

THE "SOMETIMES SAFETY" OF PRESSURE CONTROLLED VENTILATION *by John Marini MD*



In the delivery of a ventilator-aided breath, the laws of physics dictate that either pressure or flow can be directly regulated—but not both at the same time. Flow can be set and the resulting airway pressure observed or pressure can be regulated as flow becomes the passive variable. Pressure controlled ventilation (PCV) is a form of time cycled ventilatory support in which a constant pressure is applied to the airway opening, independent of the respiratory impedance or patient effort. Enthusiasm for this mode of ventilation has grown steadily over the past two decades, due in part to offer unrestricted flow, whatever the breathing effort might be. In the setting of ARDS, it has another compelling feature: the capacity to limit airway pressure exposure in delicate alveoli vulnerable to iatrogenic damage. For some, pressure controlled ventilation has become an entrenched component of "lung protective ventilation", as most of the emphasis in the published literature of ventilator induced lung injury (VILI) has been placed on capping plateau airway pressure, a value which can be carefully regulated during PCV. This viewpoint—although defensible during controlled mechanical ventilation, becomes more questionable when the ventilator cycle is patient triggered.

VILI is a complex phenomenon whose expression relates not simply to plateau pressure but to transpulmonary pressure as well as to numerous other ventilatory and nonventilatory factors. A common point of agreement among investigators is that excessive tissue strain either produces structural damage directly or signals the initiation of inflammation through more intricate molecular pathways.

Tissue strain, in turn, is a function of stress (crudely related to transpulmonary or distending pressure) and the freedom of the alveolus or small airway to expand. Thus, those lung units poised at the junction of open and closed tissues are most vulnerable to damage when exposed to high inflation pressure. The application of sufficient PEEP after recruitment often eliminates some of these high risk junctional interfaces. Other VILI cofactors include breathing frequency, microvascular pressures, body position, temperature, hypercapnia, the rate of change of alveolar pressure, and duration with which maximum strain is sustained.

Traditionally, flow controlled volume cycled ventilation—commonly known as volume controlled ventilation (VCV)—is applied with constant "square" flow profile, whereas PCV inherently delivers flow in a decelerating pattern. For an equivalent tidal volume and inspiratory time, peak flow will be higher during PCV than during square wave VCV. Moreover, once airway and alveolar pressures have come to equilibrium, flow stops during the inspiratory period thereby sustaining maximum alveolar stress. Because both the rate of change of alveolar pressure as well as the duration of application of maximum tidal stress accentuate VILI, there should be some concern that PCV has an inherent potential for increasing shear stress and stretch. The concern does not stop there. The clinician may effectively restrict airway pressure, but transpulmonary pressure is determined not only by the plateau pressure but also by the pleural pressure that surrounds the lung. The difference between these two values at end inspiration defines the maximal stress applied to the damaged tissue.

During passive ventilation, the clinician may be relatively confident that PCV protects the lung when the plateau pressure is held within acceptable limits (less than 25 to 30 cm of water under most circumstances). Actively triggered ventilation is a different story. Gentle inspiratory efforts that continue throughout the ventilator's inspiratory cycle may increase transpulmonary pressure—and tidal volume—to some degree. Vigorous inspiratory efforts that are sustained until alveolar pressure equilibrates with airway pressure may cause transpulmonary pressures to increase to dangerous levels. It seems ironic that flow controlled ventilation may guard against transpulmonary pressure as well or better than Pressure Controlled Ventilation under these active breathing conditions. Some indication of the risk of vigorous spontaneous efforts during pressure targeted ventilation can be attained by comparing plateau pressure before and after silencing the respiratory muscles while maintaining an equivalent tidal volume. Another more direct method to estimate transpulmonary pressure would be to measure end-inspiratory esophageal pressure and then subtract that value from the plateau pressure. The caregiver should not lose sight of the fact that experimental studies have documented the detrimental consequences of excessive pulmonary vascular pressure and blood flow when airway stresses are high. An increased number of ventilatory cycles per minute also has a damaging effect under these conditions. It stands to reason therefore, that reducing the ventilatory demand (which will reduce both minute ventilation requirement and the demand for cardiac output) could be an inherently "lung protective" stratagem. This reduction may be accomplished in many ways, ranging from assur-

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criteria have been selected under this procurement. No DME MACs will perform pre-pay or post-pay medical review or benefit integrity work. As a result of this reduction in the business functions to be performed by the DME MACs, the costs of the MAC contracts for the DME workload are reduced from those of the current DME Regional Carriers.

