Uh-oh, some CO₂ has gone missing

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Central Message

Increased dead space ventilation fraction, particularly if computed with a correction for shunt, is likely a prognostic marker for pulmonary morbidity in patients after unifocalization.

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In this issue of the *Journal*, Koth and colleagues¹ analyzed dead space ventilation fraction (Vd/Vt) in a cohort of 265 patients shortly after collateral unifocalization and report an association between increased Vd/Vt (>20%) and prolonged mechanical ventilation. The report is noteworthy—it describes an underappreciated aspect of physiology in a large cohort of patients with tetralogy of Fallot with pulmonary atresia treated at a center that is well-known for excellent outcomes with this group of patients. The authors' general message appeals to intuition—unifocalization may be associated with alveolar collapse, reperfusion injury, underperfused segments, capillary leak, interstitial edema, etc—all contributing to increased dead space and pulmonary morbidity.

The authors used the Bohr–Enghoff approximation based on end-tidal carbon dioxide (CO₂) to compute Vd/Vt. This is a relatively common approach that gained in popularity after the 2007 publication by Severinghaus,² and reports based on this approach have shown a relationship with outcomes in patients undergoing congenital heart surgery.^{3,4} However, we think it is important to appreciate what this measurement reflects.

In healthy lungs, gas exchange can be described by a tripartite model predominated by “ideal” alveoli with minimal ventilation/perfusion heterogeneity, a small fraction of underperfused alveoli (dead space—impacting predominantly CO₂), and minimal unventilated alveoli (shunt—impacting both oxygen and CO₂). Expansion of dead space or shunt regions indicates pathology of gas exchange. Even in simplified tripartite models, dead space is both an elusive concept and measurement. The Bohr dead space requires measurement of mixed exhaled and “ideal” alveolar gas or can be obtained by volumetric (not time-based) capnography. Enghoff's modification substitutes arterial CO₂ tension for the ideal alveolar value but still requires the collection of mixed-expired gas or volumetric capnometry. Complicating the Bohr–Enghoff

method is contamination by “shunt”—the veno-arterial CO₂ difference that increases arterial CO₂ tension above the “ideal” alveolar point.⁵ Moreover, in infants and children, low tidal volume and greater respiratory frequency may limit the accuracy of airway or sidestream-based CO₂ analysis techniques.⁶

For the infant cardiac surgical patient, particularly one just having undergone unifocalization, there are several dynamic factors that may impact the Enghoff-derived approximation that are not directly related to alveolar or physiologic dead space. In our opinion, alveolar shunt is an important contaminant not adequately addressed by categorization according to surgical procedure. There are well-established methods to correct for shunt based on easily obtained measures of arterial oxygenation.^{7,8} With such a correction, we would expect the end-tidal CO₂-derived dead space measures to be a more significant indicator of dead space physiology. Perhaps this is in part a semantic issue—the measurement the authors used may have clinical utility and be associated with various measures of pulmonary morbidity, but it is not necessarily an accurate measure of true dead space fraction in these patients. To the extent it does have clinical utility (regardless of what it is called), what threshold of “Vd/Vt,” in the absence of other clinical findings, would warrant more aggressive diagnostic or therapeutic measures? We look forward to further work and answers from the authors.

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