

CAN IGNORING AIRWAY SECRETIONS AND REPOSITIONING LIMIT LUNG INJURY?

by *John Marini MD*

In respiratory care and nursing circles, benefits from frequent repositioning of the bedridden patient and actively clearing fluids and secretions in the lower airway are seldom challenged. When clearance of retained mucus is needed, I couldn't agree more.

However, my own clinical experience suggests that very early in the course of non-symmetrical lung injury and pneumonia — when fluids are more freely mobile — this “stirring up” might actually help spread inflammation and even transform limited disease into full-blown acute respiratory distress syndrome (ARDS).

Unintended Spread of Initially Localized Injury

Current teaching holds that diffuse lung injury (acute lung injury or ARDS) begins synchronously throughout the lung, mediated by inhalation, massive aspiration or indirect injury from blood-borne leukocytes, inflammatory mediators, immune complexes or bacteria. In most instances, this perception is undoubtedly justified. But relatively little attention has been paid to the possibility that inflammatory injury may begin focally and propagate via the airways, unintentionally aided by the well meaning nurse, physician or respiratory care practitioner.

We do know that poorly chosen ventilatory patterns — those involving high tidal volumes, high plateau pressures and low levels of PEEP — exacerbate injury. Research into the damage caused by mechanical ventilation (ventilator-induced lung injury) has been focused largely on the primacy of repeated stresses and strains encountered by the lung parenchyma and unsupported small airways during tidal breathing.

Any edema that forms as a result of the primary injury or VILI, however, might also play an important and perhaps preventable role in determining the extent and distribution of damage. The acutely injured lung is comprised of units of two types, functional and damaged. Our thought is that, if uncontained, inflammatory edema may transform some of the former into the latter, thereby extending (“propagating”) trouble.

Apart from any direct inflammation caused by the mediator-rich fluid, inactivation of surfactant might result from its exposure to the proteinaceous edema that forms after breakdown of the alveolar-capillary barrier. Resulting alveolar instability predisposes to creation of high-stress foci and to ventilator-induced lung injury. Preventing transfer of noxious fluids to well-functioning lung units should help to preserve gas exchange and limit the injury process.

Because of its compartmentalized nature, the lung is well set up to confine regional damage to isolated lobes or segments. Furthermore, the circular array of segmental openings ensures that, at any given time, the bronchial channels to some lung sectors are less gravitationally dependent than others, whatever the spatial orientation of the lung might be. Such geometry would offer advantages when attempting to confine potentially damaging bio-fluids to their sites of origin and thereby impedes propagation of an initially regionalized injury.

The plausibility of this teleological argument is suggested by the pioneering experiments undertaken in the pre-antibiotic era to better understand the spread of bacterial pneumonia. Those lessons, now almost forgotten by modern practice, may hold implications for limiting all forms of ALI.

Impressive studies conducted 70 years ago demonstrated the potential for thin secretions to incite new infection in dependent lobes gravitationally predisposed to receive them. That remarkable work, reported in a purely descriptive way by Hamburger and Robertson at the University of Chicago, was undertaken in spontaneously breathing dogs with the intent of determining how multi-lobe pneumonia evolved from uni-lobe infection.

At the time, prevention of spread was a crucial issue. Without antibiotics or life support, double pneumonia was as dreaded as a highly morbid development. The multi-lobe pneumonia that often followed local inoculation of a main bronchus implied spread from the original point of inoculation.

John Marini will be a featured speaker at the 9th annual Focus Conference May 14-16, 2009 Disney's Coronado Springs Resort Orlando, Florida



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Pneumonia occurred selectively in dependent zones and was "wet" (edematous), not consolidated or mucus-like.

Interestingly, lobar pneumonia could not be induced via the bloodstream alone, even when plugging gels of sterile culture media were instilled into the bronchi. Roles for tracheal intubation and ventilation patterns were not considered or explored in those early investigations, as this work predated the mechanical ventilation era, and the syndrome of ARDS was waiting to be described three decades later.

Position is the primary driver of peripheral edema migration. However, the potential for adverse patterns of mechanical ventilation to rapidly transfer thin proteinaceous fluids mouthward was shown very recently in a series of elegant small animal studies using radio-labeled albumin.

Rats allowed to breathe spontaneously (shallowly) and those ventilated with low tidal volumes experienced less propagation of bacteria and radio-labeled fluid between lungs. Albumin dispersed quickly and evenly throughout the lung that contained the injected lobe, as well as propagated into the opposite lung soon after large tidal volumes were initiated from low PEEP.

In contrast, the tracer remained within the injected lung without detectable spreading when higher PEEP was used with smaller tidal excursions. By expanding the local airspace reservoir capacity and by favoring interstitial storage of sieved alveolar liquid, high PEEP

helps keep proteinaceous fluids marginalized and out of the conduits that link airspaces.

For intubated patients, large tidal breaths powered by enhanced expiratory recoil represent a form of ineffective coughing or "huffing" but without the possibility of expelling secretions from the airway. The higher the ventilation requirement, the larger the tidal breath and the more likely it is to be accompanied by forceful exhalation efforts. This may even drive the respiratory system below resting lung volume.

