Editorial

Can calculated central venous saturation be used as a reliable tool to guide therapy in patients with shock?

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Access this article online
Website: www.ijccm.org
DOI: 10.4103/0972-5229.114818
Quick Response Code:

Accurate and rapid measurement of pathophysiological derangements is crucial in order to intervene early and effectively. Measurement of various parameters has not only become possible but also precise, fast, and in some cases continuous. However, improved technology has brought with it increasing monitoring and treatment costs that has resulted in critical care practice becoming less affordable, particularly in developing countries such as ours. Thus the article in this issue of the *Indian Journal of Critical Care Medicine* (IJCCM), by Subramanian *et al.*,[1] is relevant. In this study,[1] the authors compared central venous saturation by standard arterial blood gas (ABG) with saturation obtained by cooximetry. Patients with different clinical conditions that are known to shift the oxygen dissociation curve and thus influence calculated oxygen saturation were included. The authors reported a sensitivity of 84.2% and specificity of 93.2% for central venous oxygen saturation (ScVO2) with the use of standard ABG against cooximetry. They concluded that "standard ABG can be a satisfactory and cost-effective surrogate for the more expensive cooximetry when assessing ScVO2 for patients with shock."

Two aspects need to be addressed. First, is the correlation between the standard ABG ScVO2 and cooximetry ScVO2, as reported by the authors,[1] acceptable? Second, if it is acceptable, can we justify its use as a surrogate to ScVO2 as measured by cooximetry? The authors[1] support the use of standard ABG ScVO2 on the basis of "satisfactory correlation" with the gold standard and the cost, given that such measurements are about half the cost of the gold standard. The sample size of 141 is relatively small and an agreement of standard ABG ScVO2 of 0.69 indicates only a modest correlation. It must be pointed out that "a statistically significant correlation coefficient is not necessarily an important one"[2] and the clinical implication of $r = 0.69$ needs to be kept in mind particularly for a test like ScVO2.

Breuer *et al.*,[3] assessed the agreement between calculated oxygen saturation and photometrically measured oxygen saturation in different clinical situations using six different equations on 1350 samples. The authors concluded that, "no calculation mode can be performed with constant accuracy and reliability when covering a wide range of acid-base values."[3] In a subsequent study, Nierman and Schechter[4] analyzed the correlation between measured and calculated oxygen saturation in 118 mixed venous blood samples from medical intensive care unit patients. They observed that calculated ABG saturations had a sensitivity of only 58.3% and specificity of 89% when compared with measured cooximetry.[4] Attempts to mathematically improve upon the equation using additional four regression equations resulted in universally worse sensitivity.[4] In another multicenter study,[5] for PaO2 in the range 45–60 mmHg, 16% of the calculated saturation values for arterial samples were different by more than

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3% above or below the measured values and in some cases the differences were up to 10%.[5] These studies suggest that calculated oxygen saturation does not accurately correlate with measured oxygen saturation.

The applicability of calculated oxygen saturation in clinical practice is further limited by the large limits of agreement (–24.2% to 19.5%) reported by Subramanian *et al*.[1] The limits of agreement of >20%, in clinical practice, may make the test meaningless for the clinician, as for any given ScVO2 on the standard ABG, the actual value may even be 20% more or less. Thus, patients can be easily misclassified if one uses a threshold of ScVO2 of 70% to identify patients at risk for poor outcome due to shock. In addition, although only four patients were misclassified as "end-points reached" on a standard ABG ScVO2, if we include only patients with shock (n = 44), then the proportion of patients misdiagnosed increases 9.1%.

The use of "measured ScVO2" needs to be looked at in the larger context of its applicability in the evaluation and management of shock. ScVO2 has traditionally been proposed as a useful tool that reflects global oxygen transport and metabolism of oxygen.[6] Although low ScVO2 levels may reflect inadequate cardiac output with excessive oxygen extraction as occurs in shock, low hemoglobin and/or inadequate oxygenation, high levels may sometimes be seen in shock as a result of mitochondrial dysfunction and reduced oxygen utilization.[6] Recent concerns on the utility of ScVO2 in sepsis[7] have led to the reevaluation of ScVO2 in larger clinical trials.[8] The results of these trials would further refine the evaluation and management of shock.

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How to cite this article: Peter JV. Can calculated central venous saturation be used as a reliable tool to guide therapy in patients with shock?. Indian J Crit Care Med 2013;17:69-70.

Source of Support: Nil, **Conflict of Interest:** None declared.