



DO NOT PUMP UP THE VOLUME: NEONATAL TIDAL VOLUME ACCURACY REDUX

by John Salyer RRT-NPS, MBA, FAARC

In an earlier column I shared you my ruminations about the importance of measuring and limiting tidal volume during neonatal mechanical ventilation. Anyone who has paid even the slightest attention over the last few years now knows that the jury is in. Pressure is not the primary suspect perpetrating mechanical lung injury. Volume has confessed to the crime. Pressure is just the get away driver. The best chance you have of limiting ventilator induced mechanical lung injury in neonates is by limiting tidal volume to 5-7 mL/Kg. There are some clinicians who are even recommending 4-6 mL/Kg in pts < 750 g. This is all fine and dandy, but there can be a fly in this ointment. Reliable accurate tidal volume determination at VT < 10 mL ain't all that reliable in some brands of ventilators.

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It is even tougher when VT < 5 mL. I have had the luxury and pleasure to have worked over the years with some gifted research oriented RT's who help me conducted a series of experiments that studied displayed tidal volume accuracy of several different brands of ventilators

under low tidal volume conditions (Respir Care 2007;52:1573, Respir Care 2005; 50:1498, Respir Care 2005;50:1498, Respir Care 1997;42:1099.) We learned several important things about measuring neonatal tidal volume accurately during mechanical ventilation.

First, we learned that proximal flow sensing was essential for accurate and precise volume display. We tested almost every widely used neonatal ventilator and found that only the brands that used proximal flow sensing produced reliable displayed tidal volume at VT between 5 and 10 mL. This was true even when circuit compliance volume correction was used. The ventilators that used proximal heated wired anemometry generally had accuracy \approx + 10% at VT between 5 and 10 mL. For this and some other reasons we settled on one brand of ventilator for our NICU-PICU-CICU intensive care units (Viasys Avea). But as we continued to push tidal volumes lower, additional limitations in the accuracy of the displayed volumes of the Avea became apparent (which we also found this in other famous neonatal ventilators).

Second, we found large variation in the accuracy of displayed tidal volumes within a group of Aveas when using the same hot wire sensor and a VT < 5 mL. On a test lung with identical settings,

the same circuit and the same sensor, a series of 27 Aveas pulled from our working fleet displayed an average inspiratory tidal volume error of 27 % (range from 13 % to 37 %) and an average expiratory tidal volume error of -17 % (range -4 % to -64 %). Thus the Avea often showed large leak when none was present, when set at very low tidal volumes. We are fairly confident this is true for other brands of ventilators, but have not yet had the opportunity to test another working fleet of other ventilator brands. Not to worry though...that distant smoke on the horizon you see...that is testing results from other brands that we are going to develop.

This led us to our third observations. Inspiratory to expiratory tidal volume measurement errors can be large at very small tidal volumes and can be misinterpreted by the ventilator as airway leaks around the endotracheal tube. This becomes problematic when the pseudo-leak is large enough to start tripping the ventilators alarm system. This is probably true in some other ventilator brands but could be obscured by the fact all other ventilator brands do not display both inhaled and exhaled tidal volume.

It turns out that the displayed inaccuracy of the Avea tidal volumes at VT < 5 mL we discovered were actually within the manufacturers published accuracy specifications, which are, in retrospect, way to liberal for such low tidal volumes.

But don't get discouraged or run out and start trashing one brand of ventilator over another. Remember, that we are pretty sure the magnitudes of the errors we found at VT < 5 mL are present in all neonatal ventilators, we just have not had the opportunity to test as many of them as we have the Avea.

At this point we could have thrown our hands up in the air, simply accepting a lot of variation and inaccuracy at VT < 5 mL (which is what I suspect the clinical community has mostly done for a number of ventilator brands, either consciously or unconsciously). Instead we invited the Viasys engineers to review our data and techniques and we recommended the addition of a zero flow calibration procedure for their hot wire sensors. There were some people who were a bit skeptical at Viasys but eventually they concurred and redesigned their software to include the calibration procedure. Our subsequent testing has shown marked improvement in sensor performance at very low tidal volume. After installation of the new software and zero flow calibration of each hot wires sensor on one Avea, we test-



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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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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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