Capnography as a Hemodynamic Indicator: Another Heart Lung Interaction*

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Impaired gas exchange most often results from respiratory disease; however, it may be result from and serve as an indicator of cardiovascular dysfunction, particularly in patients with congenital heart disease. Impaired gas exchange is most often due to the mismatching of ventilation (V) relative to perfusion (Q) (i.e., V/Q mismatch). Alveolar disease creates low V/Q ratios and pulmonary shunt, leading to hypoxemia, whereas limitations of pulmonary perfusion (Qp) create high V/Q ratios and dead space (DS), impairing carbon dioxide (CO_2) clearance, leading to hypercapnia. Although alveolar disease may contribute to a DS, it does so to a limited extent, and only as the shunt fraction rises above 30%. For these reasons, impaired gas exchange and, in particular, DS-induced hypercapnia may serve as an indicator of cardiovascular dysfunction and has been shown to be associated with outcomes in children following cardiac surgery. For these reasons, impaired gas exchange may function as an indicator of cardiovascular dysfunction and has been shown to be associated with outcomes in children following cardiac surgery.

The degree of DS ventilation may be assessed using end-tidal CO_2 monitoring. Based on the work of Bohr: $V_D / V_T = P_ACO_2 - P_ECO_2 / P_ACO_2$, where V_D is physiologic DS, V_T is tidal volume, and P_ECO_2 and P_ACO_2 are mixed expired and mean alveolar CO_2, respectively. Enghoff's modification of Bohr's equation assumes that $Paco_2$ is equal to P_ACO_2. The substitution of $Paco_2$ for P_ACO_2 and its limitations, to be discussed later, and the substitution of end-tidal CO_2 for P_ECO_2, enables us to determine the alveolar DS fraction (AVDSf; $AVDSf = Paco_2 - P_{ET}CO_2 / Paco_2$, where $P_{ET}CO_2$ is end tidal CO_2), because $P_{ET}CO_2$ is more representative of alveolar than airway gas (1).

The AVDSf has been studied extensively in critically ill children and has been shown to be strongly associated with worse outcomes (2, 3). Similar findings have been demonstrated in children following congenital heart surgery for a variety of cardiac lesions, including hypoplastic left heart syndrome, cavopulmonary anastomosis, and with this study, the third and final stage of single-ventricle palliation, the Fontan procedure (4–6). In this issue of *Pediatric Critical Care Medicine*, Shostak et al (7) conducted a retrospective study of patients who underwent the Fontan operation over an 8-year period to determine if there was an association between the AVDSf and clinical outcomes.

The primary outcome composite included one or more of the following: need for postoperative interventional cardiac catheterization, extracorporeal membrane oxygenation support, surgical reintervention, hospital stay greater than or equal to 28 days, ventilation duration greater than or equal to 170 hours, and surgical mortality. The secondary outcome measures were parameters of severity of illness, duration of chest tube drainage, and ICU length of stay. Patients were divided into two groups. One group comprised of the 34 patients who met the composite outcome. The second group of patients ($n = 94$) did not meet the criteria for the composite outcome and served as the control. Patients who received an extracardiac Fontan conduit and who remained intubated after returning from the operating room were included. Twenty-two patients in the composite group and 68 patients in the control group were fenestrated. The AVDSf was significantly higher in the composite group compared with the control group on admission. However, they were not different at 3 and 6 hours following admission. An AVDSf of 0.29 or greater was associated with a 37% increase in risk to meet composite criteria. In addition, the arterial oxygen saturation (Sao_2) was significantly lower in the composite group compared with the control group at all three time points. Using both the admission, AVDSf and Sao_2 values enhanced outcome predictability. There was no significant difference in the AVDSf between fenestrated and nonfenestrated patients. However, the difference in the Sao_2 between the two cohorts was significant. At admission and at 3 hours after admission, the Sao_2 was significantly lower only in the fenestrated patients. There was no significant association between the presence or absence of fenestration and primary

*See also p. e200.

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outcome parameters. The AVDSf was also significantly associated with the secondary outcome parameters.

The findings of the study by Shostak et al (7) demonstrate that impaired gas exchange may be due to pulmonary and/or cardiovascular disease and an understanding of the physiologic underpinning of these mechanisms are essential in the management of critically ill patients. Findings such as these provide important insight into the pathophysiology of disease states. Perhaps the results of the study by Shostak et al (7) as well subsequent studies will establish an AVDSf threshold that will impact treatment strategies by enabling the clinician to implement the right therapies at the right time and in doing so favorably impact the postoperative course and outcomes following the Fontan procedure.

The study by Shostak et al (7) has important limitations to consider. This is a retrospective study with a large proportion of intubated Fontan patients, which may not reflect current practice at the majority of centers. In addition, the Enghoff's modification of Bohr's equation assumes that $Paco_2$ is equal to P_ACO_2; however, with large intrapulmonary shunts (not relevant in these cases) or cardiac shunt (for those fenestrated), the $Paco_2$ will be higher than P_ACO_2, leading to larger calculated AVDSf where a portion of the DS is not due to true physiologic DS but rather due to "wasted perfusion" DS. Oxygenation is a "hemodynamic indicator" only if cardiac shunting is present. Low cardiac output states and the resulting low mixed venous oxygen saturation are readily corrected with minimal supplemental oxygen, unless there is significant pulmonary (unlikely) or cardiac shunting. These concepts are demonstrated in Table 4 in (7) where the Sao_2 is significantly different between the control and composite groups in the fenestrated patients but not in the nonfenestrated patients. These important limitations not withstanding an assessment of the AVDSf provide useful information and should be considered an essential tool in the management of critically ill patients, not only as a measure of impaired gas exchange but as an indicator of cardiovascular dysfunction.

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