Artigo original

Volumetric Capnography in Acute Respiratory Distress Syndrome.

Capnografia Volumétrica na Síndrome do Desconforto Respiratório Agudo.

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ABSTRACT

Volumetric capnography is especially sensitive to disturbances affecting the efficiency of ventilation for gas exchange. Because lung homogeneity is a very fragile property, it is endangered in the majority of diseases that affect the airways, lung parenchyma, or alveolar microcirculation.

Acute lung injury and acute respiratory distress syndrome can be conveniently monitored with volumetric capnography. The combination of two advanced technologies—airway flow monitoring and mainstream capnography—allows breath-by--breath bedside computerized determination of the physiological dead space, alveolar heterogeneity, and CO_2 elimination.

The use of volumetric capnography at the bedside can provide clinicians with important physiological and prognostic data, as well as allowing the effects of therapeutic interventions to be evaluated in critical ill patients receiving mechanical ventilation.

Keywords: acute lung injury; pulmonary ventilation; respiratory dead space; capnography.

RESUMO

A capnografia volumétrica é especialmente sensível aos problemas que afetam a eficiência da ventilação para a troca gasosa. Uma vez que a homogeneidade do pulmão é uma propriedade muito frágil, a medida da capnografia é um desafio na maioria das doenças que comprometem as vias aéreas, o parênquima pulmonar e a microcirculação alveolar.

A lesão pulmonar aguda e síndrome do desconforto respiratório agudo são situações que devem ser monitoradas com a capnografia volumétrica. Essa tecnologia avançada é uma combinação da medida do fluxo aéreo e a capnografia convencional, fazendo com que seja possível computar, à beira do leito, parâmetros como espaço morto, heterogeneidade alveolar e eliminação do CO_2.

O uso da capnografia volumétrica à beira do leito pode fornecer aos clínicos importantes informações fisiológicas e sobre o prognóstico, assim como seguir o efeito de intervenções terapêuticas nos doentes críticos ventilados mecanicamente.

Descritores: lesão pulmonar aguda; ventilação pulmonar; espaço morto respiratório; capnografia.

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INTRODUCTION

Capnographic monitoring has become an important tool for ensuring patient safety. It allows early detection of pulmonary embolism and ventilator malfunction (1). It also reflects alterations in respiratory mechanics and provides prognostic data during cardiopulmonary resuscitation (2,3). In recent years, increasing interest in defining prognostic factors has led to a re-evaluation of some common physiological and clinical parameters used in intensive care (4-6).

Volumetric capnography provides a great amount of information on the functional status of the lung through the instantaneous recording of the expired CO_2 fraction (F_ECO_2) or CO_2 production (VCO_2[v]) versus expired volume ($FECO_2$[v]), contain an important amount of information on the functional status of the lung. In the past, the use of volumetric capnography in clinical practice was limited because of various problems related to measurement and interpretation. Once the difficulty inherent to the phase lag between volume and CO_2 signal had been overcome, the main technical problem related to mainstream capnometry became the fact that it is difficult to parameterize this curve without any visual reference points. Therefore, capnographic monitoring has often been reserved for the determination of end-tidal CO_2 tension ($P_{ET}CO_2$), in emergency trauma surgery, or in acute respiratory distress syndrome (ARDS) patients (7,8). However, when the arterial-alveolar gradient of CO_2 is significantly altered, the $P_{ET}CO_2$ can be misleading (9,10), which limits its use in clinical practice. In recent studies conducted by our group (11,12), we revisited the VCO_2(v) and F_ECO_2(v) curves in order to obtain data that are more easily digitized than are those typically obtained from the time-based F_ECO_2 (F_ECO_2[t]) curve.

VOLUMETRIC CAPNOGRAPHY PARAMETERS

The shape of the expired capnograph depends on the homogeneity of gas distribution and alveolar ventilation (13). Lung heterogeneity creates regional differences in CO_2 concentration, and gas from regions with high ventilation/perfusion first appears in the upper airways during exhalation. This sequential emptying contributes to the positive slope of the alveolar plateau (13). Greater ventilation/perfusion heterogeneity leads to a steeper alveolar CO_2 slope (14).

