CRITICAL CARE

COMMENTARY

SvO_2 to monitor resuscitation of septic patients: let's just understand the basic physiology

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See related research by Velissaris *et al*., http://ccforum.com/content/15/4/R177

Abstract

Real-time monitoring of mixed venous oxygen blood saturation (SvO_2) or of central venous oxygen blood saturation is often used during resuscitation of septic shock. However, the meaning of these parameters is far from straightforward. In the present commentary, we emphasize that SvO_2 – a global marker of tissue oxygen balance – can never be simplistically used as a marker of preload responsiveness, which is an intrinsic marker of cardiac performance. In some septic shock patients, because of profound hypovolemia or myocardial dysfunction, SvO_2 can be low but obviously cannot alone indicate whether a fluid challenge would increase cardiac output. In other patients, because of a profound impairment of oxygen extraction capacities, SvO_2 can be abnormally high even in patients who are still able to respond positively to fluid infusion. In any case, other reliable dynamic parameters can help to address the important question of fluid responsiveness/unresponsiveness. However, whether fluid administration in fluid responders and high SvO_2 would be efficacious to reduce tissue dysoxia in the most injured tissues is still uncertain.

In a study of patients with sepsis, Velissaris and colleagues showed that high mixed venous blood oxygen saturation (SvO_2) levels do not exclude fluid responsiveness [1]. SvO_2 is assumed to reflect the balance between arterial oxygen delivery (DO_2) and oxygen consumption (VO_2) provided arterial blood oxygen saturation (SaO_2) is normal. Indeed, the modified Fick equation states:

$$SvO_2 = SaO_2 - [VO_2 / (\text{cardiac output} \times Hb \times 1.34)]$$

where Hb is the hemoglobin concentration.

Nevertheless, the interpretation of SvO_2 and its changes in shock states must be cautious for at least four reasons. First, in any shock state, oxygen demand exceeds VO_2 by definition such that SvO_2 cannot reflect the balance between DO_2 and oxygen demand, which is better assessed by markers of anaerobic metabolism such as the blood lactate level. Second, as VO_2/DO_2 dependency is a characteristic pattern of shock states [2], any increase in DO_2 during resuscitation will be associated with a simultaneous increase in VO_2 and hence with no or only a small increase in SvO_2 until a critical DO_2 is reached. Third, the tissue oxygen utilization is impaired in severe sepsis so VO_2 may decrease relative to oxygen demand, even if DO_2 is normal or high. Fourth, because of the hyperbolic relationship between SvO_2 and cardiac output (graphical representation of the modified Fick equation) [3], SvO_2 should not change much in response to an increase in cardiac output in cases of hyperdynamic shock states. For all these reasons, interest in SvO_2 monitoring in septic shock has been debated – although SvO_2 or its surrogate, central venous oxygen saturation, has been recommended as a major hemodynamic target of early resuscitation in septic shock [4,5].

Nevertheless, SvO_2 might still play an important role in the monitoring of septic shock by identifying the patients in whom DO_2 could be further augmented and then by guiding the therapy aimed at increasing DO_2. This point is of particular importance since systematic maximization of DO_2 is not recommended in every patient with septic shock [5].

Velissaris and colleagues showed that a given value of SvO_2 cannot be used to predict a positive response to fluid challenge [1]. These results are quite consistent with the basic physiology and deserve to be discussed. Preload responsiveness is an intrinsic property of cardiac performance, indicating that the heart is operating on the steep ascending part of the Frank–Starling relationship [6]. Preload responsiveness is therefore by nature a concept that differs from the concept of global VO_2/DO_2 balance and thus from SvO_2.

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Patients with low SvO_2 can be either fluid responsive in cases of hypovolemic shock or fluid unresponsive in cases of cardiogenic shock. In the study by Velissaris and colleagues almost two-thirds of patients with a low SvO_2 (<60%) were fluid nonresponders [1], suggesting that their heart was operating on the flat part of the Frank–Starling curve. This observation suggests that in the presence of low SvO_2 it is mandatory to obtain additional information to identify the cases where fluid administration should be considered the most logical therapeutic measure. Cardiac filling pressures are not appropriate to make such a therapeutic decision [7], as confirmed by Velissaris and colleagues [1]. By contrast, dynamic tests (pulse pressure variation, passive leg raising and end-expiratory occlusion tests) are far more reliable to predict fluid responsiveness/unresponsiveness [8,9]. In cases of low SvO_2 the presence of fluid responsiveness should incite infusing fluid, whereas its absence should rather incite consideration of other therapies (for example, inotropes) that enable one to increase cardiac output with the ultimate goal of decreasing tissue hypoxia.

Because septic shock is often characterized by high cardiac output and low extraction oxygen capacities, high values of SvO_2 or central venous blood oxygen saturation can be observed [10,11] as confirmed in the study by Velissaris and colleagues [1]. One striking finding of their study is that an increase in cardiac output with fluid infusion occurred in more than 50% of patients who exhibited a high SvO_2 (>70%) at baseline [1]. This confirms that preload responsiveness can coexist with the presence of reduced oxygen extraction capacities and thus high SvO_2. A reliable prediction of fluid responsiveness would also be important to obtain in cases of high SvO_2. Whether the presence of preload responsiveness should lead to the decision to infuse fluid, however, is still uncertain. One should anticipate that abnormally high SvO_2 is an indicator of profoundly decreased oxygen extraction capacities so that the additional increase in DO_2 cannot be effectively distributed and/or utilized in the most injured peripheral tissues. In other words, one can reasonably postulate in cases of tissue hypoxia that the higher the SvO_2, the less likely the correction of tissue hypoxia with fluid infusion, even in the presence of indicators of preload responsiveness. Since the study by Velissaris and colleagues [1] was not designed to confirm this hypothesis, further studies are required.

Abbreviations

DO_2, oxygen delivery; Hb, hemoglobin concentration; SaO_2, arterial blood oxygen saturation; SvO_2, mixed venous blood oxygen saturation; VO_2, oxygen consumption.

Competing interests

J-LT and XM are members of the Medical Advisory Board of Pulsion Medical Systems (Munich, Germany). OH declares that he has no competing interests.

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