What Do Dead-Space Measurements Tell Us About the Lung With Acute Respiratory Distress Syndrome?

In this issue of Respiratory Care, Kallet et al² describe the prognostic value of sequential dead-space measurements in patients with acute respiratory distress syndrome (ARDS). The simple calculation using arterial and mixed expired partial pressures of carbon dioxide (the Enghoff modification of the Bohr dead-space equation: physiologic dead-space volume divided by tidal volume [V_D/V_T]) is more convenient to acquire the data for, and simpler than the calculation of shunt or venous admixture. The data from Kallet et al² show that dead space is a useful index of lung dysfunction and a predictor of survival.

However, interpreting the pathophysiology that leads to increased V_D/V_T in an ARDS patient is more complex than the simple concept of dead space that we introduce to students in beginning respiratory physiology. Nuckton et al² measured V_D/V_T during the first 24 hours of ARDS and found that the single initial V_D/V_T measurement outperformed other readily available bedside measurements as a predictor of mortality. However, a high V_D/V_T in ARDS does not primarily reflect increased ventilation to unperfused areas of lung, as Nuckton et al originally suggested. Though there is no question that ARDS is characterized by patchy areas of severe vascular damage, no studies have demonstrated that those heavily damaged areas receive any appreciable ventilation, as would be required for the existence of regions of high alveolar-ventilation-to-perfusion ratio (V_A/Q). In fact, 4 studies that used the multiple inert gas elimination technique (MIGET) with ARDS patients found that less than one quarter of the patients had discrete high-V_A/Q regions that could contribute substantially to elevated V_D/V_T. Nonetheless, Nuckton et al and Kallet et al document that elevated carbon dioxide V_D/V_T is a universal finding with ARDS patients from the onset of the illness. How can this apparent paradox be resolved?

In abnormal lungs an increased V_D/V_T reflects a global assessment of abnormal gas exchange, not simply the contribution of discrete high-V_A/Q regions and true anatomic dead space. Hence, V_D/V_T can be increased by shunt, V_A/Q heterogeneity across the entire spectrum from low to high V_A/Q regions, anemia, and by increased anatomic dead space. Hlastala and Robertson presented an inert gas model that demonstrated that the levels of shunt and mid-range V_A/Q heterogeneity commonly observed in ARDS substantially increase the carbon dioxide V_D/V_T, without high V_A/Q. In human ARDS studies that used MIGET, the primary gas exchange abnormalities were shunt and increased mid-range V_A/Q heterogeneity, with only an inconsistent component of ventilation to high-V_A/Q units. Severe anemia decreases the functional solubility of carbon dioxide in blood and increases the dead-space measurement, albeit by a lesser amount.

To analyze the various components of V_A/Q abnormality that contribute to elevated V_D/V_T, Coffey et al used MIGET to study dogs with acute oleic-acid injury. Various levels of positive end-expiratory pressure (PEEP) were used to alter the fractional contribution of each V_A/Q abnormality (shunt, mid-range V_A/Q heterogeneity, high V_A/Q peaks, and anatomic dead space) to the V_D/V_T. After injury, application of low-level PEEP reduced the overall V_D/V_T, primarily by reducing shunt. At the highest PEEP level, discrete high-V_A/Q regions developed, but that V_D/V_T augmentation was counterbalanced by reduced shunt and mid-range V_A/Q. The net result (reduced V_D/V_T) was relatively constant at all PEEP levels, despite there being different physiologic abnormalities. Likewise, it is plausible that 2 patients with ARDS who have identical V_D/V_T elevations might have...
different physiological components explaining their \( \frac{V_D}{V_T} \) elevation. At this point we can only speculate as to whether knowledge of the physiologic components responsible for the elevated \( \frac{V_D}{V_T} \) in ARDS would provide additional prognostic information.

Kallet et al\(^1\) demonstrate that, with patients who have ARDS, \( \frac{V_D}{V_T} \) is an easily obtained indicator of pulmonary gas exchange efficiency and that \( \frac{V_D}{V_T} \) has good clinical prediction characteristics. The value of \( \frac{V_D}{V_T} \) as a global index of lung dysfunction ought not be surprising, as it incorporates all the mechanisms by which gas exchange abnormalities can manifest in ARDS. Based on the most sophisticated MIGET measurements of gas exchange in patients with ARDS, an increased \( \frac{V_D}{V_T} \) (as calculated with the Bohr-Enghoff equation) usually represents a combination of shunt and mid-range-\( \dot{V_A}/\dot{Q} \) heterogeneity rather than discrete high-\( \dot{V_A}/\dot{Q} \) regions alone. However, regardless of the physiologic interpretation of the elevated \( \frac{V_D}{V_T} \) in a given patient, the clinical prognostic value of the measurement described by Kallet et al stands unchallenged.

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**REFERENCES**