Classically, three distinct phases have been identified in the F_ECO_2 versus F_ECO_2(v) curves: phase I, in which there is no CO_2 elimination corresponding to the exhalation of the gas content of the physiological dead space; phase II, a transition phase during which F_ECO_2 increases progressively; and phase III, or the "alveolar phase", a plateau during which F_ECO_2 increases almost in parallel with expired volume. These phases cannot be identified without visual reference points defining the transitions between phases. Many computerized procedures have been unsuccessful because the transitions between phases are seamless. A new approach to volumetric capnography involves the use of the F_ECO_2 versus VCO_2(v) curve, rather than the F_ECO_2 versus F_ECO_2(v) curve or the F_ECO_2 versus F_ECO_2(t) curve. In recent years, our group defined physiologically based parameters that do not require visual reference points on the curve and can be obtained by computerized procedures (11,12,15). Such parameters, old and new, can be continuously evaluated and averaged over many cycles in real time, therefore being useful for monitoring purposes.

End-Tidal CO_2 Fraction

To avoid the cardiac motion artifact, the end-tidal CO_2 fraction ($F_{ET}CO_2$) can be measured on the VCO_2(v) curve:

$$VCO_2 = \int F_ECO_2 \cdot dV \qquad [1]$$

As the linear slope between expired volume and the VCO_2 of the segment defined by the end-expiratory 10% of the total number of expiratory samples of each breath (Figure 1).

Bohr Dead Space

The Bohr dead space (V_D^{Bohr}) can be calculated according to classical principles, assuming that $FETCO_2$ represents the alveolar fractional concentration of CO_2 in the following equation:

$$V_D^{Bohr}/V_T = 1 - (VCO_2tot/V_T)/F_{ET}CO_2 \qquad [2]$$

where V_T is the tidal volume and VCO_2tot is the total CO_2 eliminated in the breath.

Pre-Interface Expirate

The pre-interface expirate (PIE) can be calculated according to the method devised by Wolff and Brunner (16,17) as the mean of the normalized distribution function of phase II. The volume at which this mean value is obtained represents the minimal mean volume of the convective airways, or PIE (16). Physiologically speaking, the PIE represents the expired volume at which the interface between the airways and alveolar gas becomes identifiable upon the opening of the airways.

Slope of Phase III

The portion of the F_ECO_2(v) curve between PIE and V_T is divided into four segments. In accordance with Åström et al. (17), the slope of phase III (slope III) is calculated as the slope of the linear regression line between F_ECO_2 and volume for the two central segments.

Series Dead Space

The $FECO_2$(v) curve is corrected for the slope III between the PIE and end-tidal CO_2 volume. From the corrected new curve, the volume of the series dead space (V_D^{ser}) is calculated by the Fowler equal area method.

This procedure prevents overcorrection of phase II (17). The airway dead space ($V_D$aw) can be obtained by subtracting the instrumental dead space from the V_D^{ser}.

Index of Alveolar Heterogeneity

According to various authors (18,19), the difference between V_D^{ser} and V_D^{Bohr} is mainly attributable to unequal regional distribution, which distorts the curve beyond the PIE. An index of alveolar heterogeneity (IAH) can be calculated by relating the two magnitudes:

$$IAH(\%) = \left[1 - \frac{(V_T - V_D^{Bohr})}{(V_T - V_D^{ser})}\right] . 100 \quad [3]$$

Alveolar Ejection Volume

As previously demonstrated (12,15), alveolar ejection volume (V_{AE}) can be determined from the VCO_2(v) curve (Figure 1). It has been shown that, by pivoting on the end-expiratory point after linear fitting of the last end-expiratory segment, the slope of the VCO_2(v) curve can be decreased by 5% in ventilated patients (11) and by 6% in spontaneously breathing patients (20). The new line crosses the VCO_2(v) curve at a single point. The volume difference between this and the end-expiratory point corresponds to the V_{AE}. The V_{AE} tends to decrease as serial contamination of alveolar gas, heterogeneity, and phase II increase. According to the hypothesis of sequential gas exhalation, the V_{AE} fraction (V_{AE}/V_T ratio) is described as the fraction of V_T that is contaminated (because of alveolar heterogeneity and airway mixing), which is smaller than is that of the physiological dead space in terms of the end-tidal expired gas. In previous studies, performed in intubated individuals (healthy subjects and patients), V_{AE}/V_T was a satisfactory measure of the degree of lung impairment and correlated with other indices of the distribution of ventilation (13).

