CRITICAL CARE

COMMENTARY

Central venous oxygen saturation in septic shock - a marker of cardiac output, microvascular shunting and/or dysoxia?

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See related research by Textoris *et al.*, http://ccforum.com/content/15/4/R176

Abstract

Shock therapy aims at increasing central venous oxygen saturation ($ScvO_2$), which is a marker of inadequate oxygen delivery. In this issue of *Critical Care*, Textoris and colleagues challenge this notion by reporting that high levels of $ScvO_2$ are associated with mortality in patients with septic shock. This is of obvious interest, but as their retrospective design has inherent limitations, the association should be confirmed in a prospective, multicenter study with protocolized $ScvO_2$ measurements and detailed registration of potentially confounding factors.

In this issue of *Critical Care*, Textoris and colleagues [1] report that high levels of central venous oxygen saturation ($ScvO_2$) are associated with mortality in patients with septic shock. $ScvO_2$ is easily measured in most ICU patients and thus represents a convenient therapeutic marker in the resuscitation of the critically ill patient. Low $ScvO_2$ is a valid therapeutic target in early septic shock [2,3] and a diagnostic marker for low cardiac output [4]. $ScvO_2$ itself is a surrogate marker of mixed venous oxygen saturation, which in theory reflects the balance between global oxygen delivery and consumption, so that low $ScvO_2$ is a marker of inadequate oxygen delivery. Still we do not fully understand the pathophysiological and clinical meaning of altered $ScvO_2$.

So far, the focus has mostly been on low levels of $ScvO_2$, but Textoris and co-workers [1] have focused on higher levels of $ScvO_2$ in septic shock patients. They hypothesise that levels of $ScvO_2$ above 80% correlate with increased mortality and that this correlation is likely due to impaired ability to extract oxygen. In a retrospective design, they identified all adult ICU patients with septic shock in a 2-year period and registered lowest and highest $ScvO_2$ measurements during the first 3 days in the ICU. They found that the maximum $ScvO_2$ was significantly higher in the patients that died in hospital than in those who survived (85% versus 79%, $P = 0.009$). In contrast, the minimum $ScvO_2$ did not differ between these groups. The association between maximum $ScvO_2$ and mortality persisted in a multivariate analysis adjusting for other variables that differed between the survivors and non-survivors.

This is of obvious interest, but the study has several limitations, as the authors point out. The retrospective design carries an inherent risk of selection bias since patients with mild disease or early death might not have had any $ScvO_2$ measurements and were then excluded from the study. Furthermore, $ScvO_2$ might have been measured more frequently in the most severely ill patients, increasing the chance for a high $ScvO_2$ measurement, especially as $ScvO_2$ varies over time.

The interpretation of $ScvO_2$ remains a challenge. $ScvO_2$ depends on arterial oxygen saturation, cardiac output, oxygen consumption, haemoglobin levels and shunting. The ability of $ScvO_2$ to reflect systemic oxygen delivery/consumption is not constant in time as it depends on many conditions, including sedation, ventilator treatment [5], redistribution of blood as seen in shock [6] and thus shock severity, the position of the catheter tip, which depends on the body position [7], and so on. Thus, the complex mechanisms influencing $ScvO_2$ hamper the interpretation.

Textoris and co-workers hypothesise that the high $ScvO_2$ in the non-survivor group is likely due to impaired oxygen extraction, but there may be other explanations. It is likely that very aggressive resuscitation with high doses of fluid, vasopressors, inotropes and blood resulting in supranormal oxygen delivery, and thus high $ScvO_2$,

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negatively impacts on survival [8]. Alternatively, impaired regulation of the microcirculation might have resulted in shunting and thus high $ScvO_2$. Other, unknown confounders may also have existed. The current data cannot distinguish between these alternative hypotheses as discussed by the authors.

If high $ScvO_2$ associates with increased mortality in sepsis, it may have clinical implications. But the hypothesis should be evaluated in a prospective, multicentre study with protocolized $ScvO_2$ measurements and detailed registration of potentially confounding factors, including use of fluid, vasopressors, inotropes and blood, to reduce the risk of bias. Results from such a study have the potential to influence the design of further clinical trials evaluating $ScvO_2$ as a target for shock therapy.

Abbreviations

$ScvO_2$, central venous oxygen saturation.

Competing interests

The authors declare that they have no competing interests.

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