CRITICAL CARE

LETTER

Effect of acute hyperventilation on the venous-arterial PCO_2 difference

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See related letter by Morel *et al.*, http://ccforum.com/content/15/6/456

I read with great interest the letter by Morel and colleagues [1] in the previous issue of *Critical Care*. The letter suggested that acute changes in the arterial partial pressure of carbon dioxide ($PaCO_2$) can affect the venous-arterial difference in carbon dioxide tension (ΔCO_2). In a study by the authors, 10 ventilated and hemodynamically stable patients were included after elective cardiac surgery. Hypocapnia was induced by increasing the respiratory rate. The authors found that a decrease of $PaCO_2$ was associated with a significant increase in ΔCO_2. This was explained by the fact that acute hypocapnia resulted in systemic vasoconstriction, thus decreasing the elimination of the total CO_2 produced by the peripheral tissues and increasing the gap. However, as all patients were monitored with a pulmonary artery catheter (PAC), the authors should have shown whether there was any increase in systemic vascular resistance to support their hypothesis. Furthermore, there is another possible explanation of the ΔCO_2 increase induced by the decrease in $PaCO_2$. Indeed, acute respiratory alkalosis has been shown to increase systemic oxygen consumption and CO_2 production [2,3]. Thus, for a given venous blood flow, the increase of tissue CO_2 production should increase the partial pressure of carbon dioxide (PCO_2) gap.

On the other hand, it is unclear why the authors have used the central venous sample to calculate ΔCO_2 instead of using the mixed venous sample (PAC), which is the gold standard. If a PAC is in place, the clinical utility of an alternative method of measurement is diminished even though the mixed and central PCO_2 difference showed good agreement [4]. Nevertheless, I agree that acute hyperventilation could be a potential limitation of the clinical application of the ΔCO_2.

Authors' response

Jerome Morel and Laurent Gergele

We thank Mallat for his comments. Indeed, we hypothesized that hypocapnia resulted in microcirculatory vasoconstriction, thus decreasing the elimination of CO_2 produced by the peripheral tissues and increasing the venous-arterial CO_2 gradient [1]. Only specific microcirculatory monitoring can answer this question. Measurement of systemic vascular resistance could not help as microcirculation and systemic circulation are relatively dissociated, particularly when systemic hemodynamics are within normal ranges (as was the case in our study) [5].

As mentioned, it has been shown that central venous CO_2 and mixed venous CO_2 were in good agreement for the calculation of venous-arterial CO_2 gradient, as was the case in our study (data not shown). We chose to present central venous data because nowadays patients are more frequently monitored with a central venous catheter than a PAC. However, Mallat raised an interesting hypothesis concerning the effects of acute respiratory alkalosis on systemic oxygen consumption and CO_2 production, which could be an explanation of our results [2].

Abbreviations

ΔCO_2, venous-arterial difference in carbon dioxide tension; CO_2, carbon dioxide; PAC, pulmonary artery catheter; $PaCO_2$, arterial partial pressure of carbon dioxide; PCO_2, partial pressure of carbon dioxide.

Competing interests

The author declares that he has no competing interests.

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