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PHYSIOLOGICAL NOTE

Frank Bloos
Konrad Reinhart

Venous oximetry

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F. Bloos · K. Reinhart (✉)
Klinik für Anästhesiologie und Intensivtherapie,
Klinikum der Friedrich-Schiller-Universität,
Erlanger Allee 101, 07747 Jena, Germany
e-mail: Konrad.Reinhart@med.uni-jena.de
Tel.: +49-3461-9323101
Fax: +49-3641-9323102

Introduction

The primary physiological task of the cardiovascular system is to deliver enough oxygen (O_2) to meet the metabolic demands of the body. Shock and tissue hypoxia occur when the cardiorespiratory system is unable to cover metabolic demand adequately. Sustained tissue hypoxia is one of the most important cofactors in the pathophysiology of organ dysfunction [1]. Therefore determining the adequacy of tissue oxygenation in critically ill patients is central to ascertain the health of the patient. Unfortunately, normal values in blood pressure, central venous pressure, heart rate, and blood gases do not rule out tissue hypoxia or imbalances between whole-body oxygen supply and demand [2]. This discrepancy has led to increased interest in more direct indicators of adequacy of tissue oxygenation such as mixed and central venous oxygen saturations. Pulmonary artery catheterization allows obtaining true mixed venous oxygen saturation (SvO_2) while measuring central venous oxygen saturation ($ScvO_2$) via central venous catheter reflects principally the degree of oxygen extraction from the brain and the upper part of the body. This brief review discusses the role and limitations of SvO_2 and $ScvO_2$ as indicators of the adequacy of tissue oxygenation.

Physiology of mixed venous and central venous oxygen saturation

O_2 delivery (DO_2) describes whole-body oxygen supply according to the following formula:

$$DO_2 = CO \times CaO_2 \quad (1)$$

where CO is cardiac output and CaO_2 is arterial oxygen content, which itself is the sum of oxygen bound to hemoglobin [product of hemoglobin concentration (Hb) and arterial O_2 saturation (SaO_2)] and physically dissolved oxygen [arterial PO_2 (PaO_2)]:

$$CaO_2 = (Hb \times 1.36 \times SaO_2) + (PaO_2 \times 0.0031) \quad (2)$$

Oxygen demand can be summarized in the whole-body oxygen consumption (VO_2), which is expressed mathematically by the Fick principle as the product of CO and arteriovenous O_2 content difference (CaO_2–CvO_2):

$$VO_2 = CO \times (CaO_2 - CvO_2) \quad (3)$$

where mixed venous O_2 content (CvO_2) is:

$$CvO_2 = (Hb \times 1.36 \times SvO_2) + (PvO_2 \times 0.0031) \quad (4)$$

Equation 3 may be transposed to:

$$CvO_2 = CaO_2 - \frac{VO_2}{CO} \quad (5)$$

As physically dissolved oxygen can be neglected, Eq. 5 may be written as:

$$Hb \times 1.36 \times SvO_2 \approx (Hb \times 1.36 \times SaO_2) - \frac{VO_2}{CO}$$

$$\Leftrightarrow SvO_2 \sim \frac{VO_2}{CO} \quad (6)$$

Equation 6 also demonstrates that SvO_2 is directly proportional to the ratio of VO_2 to CO. Thus SvO_2 reflects the relationship between whole-body O_2 consumption and cardiac output. Indeed, it has been shown that the SvO_2 is well correlated with the ratio of O_2 supply to demand [3].

Pathophysiology of central or mixed venous O_2 saturation during shock

Usually VO_2 is independent of DO_2 since tissues can maintain O_2 needs by increasing O_2 extraction when DO_2 decreases. However, this mechanism has its limits. Below a so-called critical DO_2 compensatory increase in O_2 extraction is exhausted, and VO_2 becomes dependent on DO_2. In this case tissue hypoxia occurs, and a rise in serum lactate levels may be observed [4].

A decrease in SvO_2 and $ScvO_2$ represents an increased metabolic stress, because the O_2 demands of the body are not completely met by DO_2. The causes of a decreasing SvO_2 are multiple and reflect the forces operative in Eqs. 5 and 6. That is, either DO_2 does not increase in such a way to cover an increased VO_2, or DO_2 drops because of decrease in either arterial O_2 content, cardiac output, or both. Importantly, the normal cardiovascular response of increasing VO_2 is to increase O_2 extraction and cardiac output. Thus SvO_2 normally decreases during exercise despite increasing DO_2. Therefore a drop in SvO_2 or $ScvO_2$ does not necessarily mean that tissue hypoxia occurs. The magnitude of the decrease indicates the extent to which the physiological reserves are stressed (Table 1). Whereas in otherwise healthy individuals anaerobic metabolism may occur when SvO_2 drops below its normal value of 75% to 30–40% for a substantial period of time, patients with chronic heart failure may live with an SvO_2 in this low range without apparent tissue hypoxia, presumably because they have adapted to higher oxygen extraction. These patients can increase their VO_2 to a limited degree, however, because O_2 extraction is close to its limits as is cardiac output.

The cardiocirculatory system may be challenged by two different conditions. Firstly, a drop in DO_2 can be induced by anemia, hypoxia, hypovolemia, or heart failure. Secondly, fever, pain, stress etc. may also decrease SvO_2 or $ScvO_2$ by increasing whole-body VO_2 (Table 2)

Since central venous catheterization is commonly performed for a variety of reasons in critically ill patients, it would be useful if $ScvO_2$ could function as a surrogate for SvO_2. The central venous catheter sampling site usually resides in the superior vena cava. Thus central venous blood sampling reflects the venous blood of the upper body but neglects venous blood from the lower body (i.e., intra-abdominal organs). As presented in Fig. 1, venous O_2 saturations differ among several organ systems since they extract different amounts of O_2. $ScvO_2$ is usually less than SvO_2 by about 2–3% because the lower body extracts less O_2 than the upper body making inferior vena caval O_2 saturation higher. The primary cause of the lower O_2 extraction is that many of the vascular circuits that drain into the inferior vena cava use blood flow for nonoxidative phosphorylation needs (e.g., renal blood flow, portal flow, hepatic blood flow). However, SvO_2 and $ScvO_2$ change in parallel when the whole-body ratio of O_2 supply to demand is altered [5].