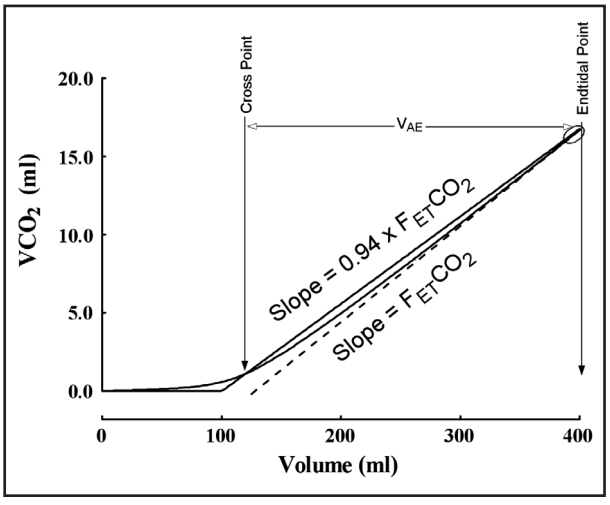

Figure 1 - Determination of V_{AE} on the VCO2(v) curve. $F_{ET}CO_2$ is obtained by linear fitting of the last end-expiratory segment (50 points) of the curve (ellipse).

Index of Ventilatory Efficiency

Because V_{AE} is directly dependent on V_T and V_D^{ser}, it seems appropriate to express it in relation to the magnitudes of those parameters. An index of ventilatory efficiency (IVE) can be calculated as follows:

$$IVE(\%) = \frac{V_{AE}}{(V_T - V_D^{ser})} . 100 \quad [4]$$

In healthy subjects and in patients, the IVE is less dependent on V_T than are other capnographic indices (20).

VOLUMETRIC CAPNOGRAPHY IN ACUTE LUNG INJURY AND ARDS

Acute lung injury (ALI) is characterized by diffuse alveolar injury, alveolar collapse, or consolidation, together with severe vascular damage, protein-rich lung edema, surfactant inactivation, and inflammation. Patients with ALI or ARDS present with low ventilation/perfusion (and high alveolar CO_2 tension) in some regions of the lung, which typically coexist with other regions in which there is high ventilation/perfusion (and low alveolar CO_2 tension). The combination of these two conditions (caused by severe alveolar and vascular damage) results in increased pulmonary dead space and alveolar heterogeneity. In addition, the pulmonary dead space is increased in individuals suffering from shock, systemic or pulmonary hypotension, and obstruction of pulmonary vessels (massive pulmonary embolus or microthrombosis). Artificial ventilation adds to the complexity of understanding in variations of dead space at the bedside because it can substantially affect dead space. Positive end-expiratory pressure (PEEP) levels that recruit collapsed lung can reduce the dead space, primarily by reducing intrapulmonary shunt, whereas overdistension of the lung promotes the development of high ventilation/perfusion regions and increases the dead space (21). Therefore, a number of pulmonary and extrapulmonary factors can affect the bedside interpretation of changes in the volume of the dead space.

Studies have shown that the hypoxemia seen in patients with ARDS is caused by intrapulmonary shunt and by low ventilation/perfusion ratios in some regions of the lung (22). In addition, the use of the multiple inert gas elimination technique has shown that, in patients with ARDS, a large portion of the ventilation is distributed to nonperfused or poorly perfused regions (22). In the oleic acid-injured lungs of dogs, Coffey et al. (21) found that high V_D/V_T correlated with shunt, inert gas dead space, and mid-range ventilation/perfusion heterogeneity. The available capnographic data indicate that, in ALI and ARDS patients, the distribution of ventilation is quite uneven and the ventilatory process is inefficient. In a study conducted by Blanch et al. (12), indices obtained from volumetric capnography (V_D^{Bohr}/V_T, slope III, and V_{AE}/V_T)

were markedly different in ALI and ARDS patients than in the controls. The V_D^{Bohr} and slope III were significantly higher in ALI and ARDS patients than in the controls, as well as being significantly higher in the patients with ARDS than in those with ALI. The V_{AE}/V_T was significantly lower in the ALI and ARDS patients than in the controls and was significantly lower in the ARDS patients than in ALI patients.

