



MINUTE VENTILATION - THE NEGLECTED PARAMETER

by John Marini MD

In most recent discussions of mechanical ventilation for acute respiratory failure, attention has focused almost exclusively on PEEP and tidal volume. In the context of ventilator-induced lung injury (VILI), little mention or concern is expressed for minute ventilation (VE), the product of frequency and tidal volume. Although laboratory experiments have warned that higher breathing frequencies are damaging when the lung repeatedly undergoes stressful tidal ventilation cycles, the linked roles of frequency and minute ventilation in the generation of VILI are generally downplayed. In fact, minute ventilation could be considered the Rodney Dangerfield of the monitoring spectrum—it gets little or no respect. In my view, however, there are many good reasons to pay attention to VE, as it plays important roles as a physiologic indicator and should be considered a key target for lung protection.

The interest value of Minute Volume is not limited to its role in Ventilator Induced Lung Injury - far from it

The interest value of VE is not limited to its role in VILI - far from it. Increased minute ventilation causes several things to happen that may not be physiologically desirable. If ventilation is spontaneous, the work of breathing rises exponentially (as a quadratic)

as the need for ventilation increases, often overtaxing weakened respiratory muscles. Associated oxygen consumption obligates an increase of cardiac output that strains the capacity of a heart that is ischemic, failing or in overt shock. Moreover, hyperpnea activates the chest-compressing expiratory muscles, which may compromise resting end-expiratory lung volume (FRC). Relative hypoxemia then results as the diaphragm is pushed higher into the chest at end-expiration by the rising abdominal pressure. These adverse effects on oxygenation can be minimized or avoided entirely by deep sedation or paralysis, establishing passive inflation. When ventilation is machine-controlled, however, mean airway pressure rises as a direct function of VE. This increase in mean airway pressure (which reflects a similar increase in alveolar pressure) may help improve oxygenation but also simultaneously raises the average intra-pleural pressure, impedes venous return and may reduce cardiac output.

When the lungs are passively inflated, the reasons that mean airway pressure must rise in parallel with VE are two-fold: First, because expiration is driven only by passive recoil of the respiratory system, average lung volume must rise as minute ventilation and mean expiratory flow do; Second, auto-PEEP may be gener-

ated by reduced expiratory time. As most respiratory professionals realize, auto-PEEP is a function not only of the expiratory resistance, but also of minute ventilation. In fact, experienced practitioners know that reducing the minute ventilation requirement—even accepting hypercapnia in the bargain—is the surest and most powerful tool we have to reduce the auto-PEEP effect and its consequences.

While an increasing VE is usually accompanied by the need to keep PaCO₂ appropriate to the physiologic needs of the patient, hyperventilation—signified by an inappropriately low PaCO₂ and an elevated pH, impairs release of oxygen from hemoglobin at the tissue level. The well known tingling, cramping, chest tightness and lightheadedness associated with psychogenic hyperventilation are manifestations of alkalosis-induced neurological dysfunction. The myocardium and conduction pathways of the heart are also affected by alkalosis, predisposing to arrhythmias.

So much for the consequences of VE ...What causes VE to rise? Answering this question provides vital clues to crisis intervention and improved ventilation management. In general terms, the VE is set by three factors: metabolic need, the lung's gas exchanging efficiency, and the set point of PaCO₂, as determined by the patient's drive to breathe or the clinician's own manipulations of the ventilator. Therefore, when faced with a high VE, it is important to seek the reason along all three axes. Fever, agitation, and pain each elevate oxygen consumption and CO₂ production. Metabolic acidosis, usually caused in the critical care setting by renal insufficiency, anerobic metabolism, ketoacidosis or even injudicious administration of chloride, obligates increased ventilation to keep pH closer to normal. It is not unusual to encounter patients who are bicarbonate depleted due to gastrointestinal losses, renal disease, or prior treatment with excessive chloride (in the form of "normal" or "physiologic" saline). 0.9% saline solution is not normal, in that it infuses a concentration of chloride ion more than 33% higher than normal plasma. Preserving electroneutrality mandates that bicarbonate concentration falls to compensate. Provided that the sodium load can be dealt with, administration of bicarbonate to such patients with chloride excess may allow the PaCO₂ to rise and the pH-mandated ventilation requirement to fall. Reducing VE in this way yields considerable benefit, as the work of breathing drops disproportionately.