Conversely, the rapid inspiratory peak flows typically selected for flow-controlled, volume-cycled ventilation inhibit mouthward fluid migration. Using a two-compartment test lung system with different compliances, Marcia Volpe (working in my group) demonstrated that high tidal volumes and low PEEP moved a mucus simulant from one ventilated test lung compartment to another across their shared carinal divider.

Intervention Timing

With the risk of propagation in mind, it is noteworthy that pleurisy in humans is often the initial symptom of pneumonia. This prompts the patient to limit physical activity, to draw shallow breaths, to inhibit vigorous coughing, and to avoid stretching the injured pulmonary tissue by lying with the affected side in the lateral decubitus position. In an earlier age that preceded modern medicine, such innate propagation-inhibiting instincts may have offered survival advantages.

By limiting PEEP, allowing high tidal volumes, encouraging cough and aggressively hydrating and turning the patient when edema is forming, modern management may be on the wrong track. In many cases, such airway propagation mechanisms may better explain the transformation or "blossoming" of infiltrates from segmental/lobar pneumonia to ARDS within the first 24 to 48 hours of hospitalization. In fact, most such patients are neither in acute heart failure nor massively fluid overloaded.

If intra-airway propagation is a genuine hazard, timing of PEEP application and positioning may be of under-recognized importance for reasons unrelated to atelectrauma. ARDS generally progresses from an edematous to an organizing phase over a few days. In pneumonia, leukocyte influx and fibrinaceous exudate congeal airway fluids over a two- to three-day period, rendering them hostile to bacterial viability.

After that initial period, the problem turns from one of confining highly mobile fluids to one of unclogging airways and clearing inflammatory debris. Minimized PEEP and dynamic maneuvers intended to improve secretion clearance (positioning, deep breathing, and coughing) are then quite rational. Re-enter the respiratory therapy team.

Bottom Lines

What are the key elements of a lung protective ventilation strategy targeted to the specific aspect of propagation prevention? In the earliest stage of lung inflammation, these would include: relatively high PEEP, small tidal volumes, minimized minute ventilation (to avoid larger tidal volumes and expulsive expiratory efforts), avoidance of fluid excess and edema, dependent orien-

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tation of the most involved segments or lobes, and silencing vigorous expiratory effort.

Early intervention is vital in trying to limit trans-airway propagation of noxious bio-fluids during their high mobility phase. Later on, when gelling has occurred, the positioning priority shifts to expulsion of the thickened secretions, which otherwise act to plug airways rather than to propagate disease. With fluids relatively immobile or neutralized, PEEP gradually loses its value in preventing airway flooding.

While there is little doubt that certain postural reorientations encourage airway drainage and should be strongly considered when it is safe, it is sobering to think that side-to-side repositioning undertaken from the first hours of care to prevent skin breakdown might actually help to distribute noxious lung fluids more widely into previously unaffected zones before gelling inhibits secretion mobility.

Moreover, bacteria contained in situ, are neutralized over time by immune defenses and/or antibiotics. After the first few days, clearance of thickened mucus — rather than prevention of spread — assumes therapeutic primacy.

What, then, are the bottom lines of VILI avoidance and propagation prevention? If disease is already diffusely distributed, the cat may be already out of the bag with regard to preventing propagation. But if not, reasonable measures should be taken to keep it contained. Although some airway suctioning may be unavoidable, aggressive respiratory therapy and repositioning are appropriate only after the injury process has matured and sputum has formed. This is not within the first 24 to 48 hours, during which a keeping the injured portion of the lung quiet and dependent is of utmost importance.

When injury is fresh and initially localized, the potential for airway propagation re-enforces the justification for dependent positioning of the affected zone until secretions are no longer mobile. Furthermore, in the earliest phase of ALI and lobar pneumonia, it is rational to employ a recruiting strategy and generous PEEP so as to expand the local reservoir for fluid within the interstitium and PEEP-distended airspaces.

Avoiding high plateau pressures and vigorous expiratory efforts also makes good sense in the early phase of illness, as these measures not only reduce tissue strain but also lessen the difference between alveolar and airway outlet pressures. This attenuates the expiratory driver of fluid translocation toward the main carina.

The rapidity with which spreading is detected after initiating an adverse ventilating pattern strongly suggests that vigilance to avoid high tidal volumes and release of PEEP is prudent from the very onset of ventilatory support. To these already accepted components of the protective ventilatory prescription would be added the advice to:


- reduce minute ventilation requirements
- minimize large volume inflations
- use a conservative fluid administration strategy
- pay close attention to positional orientation, especially in patients with highly asymmetrical disease.

Prone during this period of mobile edema might be hazardous. I must close by re-emphasizing that these speculations require careful research to confirm or disprove their value.

Dr. Marini, Professor of Medicine at the Univ. of Minnesota, is a clinician-scientist whose investigative work has concentrated in the cardiopulmonary physiology and management of acute respiratory failure.

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