper inflection zone of the static pressure-volume curve of adults with ARDS.16 Therefore, the same pressures needed to open and recruit some alveoli can simultaneously overdistend others. There is clear evidence for these opposing effects in Cereda’s study; at a fixed Vt, higher levels of PEEP prevented deterioration of compliance, suggesting recruitment of some lung units), even as they reduced its absolute value (indicating overdistention of many others). Whether PEEP simply adds to the end-expiratory volume of units that are already open or maintains patency of unstable alveoli depends on the patient, the stage of disease, and the vertical position of the alveolus within the injured lung.

Once initiated, alveolar opening occurs abruptly. However, the threshold opening pressure must be
applied for sufficient time. Patency may often be preserved by pressures that are lower than those that opened the unit, depending on the type and stage of lung injury. Relatively high stretching pressures applied for 20 to 30 s improve lung compliance for extended periods in surfactant-depleted small animals and in babies with the infant respiratory distress syndrome. However, the value of such recruitment maneuvers in large animals or adults has yet to be convincingly shown. Lasting benefit would seem less likely in animals with deep chest dimensions, as dependent airways are vulnerable to immediate collapse under the weight of the overlying edematous lung. Pressure-volume hysteresis is often difficult to demonstrate only a few days after disease onset.

It is interesting, therefore, that the data provided by Cereda et al demonstrate that pressure controlled ventilation (as opposed to constant flow, volume cycled ventilation) transiently improved tidal compliance and CO₂ elimination efficiency. One possible explanation is that the decelerating flow waveform of pressure controlled ventilation established a higher lung volume earlier in the tidal cycle, exceeding the threshold opening pressure in marginal lung units for the requisite duration—a recruitment effect. Without sufficient PEEP, this benefit gradually dissipated. Sustained higher pressure during volume cycled ventilation could explain the tendency for increased PEEP to eliminate the relative advantage of pressure controlled ventilation.

After nearly 30 years of investigation and clinical experience, certain fundamental questions in the ventilatory management of ARDS still remain unanswered. Physiological insights from simple yet thought-provoking bedside observations such as these should help to eventually provide them.

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REFERENCES
8 Webb H, Tierney D. Experimental pulmonary edema due to intermittent positive pressure ventilation with high inflation pressures: protection by positive end-expiratory pressure. Am Rev Respir Dis 1974; 110:556-65