Along the second axis, increased alveolar dead space, commonly due to acute lung injury, volume overload, obstruc-



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Neonatal Tidal Volume... Continued from page 66

ed 4 sensors, the average inspiratory VT error rate = -6%, and the average expiratory VT error rate = - 1%.

This story has a couple of morals. One is that the kind of instrumentation and expertise required to do this kind of testing is really not very complex and well within the wheelhouse of many RT departments. We used a simple static test lung which had a known compliance. The ventilator measures airway pressure. As long as you have the inspiratory time long enough for a complete pressure equilibration you can calculate the actual tidal volume very simply and compare this to the displayed tidal volumes produced by the ventilator. Test the equipment you are going to buy. Test it thoroughly. Trust your vendors, but verify, to coin a phrase.

The second moral is that some companies are very responsive to persuasive, data driven arguments. Perhaps some are not. This would be something to think about when making large capital purchase decisions. Our duty as the neonatal RT community is to do this kind of performance verification testing and push, cajole, persuade and otherwise convince the manufacturers to make changes when the performance data and the potential clinical impact warrant an immediate change (not in the next generation of ventilators). We will be presenting more of these data at the International Respiratory Congress in Anaheim and should be publishing a full manuscript on the results of our testing within the six months.

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Minute Ventilation... Continued from page 72

tive lung disease, hypovolemia, excess PEEP or pulmonary arterial occlusion, impairs the lung's CO₂-exchanging efficiency. Volume-based capnometry of the exhaled gas stream helps assess not only the CO₂ production, but also the wasted fraction of ventilation (dead-space). Finally, along the third axis, the CO₂ set point must be assessed by blood gas analysis. Although always offset from the corresponding arterial values, venous blood often serves as well as arterial blood for tracking PaCO₂ and pH.

Can a rising VE actually contribute to ventilator-induced lung injury? Experimental evidence indicates that there are definite reasons to think so. For example, hypercapnia has a protective and generally beneficial effect on inflammation, and this potential benefit may be lost when VE elevates to keep pH normal. On a purely mechanical level, it has been clearly shown that reducing the application frequency of high pressure tidal cycles can protect the lung, even when the same plateau pressure, mean airway pressure and PEEP are maintained. (When the tidal cycles are not mechanically stressful, no level of frequency or minute ventilation encountered in clinical practice is likely to result in lung injury.) Finally, increasing minute ventilation without lengthening the I:E ratio requires an increase of mean (and usually peak) inspiratory flow. Although the experimental literature supporting a tissue-damaging role for high inspiratory flow is not yet conclusive, higher flow rates clearly amplify the shearing forces encountered by the airways and micro-vessels, with the potential to damage tissues within a heterogeneously injured lung.

With such a broad range of associated causes and effects, it should come as no surprise that tracking VE is a valued means by which to monitor the ventilated patient. In the throes of acute illness, changes of this key indicator warn of metabolic, psychological, or pathological disturbances. Later in the course of the illness, minute ventilation may provide information that facilitates the often difficult decision to extubate. I have found that when judging the ability of a patient to breathe spontaneously, the f/VT ratio should not be interpreted without reference to the simultaneous change of VE. While a rising f/VT ratio may indicate distress if VE falls, it may simply reflect an exercise-like response if VE rises in parallel with the rapid shallow breathing index. If VE is relatively high but has shown a wide range of variation in the preceding 24 hour period, the cause may relate more to psychological than to physiological factors, in other words, the patient is likely to have good ventilatory reserve.

Alterations of minute ventilation are rooted in underlying clinical physiology and offer important clues to pathogenesis and treatment. As these foregoing examples illustrate, ignoring fluctuations of VE is ill-advised, as doing so degrades the practitioner's ability to make well-reasoned and physiologically-grounded judgments for managing the ventilated patient with critical illness. The characteristics of the individual breathing cycle--tidal volume, plateau pressure and PEEP--are of unquestioned clinical importance and currently occupy center stage in the ventilation of acute respiratory failure. But for reasons that span a wide range of issues, it is unwise to overlook the vital VE parameter!

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