Does the proposed bid evaluation process set forth any criteria whereby DHHS may not award a contract to a DME provider?

Yes, DHHS may not award a contract to any DME provider unless the entity meets applicable quality and financial standards. The total amounts paid to contractors within a competitive acquisition area must also be less than the total amounts that would otherwise be paid; and access to multiple suppliers is maintained.

What types of contracts have been awarded to the DME MACs?

The DME MACs were awarded under 'cost plus award fee' contracts. The period of performance for each of the four contracts is one base year with four 1-year options. The contracts have a potential value of \$524 million over the 5-year period.

Which companies have been awarded contracts for DME?

On January 6, 2006, CMS announced the four Medicare Administrative Contractors who are designated as specialty contractors to handle the administration of Medicare claims for suppliers of DME. The four DME MAC companies are:

- National Heritage Insurance AdminaStar Federal, Inc. has been awarded the DME MAC contract for Jurisdiction A, which includes CT, DE, DC, ME, MD, MA, NH, NJ, NY, PA, RI, VT.
- AdminaStar Federal, Inc. has been awarded the DME MAC contract for Jurisdiction B, which includes the states of IL, IN, KY, MI, MN, OH, and WI.
- Palmetto GBA, LLC has been awarded the DME MAC contract for Jurisdiction C, which includes the states of AL, AR, CO, FL, GA, LA, MS, NM, NC, OK, Puerto Rico, SC, TN, TX, U.S. Virgin Islands, VA, and WV.
- Noridian Administrative Services Company has been awarded the DME MAC contract for Jurisdiction D, which includes the states and territories of AK, American Samoa, AZ, CA, Guam, HI, ID, IA, KS, MO, MT, NE, NV, ND, Northern Mariana Islands, OR, SD, UT, WA, and WY.

How will the competitive bidding program impact your Sleep Lab and DME Provider?

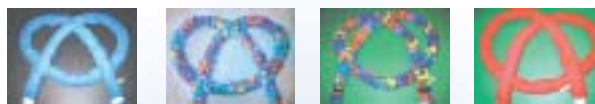
DME suppliers who provide DME, including CPAP, to Medicare patients will be required to participate in the competitive bidding program. DME providers will also have to comply with certain quality standards and become accredited by a CMS designated organization.

What is the overall volume of DME claims and benefit payout?

The four current DME Regional Carriers processed over 68 million claims in fiscal year 2004 from suppliers of DME, orthotics and prosthetics that amounted to Medicare program benefit payouts in excess of \$9.1 billion.

For more information readers can reference the Centers for Medicare and Medicaid's DME Center website section at <http://www.cms.hhs.gov/center/dme.asp>.

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The "Sometimes Safety"... *Continued from page 54*

ing comfort to correcting derangements of metabolic and fluid balance. When other measures fail, deep sedation and even pharmacologic paralysis may be initiated.

Two other characteristics of PCV that may have an adverse bearing on the problem of VILI are the rapidity with which alveolar pressure rises (the so-called dP/dt) which accentuates shearing stresses at the boundary of closed and open tissue at the very onset of inspiration and a plateau pressure that is sustained by an inappropriately long inspiratory time. In theory, pressure support ventilation with the same targeted airway pressure could obviate the latter. Adjustment of the "attack" rate would help to moderate the proto-inspiratory shearing stresses. Even during passive controlled mechanical ventilation, a pressure that gently rises under the influence of flow controlled VCV applied with constant flow may paradoxically present a lower VILI risk when compared to either of the pressure targeted modes.

In the daily care of patients with complex critical illness we often learn that simple rules must bend. In today's practice, the development of management protocols for the bedside demands that rules be set and apply to all conditions. I quite understand this. However although it is appealing to simplify, there is inherent danger in making the same choices for everyone, ignoring the underlying physiology and requirements of the situation at hand. Such is the case with PCV. The respiratory care practitioner must carefully assess the vigor of breathing as well as the characteristics of the individual tidal cycle that relate to plateau pressure, attack rate, and end inspiratory flow before endorsing the initiation or continued use of this mode in the critically ill patient with acute lung injury or ARDS. Depending on the specific circumstances, PCV may prove safer or more hazardous than its traditional VCV alternative.