Table 1 Limits of mixed venous oxygen saturation

SvO_2 >75%	Normal extraction O_2 supply >O_2 demand
75% >SvO_2 >50%	Compensatory extraction Increasing O_2 demand or decreasing O_2 supply
50% >SvO_2 >30%	Exhaustion of extraction Beginning of lactic acidosis O_2 supply <O_2 demand
30% >SvO_2 >25%	Severe lactic acidosis
SvO_2 <25%	Cellular death

Table 2 Clinical conditions and their effects on O_2 delivery and O_2 consumption and on venous oximetry

Decrease in $ScvO_2$/SvO_2
O_2 consumption ↑
Stress
Pain
Hyperthermia
Shivering
O_2 delivery ↓
CaO_2 ↓ (anemia, hypoxia)
Cardiac output ↓
Increase in $ScvO_2$/SvO_2
O_2 delivery ↑
CaO_2 ↑
Cardiac output ↑
O_2 consumption ↓
Analgesia
Sedation
Mechanical ventilation
Hypothermia

The difference between the absolute value of $ScvO_2$ and SvO_2 changes under conditions of shock [6]. In septic shock $ScvO_2$ often exceeds SvO_2 by about 8% [7]. During cardiogenic or hypovolemic shock mesenteric and renal blood flow decreases followed by an increase in O_2 extraction in these organs. In septic shock regional O_2 consumption of the gastrointestinal tract and hence regional O_2 extraction increases despite elevated regional blood flows [8]. On the other hand, cerebral blood flow is maintained over some period in shock. This would cause a delayed drop of $ScvO_2$ in comparison to SvO_2, and the correlation between these two parameters would worsen. Some authors therefore argued that $ScvO_2$ cannot be used as surrogate for SvO_2 under conditions of circulatory shock [9].

However, changes in SvO_2 are closely mirrored by changes in $ScvO_2$ under experimental [10] and clinical conditions [7] despite a variable difference between these two variables. This may explain why Rivers et al. [11] were able to use $ScvO_2$ higher than 70% in addition to conventional hemodynamic parameters as therapeutic endpoint for hemodynamic resuscitation to improve outcome in patients with severe sepsis and septic shock. From a physiological point of view, SvO_2 monitoring for "early goal directed therapy" should provide similar re-

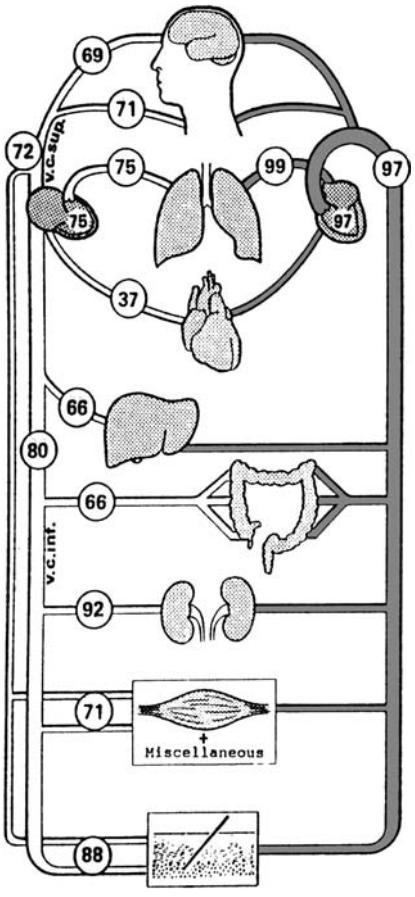

Fig. 1 Arterial and venous oxygen saturations in various vascular regions [2]

sults. Given the fact that $ScvO_2$ exceeds SvO_2 on average by 8% in patients with septic shock, an SvO_2 of about 62–65% should suffice as endpoint for hemodynamic resuscitation in these conditions, although this has not been tested prospectively. However, the placement of pulmonary artery catheters and the potentially higher risk of this should not result in a delay in the start of the resuscitation of critically ill patients.

Venous oximetry can reflect the adequacy of tissue oxygenation only if the tissue is still capable of extracting O_2. In the case of arteriovenous shunting on the microcirculatory level or cell death, SvO_2 and $ScvO_2$ may not decrease or even show elevated values despite severe tissue hypoxia. As demonstrated in patients after prolonged cardiac arrest, venous hyperoxia with an $ScvO_2$ higher than 80% is indicative of impaired oxygen use [12].

Conclusion

Low values of SvO_2 or $ScvO_2$ indicate a mismatch between O_2 delivery and tissue O_2 need. While measurement of SvO_2 requires the insertion of a pulmonary artery catheter, measurement of $ScvO_2$ requires only central venous catheterization. $ScvO_2$ directed early goal-directed therapy improves survival in patients with septic shock who are treated in an emergency department. However, $ScvO_2$ values may differ from SvO_2 values, and this difference varies in direction and magnitude with cardiovascular insufficiency. $ScvO_2$ should not be used alone in the assessment of the cardiocirculatory system but combined with other cardiocirculatory parameters and indicators of organ perfusion such as serum lactate concentration and urine output.

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Fick's principle:

$$\dot{V}O_2 = CO \times Hb \times 1.34 \times (S_aO_2 - S_{\bar{v}}O_2)$$