Effect of V_T

In recumbent, anesthetized healthy subjects, an increase in V_T increases ventilatory efficiency. Studies involving healthy subjects (23) have shown that relatively small increases in V_T result in greater convection-dependent heterogeneous ventilation, whereas that due to the interaction of convection and diffusion in the lung periphery decreases. In a study conducted by Romero et al. (11), volume had a significant effect on V_{AE}/V_T in healthy subjects but not in ARDS patients. These results are in agreement with those of Paiva et al. (24), who also showed that an increase in V_T reduces slope III in healthy subjects. In ARDS patients, an increase in V_T might be expected to recruit some alveolar units and to increase, to some extent, the degree of alveolar homogeneity (25). In fact, only if recruited units were strictly normal and homogeneous would they contribute to improving ventilatory and mechanical efficiency. We can reasonably suppose that the absence of a V_T-related increase in V_{AE}/V_T and IVE in ARDS patients is attributable to the fact that that an increase in V_T does not effectively recruit new lung areas or that most of the alveoli recruited are diseased. This raises the hypothesis that increased physiologic dead space and decreased V_{AE}/V_T are indicators of a poor prognosis in ARDS and that their evolution during treatment has an impact on outcomes (26-29).

Effect of PEEP

The alveolar dead space is significantly increased in ALI and is not affected by the use of PEEP. However, when PEEP is administered to recruit collapsed lung units (resulting in improved oxygenation), the alveolar dead space decreases unless overdistension impairs alveolar perfusion. Breen and Mazumdar (30) found that the application of 11 cmH_2O of PEEP in anesthetized, mechanically ventilated, open-chested dogs increased the physiological dead space, reduced VCO_2tot and resulted in a poorly defined alveolar plateau. These changes were mainly produced by a significant decrease in cardiac output related to the use of PEEP. Tusman et al. (31) tested the usefulness of the dead space parameter for determining open-lung PEEP in eight pigs submitted to lung lavage. We find it interesting that the alveolar dead space correlated well with arterial oxygen tension, normally aerated areas, and non-aerated areas in all animals, with a sensitivity of 89% and a specificity of 90% for detecting lung collapse. However, in saline lavage-induced experimental animal models of ARDS, there is considerable potential for recruitment that increases in parallel with increases in PEEP (32), and comparisons with ARDS in humans should therefore be made with caution.

The relationship between the effects of PEEP on volumetric capnography and respiratory mechanics have been studied in patients with normal lungs, patients with moderate ALI, and patients with severe ARDS. Blanch et al. (12) found that patients with ARDS presented with markedly lower respiratory system compliance and greater total respiratory system resistance than did controls. Although an increase in PEEP improved respiratory mechanics in healthy subjects and worsened lung tissue resistance in patients with respiratory failure, it did not affect volumetric capnography indices. Smith and Fletcher (33) studied heart surgery patients and also found that PEEP did not modify CO_2 elimination in the immediate postoperative period. Beydon et al. (34) studied the effect of PEEP on the dead space in patients with ALI. The authors found a high V_D/V_T that was unaffected by raising PEEP from 0 to 15 cmH_2O. Patients in whom oxygenation improved with PEEP showed a concurrent decrease in V_D/V_T and vice versa. In an experimental animal model of oleic acid-induced ARDS, Coffey et al. (21) found that low PEEP reduced physiological V_D/V_T and intrapulmonary shunt. Conversely, in the same animals, high PEEP increased the fraction of ventilation delivered to areas with high ventilation/perfusion, resulting in increased physiological V_D/V_T. Variations in V_D/V_T after the initiation of PEEP largely depend on the type, degree, and stage of lung injury.

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