Chiumello and Gallazzi *Critical Care* 2010, **14**:195
http://ccforum.com/content/14/5/195

CRITICAL CARE

COMMENTARY

Estimation of dead space fraction can be simplified in the acute respiratory distress syndrome

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See related research by Siddiki *et al*., http://ccforum.com/content/14/4/R141

Abstract

Acute lung injury and acute respiratory distress syndrome are characterized by a non-cardiogenic pulmonary edema responsible for a significant impairment of gas exchange. The pulmonary dead space increase, which is due primarily to an alteration in pulmonary blood flow distribution, is largely responsible for carbon dioxide retention. Previous studies, computing the pulmonary dead space by measuring the expired carbon dioxide and the Enghoff equation, found that the dead space fraction was significantly higher in the non-survivors; it was even an independent risk of death. The computation of the dead space not by measuring the expired carbon dioxide but by applying a rearranged alveolar gas equation that takes into account only the weight, age, height, and temperature of the patient could lead to widespread clinical diffusion of this measurement at the bedside.

In the previous issue of *Critical Care*, Siddiki and colleagues [1] presented the dead space fraction data collected at admission and on day 3 from two acute lung injury/acute respiratory distress syndrome (ALI/ARDS) databases (109 patients in the Mayo Clinic and 1,896 patients in the ARDS Network). The hospital mortality increased in direct proportion to an increase in the dead space fraction. For every 0.05 increment of the dead space fraction, the odds ratios for hospital mortality were 1.07 at day 1 and 1.12 at day 3. Thus, at first sight, the results of Siddiki and colleagues represent merely a repetition of previous studies [2-4]. However, their study added a novel element in that the dead space fraction was computed more simply than in the previous studies [2-4] and, unlike in those studies, without monitoring of the expired carbon dioxide (CO_2). So that the current results may be better understood, a short summary of the theoretical aspects of the dead space computation is presented.

ALI and ARDS are characterized by a non-cardiogenic pulmonary edema with significant impairment of gas exchange. The increases in the right-to-left intrapulmonary shunt and in low ventilation-to-perfusion ratio lead to hypoxemia, whereas the increase in pulmonary dead space reduces CO_2 removal [5,6]. The increase in pulmonary dead space is due mainly to alterations in the distribution of pulmonary blood flow originating from vascular obstruction and to regional overdistension of ventilated alveoli induced by the application of positive end-expiratory pressure (PEEP) and sometimes by the reduction in cardiac output [7-9].

Nuckton and colleagues [10] found that in patients with ARDS the pulmonary dead fraction measured at admission was significantly higher in the non-survivors than in the survivors (0.63 ± 0.09 versus 0.54 ± 0.09); for every increase of 0.05 in the dead space fraction, the odds ratio of death increased by 45%. Subsequent studies showed that the dead space fraction, measured during the first 6 days of mechanical ventilation, was significantly higher in the non-survivors than in the survivors and in patients with ARDS compared with patients with ALI [2-4]. The odds ratios of death were 1.59 and 2.87 in the early and intermediate phases of ARDS, respectively [4].

The original Bohr's equation required the mean alveolar (PA_{CO2}) and mixed expired (PE_{CO2}) partial pressures of CO_2 in order to estimate the physiologic dead space: (PA_{CO2} – PE_{CO2})/PA_{CO2}. Subsequently, given the difficulty of measuring the PA_{CO2} and given that PA_{CO2} and arterial pressure of CO_2 (Pa_{CO2}) are nearly identical in normal subjects, Enghoff suggested that Pa_{CO2} be used instead of PA_{CO2}; consequently, the new equation was (Pa_{CO2} – PE_{CO2})/Pa_{CO2}. Thus, any changes in intrapulmonary shunting, ventilation/perfusion ratio, or CO_2 solubility (Haldane effect) can affect the pulmonary dead space.

Traditionally, the PE_{CO2} has been measured over several minutes by collecting the expired gas (into a Douglas

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bag) and analyzing it with a blood gas analyzer [11]. Nowadays, indirect calorimeters or volumetric capnography allows accurate PE_{CO2} measurements at the bedside [11-13]. Unfortunately, these methods can be cumbersome and thus may limit the widespread measurement of dead space in clinical practice.

In the present study, Siddiki and colleagues [1] 'estimated' the physiologic dead space not by taking into account the PE_{CO2} but by applying a rearranged alveolar gas equation. The dead space was equal to 1 – [(0.86 × $\dot{V}_{CO2}$ estimated)/(minute ventilation × Pa_{CO2}]. The $\dot{V}_{CO2}$ estimated (the estimated CO_2 production) is simply measured by the Harris-Benedict equation, which takes into consideration the weight, height, age, and body temperature of the patient. The Harris-Benedict equation is commonly used to predict the resting energy expenditure (REE) [14].

There are some important limitations to the study by Siddiki and colleagues: (a) the absence of any comparison of the physiologic dead space measurements obtained with the rearranged alveolar gas equation and with the Enghoff equation and (b) the use of the Harris-Benedict equation, which has been reported to be weakly related to the REE in severely ill patients [15]. However, even with these limitations, these data suggest that the pulmonary dead space can be measured in ALI/ARDS patients non-invasively, rapidly, and without any expensive equipment. Thus, in addition to taking daily measurements of oxygenation, compliance, and transpulmonary pressure, clinicians should include the pulmonary dead space for better clinical management.

Abbreviations

ALI, acute lung injury; ARDS, acute respiratory distress syndrome; CO_2, carbon dioxide; Pa_{CO2} arterial partial pressure of carbon dioxide; PA_{CO2}, alveolar partial pressure of carbon dioxide; PE_{CO2}, expired partial pressure of carbon dioxide; REE, resting energy expenditure; $\dot{V}_{CO2}$ estimated, estimated carbon dioxide production.

Competing interests

The authors declare that they have no competing interests.

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Published: 9 September 2010

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doi:10.1186/cc9237
Cite this article as: Chiumello D, Gallazzi E: **Estimation of dead space fraction can be simplified in the acute respiratory distress syndrome.** *Critical Care* 2010, **14:**195.