In a study of patients with sepsis, Velissaris and colleagues showed that high mixed venous blood oxygen saturation (SvO₂) levels do not exclude fluid responsiveness [1]. SvO₂ is assumed to reflect the balance between arterial oxygen delivery (DO₂) and oxygen consumption (VO₂) provided arterial blood oxygen saturation (SaO₂) is normal. Indeed, the modified Fick equation states:

\[ \text{SvO}_2 = \text{SaO}_2 - \left[ \frac{\text{VO}_2}{\text{cardiac output} \times \text{Hb} \times 1.34} \right] \]

where Hb is the hemoglobin concentration.

Nevertheless, the interpretation of SvO₂ and its changes in shock states must be cautious for at least four reasons. First, in any shock state, oxygen demand exceeds VO₂ by definition such that SvO₂ cannot reflect the balance between DO₂ and oxygen demand, which is better assessed by markers of anaerobic metabolism such as the blood lactate level. Second, as VO₂/DO₂ dependency is a characteristic pattern of shock states [2], any increase in DO₂ during resuscitation will be associated with a simultaneous increase in VO₂ and hence with no or only a small increase in SvO₂ until a critical DO₂ is reached. Third, the tissue oxygen utilization is impaired in severe sepsis so VO₂ may decrease relative to oxygen demand, even if DO₂ is normal or high. Fourth, because of the hyperbolic relationship between SvO₂ and cardiac output (graphical representation of the modified Fick equation) [3], SvO₂ should not change much in response to an increase in cardiac output in cases of hyperdynamic shock states. For all these reasons, interest in SvO₂ monitoring in septic shock has been debated – although SvO₂ or its surrogate, central venous oxygen saturation, has been recommended as a major hemodynamic target of early resuscitation in septic shock [4,5].

Nevertheless, SvO₂ might still play an important role in the monitoring of septic shock by identifying the patients in whom DO₂ could be further augmented and then by guiding the therapy aimed at increasing DO₂. This point is of particular importance since systematic maximization of DO₂ is not recommended in every patient with septic shock [5].

Velissaris and colleagues showed that a given value of SvO₂ 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 VO₂/DO₂ balance and thus from SvO₂.
Patients with low SvO₂ 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 SvO₂ (<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 SvO₂ 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 SvO₂ 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 SvO₂ 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 SvO₂ (>70%) at baseline [1]. This confirms that preload responsiveness can coexist with the presence of reduced oxygen extraction capacities and thus high SvO₂. A reliable prediction of fluid responsiveness would also be important to obtain in cases of high SvO₂. Whether the presence of preload responsiveness should lead to the decision to infuse fluid, however, is still uncertain. One should anticipate that abnormally high SvO₂ is an indicator of profoundly oxygen extraction capacities so that the increased increase in DO₂ 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 SvO₂, 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**
DO₂, oxygen delivery; Hb, hemoglobin concentration; SaO₂, arterial blood oxygen saturation; SvO₂, mixed venous blood oxygen saturation; VO₂, 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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