Volumetric Capnography in Acute Respiratory Distress Syndrome: Is the Era of Day-to-Day Monitoring Finally Here?

Acute lung injury (ALI) and the acute respiratory distress syndrome (ARDS) affect 4.5–7.1% of the patients admitted to the intensive care unit with acute respiratory failure.\(^1,2\) In our population, we found that ARDS affects 15.3 per 100,000 persons per year.\(^3\) Although the reported mortality of patients with ALI and ARDS has improved, it remains high among the causes of death in the intensive care unit. Affected patients usually die secondary to sepsis and multisystem organ dysfunction. Some predictive factors associated with mortality include the triggering disease, the severity of the respiratory illness, and the presence of dysfunction of the right ventricle.\(^4\) Although hypoxemia is the protean gas exchange abnormality in ALI and ARDS (it is part of the definitions),\(^5\) the degree of hypoxemia does not predict outcome.\(^6\) As a matter of fact, patients treated with lower tidal volume (VT) in the ARDS Network trial had a lower ratio of PaO\(_2\) to fraction of inspired oxygen in the first 3 days of therapy but still had a lower mortality and a higher number of ventilator-free days.\(^7\)

In this issue of RESPIRATORY CARE, Kallet et al\(^8\) demonstrate that using volumetric capnography to calculate the physiologic dead space in patients with ARDS gives values that correlate with those obtained by the metabolic-cart method used in their important 2002 paper.\(^9\) Kallet et al showed a statistically strong correlation between the 2 techniques in terms of the expired carbon dioxide tension and physiologic dead-space-to-V\(_T\) ratio, with a fair bias and precision. However, the sample of patients was small and it will be important to independently confirm these exciting findings. These results are important because they simplify the technique of a bedside measurement that can predict survival in patients with ARDS.\(^9\)

Enghoff defined the physiologic dead space in 1938 as the “wasted ventilation.”\(^10\) He modified the Bohr’s equation by replacing the alveolar CO\(_2\) concentration with the PaCO\(_2\), adding to the anatomic dead space the alveolar units that are either completely nonventilated or subject to varying degrees of ventilation-perfusion mismatch. The physiologic dead space is therefore a measure of the efficiency of ventilation. In 2002, Nuckton et al identified the physiologic dead-space fraction measured within several hours of the onset of respiratory failure as the first lung-specific predictor of mortality in ARDS.\(^9\) To calculate the physiologic dead space they used a metabolic monitor, also known as indirect calorimetry cart, that allows the collection of the exhaled V\(_T\) in the cart’s built-in mixing chamber. An infrared censor then measures the expired CO\(_2\) fraction in the collected gas, which in turns allows the calculation of the partial pressure of CO\(_2\) in the exhaled V\(_T\). The technique is accurate if water vapor pressure is well controlled, the system is relatively free of leaks, and the exhaled V\(_T\) is collected for several cycles to average the breath-to-breath variation in CO\(_2\) composition. The bedside metabolic monitor has been validated against the classic method of collecting exhaled gases in a Douglas bag and measuring the CO\(_2\) partial pressure in an arterial blood gas analyzer.\(^11\) The metabolic cart is less cumbersome than the Douglas bag, and it allows a minute-to-minute bedside evaluation.

However, in our experience, the use of the metabolic cart still poses a considerable work load for the intensive care unit staff and may not be readily available, since its use for the assessment of the patient’s nutritional needs and oxygen consumption seems not to be common.

Volumetric capnography, also known as the single-breath test for CO\(_2\), relies on a bedside instrument that displays a plot of the partial pressure of CO\(_2\) (or fractional concentration of CO\(_2\)) in the exhaled gas as a function of the exhaled V\(_T\). It records the partial pressure of CO\(_2\) throughout a tidal breath by acquiring a calibrated signal from an infrared light.\(^12\) The light emits a wavelength near 4.28 mm, at which the maximum absorbance of carbon dioxide occurs. The exhaled V\(_T\) is simultaneously acquired by measuring the expiratory flow that results in the familiar S-shaped CO\(_2\)-V\(_T\) plot. The mean exhaled pressure of CO\(_2\) can be electronically derived and used in the calculation of the physiologic dead space. Alternatively, the arterial partial pressure of CO\(_2\) can be represented on the exhaled CO\(_2\)-V\(_T\) curve as a horizontal line. The rectangular area formed by this line and the exhaled V\(_T\) represents an ideal system, free of dead space or ventilation-perfusion mismatching, where there is complete equilibration between the alveolar and arterial CO\(_2\).
ideal area and the area under the S-shaped curve would then be the physiologic dead space.\textsuperscript{13}

Why is an elevated physiologic dead space associated with an increased mortality in patients with ARDS?\textsuperscript{9} Is it that it identifies a subset of patients with the “worst” degree of lung injury, as does the quasistatic respiratory compliance, another predictor of mortality?\textsuperscript{9} If the physiologic dead space reflects pulmonary vascular injury, does it correlate with the degree of right-ventricular dysfunction, or with levels of Von Willebrand factor, which is a marker of endothelial injury in ALI/ARDS?\textsuperscript{14,15} Do patients with an increased dead-space fraction also have an impaired maximum alveolar-fluid clearance?\textsuperscript{16} All of these are questions that need to be answered in the near future.

The strong statistical agreement between the metabolic cart and the volumetric capnograph implies that the latter can be accurately used to predict mortality in patients with ARDS. A simpler technique to estimate the physiologic dead space in mechanically ventilated patients should lead to important clinical and research applications. We should anticipate trials that monitor the day-to-day severity of lung injury and that potentially follow the response to therapy.

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REFERENCES


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