

COMMENTARY

Tissue saturation measurement - exciting prospects, but standardisation and reference data still needed

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See related research by Možina and Podbregar, http://ccforum.com/content/14/2/R42

Abstract

Sepsis and shock result in disturbances in microcirculatory perfusion and tissue oxygen utilisation that may not be reflected in global measures of haemodynamics. Near-infrared spectroscopy enables measurement of tissue oxygen saturation (StO₂) and provides information on local microvascular and mitochondrial function. This measure could be incorporated with existing targets of goal-directed therapy to provide an integrated approach to haemodynamic resuscitation of both the macro- and microcirculation in various shock states. However, key methodological factors must be addressed before widespread clinical application.

In the previous issue of Critical Care, Možina and Podbregar report that changes in thenar muscle tissue saturation (StO₂) in response to vascular occlusion can be used to predict discrepancy between central (ScvO₂) and mixed (SvO₂) venous oxygen saturation in patients with combined severe left ventricular failure and sepsis [1].

On first read this study is conceptually difficult. Essentially, ScvO₂ is a surrogate marker of SvO₂ and is widely used to assess adequacy of oxygen delivery, and to guide the initial resuscitation of septic patients [2]. Trends in ScvO₂ may reflect those in SvO₂, but alterations in oxygen extraction and redistribution of blood flow away from peripheral and splanchnic circulations in various shock states may cause clinically important differences between these values [3]. In these circumstances ScvO₂ may be maintained despite abnormally low

SvO₂ and significant tissue hypoxia. The current study suggests a means to identify these discrepancies which may be useful in the management of sepsis/septic shock, particularly in patients with complex haemodynamic disturbances. However, several issues relating to methodological factors require further consideration.

Microcirculatory perfusion and tissue oxygen utilization are affected by sepsis and shock [4]. These derangements can be studied non-invasively using near-infrared spectroscopy, a technique that is able to determine the oxygenation status of tissue haemoglobin. Decreased StO₂ reflects the presence of severe hypoperfusion and has been used clinically to guide resuscitation during hypovolaemic shock [5]. Unfortunately, in sepsis/septic shock absolute values of StO₂ do not reliably differentiate patients from healthy individuals [6].

The discriminatory power and predictive ability of StO₂ can, however, be improved by measuring the response to an ischaemic challenge. The vascular occlusion test (VOT) is a provocative test in which StO₂ is measured at a peripheral site (such as the thenar eminence) whilst a transient rapid vascular occlusion is performed for either a defined time interval or until a pre-defined StO₂ value is reached. The VOT-derived StO₂ trace can be divided into four phases - baseline, ischemia, reperfusion, and hyperemia - and parameters can be measured for each of these. These include: rate of deoxygenation (DeO₂), measured from the StO₂ downslope during the ischaemic phase - this is generally considered to reflect local muscle metabolism and mitochondrial oxygen consumption [7]; and rate of reoxygenation (ReO₂), measured from the StO₂ upslope during reperfusion - this is the time required to wash out stagnant blood and is thought to reflect the reactivity of the microcirculation [8].

Studies suggest that the response to the VOT differs in sepsis, with a slower DeO, during cuff occlusion and a prolonged ReO2 during reperfusion when compared to healthy individuals [9,10]. Furthermore, it seems that VOT reflects severity of illness and is sensitive to intervention with activated protein C [8,11]. According

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to the current study it may also have a role in guiding resuscitation and optimisation of haemodynamics.

These findings are exciting, but at present enthusiasm must be tempered. Firstly, there are wide variations in StO₂ and VOT-derived parameters dependent upon probe position and probe size [12]. Secondly, the VOT procedure has yet to be standardized; currently, different types and degrees of deflation thresholds (for example, absolute StO₂ of 40% or 50% versus duration of 3 minutes, 4 minutes, 5 minutes) are employed [13]. Thirdly, context-specific ranges for VOT-derived StO₂ parameters need to be established in health to ensure correct interpretation of responses in pathological states. High quality data are also needed on the VOT parameter values for different levels of disease severity, and indeed for different diseases. Finally, the mechanisms underlying differences in StO₂ response observed in sepsis need to be fully characterized.

Dynamic ${\rm StO}_2$ monitoring is a promising technique with the potential to provide a non-invasive 'biopsy' of microvascular and mitochondrial function. Used in conjunction with global measures of oxygen delivery it could provide an integrated approach to haemodynamic resuscitation of both the macro- and microcirculation in various shock states. Furthermore, it may have utility in determining disease severity, outcome prediction, assessment of the biological effects of interventions, and in guiding future research. However, as with all new technologies it is essential that operating characteristics are robustly defined and that the limitations are fully appreciated before wider application to the clinical setting.

Abbreviations

 $\label{eq:equation} \text{DeO}_2 = \text{rate of deoxygenation; ReO}_2 = \text{rate of reoxygenation; ScvO}_2 = \text{central venous oxygen saturation; StO}_2 = \text{tissue oxygen saturation; SvO}_2 = \text{mixed venous oxygen saturation; VOT} = \text{vascular occlusion test.}$

Competing interests

 MT has received StO_2 equipment in support of research from Hutchinson Technology Inc.

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