$SvO_2/ScvO_2$

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Learning Objectives

- The primary goal of hemodynamic optimization is to restore and maintain the balance between oxygen supply (DO_2) and consumption (VO_2) in critically ill patients. There is increasing evidence that patients may benefit from a multimodal individualized approach as compared to protocolized therapy, when predefined hemodynamic goal or goals are targeted. For this purpose, monitoring actual tissue oxygenation/metabolism of a given patient is a very important piece in this hemodynamic puzzle.
- Mixed venous oxygen saturation (SvO_2) and its surrogate, central venous oxygen saturation ($ScvO_2$), are two easily determined blood gas-driven parameters that can mirror changes of the relationship between DO_2 and VO_2.
- This article summarizes the physiological rationale, current knowledge, and some aspects of the clinical applications of SvO_2/$ScvO_2$ and also highlights some of the most important pitfalls of their interpretation at the bedside.

15.1 Introduction

Physical examination plays a very important role in the evaluation of critically ill patients. Certain features such as skin color, capillary refill, mentation, urine output, and pulse quality can tell us a lot about the patient's hemodynamic status. However, some very important features remain hidden even from the most experienced observer or become obvious only at their extremes. These are bicarbonate and lactate levels, hydrogen ion concentrations (i.e., pH), and the balance between oxygen delivery and consumption. Although for detailed monitoring invasive hemodynamic measurements are required, these are not available in every patient. However, arterial and central venous catheters are part of routine monitoring of the intensive care patient, and a simple blood gas measurement can reveal important physiological processes, which cannot be detected otherwise. In the coming chapter, we are going to discuss the rationale and clinical implication of the venous oxygen saturation.

15.2 Physiological Notes

Tissue oxygenation is the net product of oxygen delivery and oxygen consumption, which can be described by the following formulae:

$$DO_2 = CO \times CaO_2$$

$$DO_2 = CO \times \left(Hb \times 1.34 \times SaO_2 + 0.003 \times PaO_2\right)$$

$$VO_2 = CO \times \left(CaO_2 - CvO_2\right)$$

$$VO_2 = CO \times \left[\left(Hb \times 1.34 \times SaO_2 + 0.003 \times PaO_2\right) - \left(Hb \times 1.34 \times SvO_2 + 0.003 \times PvO_2\right)\right]$$

$$\text{Oxygen extraction}\left(O_2ER\right) = VO_2 / DO_2$$

$$O_2ER : \left(SaO_2 - SvO_2\right) / SaO_2$$

If SaO_2 is taken as 1, as under normal circumstances the hemoglobin is almost fully saturated with oxygen, and the other hemodynamic variables are kept constant, then:

$$O_2ER \approx 1 - SvO_2$$

where DO_2 is oxygen delivery; C, cardiac output; Hb, hemoglobin; SaO_2, arterial oxygen saturation; PaO_2, partial pressure of oxygen in the arterial blood; CaO_2, arterial oxygen content; VO_2, oxygen consumption; SvO_2, mixed venous oxygen saturation; and CvO_2, mixed venous oxygen content.

Taking a 75 kg healthy adult man when resting, the relationship between DO_2 and VO_2 can be estimated as:

$$\text{Oxygen delivery} : CO = 70\ \text{ml} \times 70\ /\ \text{min} \sim 5000\ \text{ml} / \text{min}$$

$$CaO_2 = (150\ \text{g} / \text{L} \times 1.34\ \text{ml} \times 1.00) + (0.003 \times 100\ \text{mmHg}) \sim 200\ \text{ml} / \text{L}$$

$$DO_2 \sim 1000\ \text{ml} / \text{min}$$

$$\text{Oxygen consumption} : CO = 70\ \text{ml} \times 70 / \text{min} \sim 5000\ \text{ml} / \text{min}$$

$$CvO_2 = (150\ \text{g} / \text{L} \times 1.34\ \text{ml} \times 0.75) + (0.003 \times 40\,\text{mmHg}) \sim 150\ \text{ml} / \text{L}$$

$$VO_2 = 5\ 1 / \text{min} \times (200\ \text{ml} / \text{L} - 150\ \text{ml} / \text{L}) \sim 250\ \text{ml} / \text{min}$$

$$\text{Oxygen extraction} : O_2ER : 250\ \text{ml} / \text{min} / 1000\ \text{ml} / \text{min} \times 100 = 25\%$$

The main difference between the equations of DO_2 and VO_2 is the oxygen content (CaO_2 vs. CvO_2), especially the venous oxygen saturation (this can either be mixed venous, SvO_2, or central venous, $ScvO_2$). Therefore, it can be useful to assess the imbalance between DO_2 and VO_2 in the critically ill. The potential causes of an imbalance between DO_2 and VO_2 and the basic therapeutic interventions are summarized in Fig. 15.1.

15.3 Interpreting Venous Saturations

When DO_2 is decreasing, oxygen consumption can be maintained – due to an increase in O_2ER – for a considerable period of time. However, without intervention, compensatory mechanisms will become exhausted, and beyond that critical point, VO_2 becomes DO_2 dependent (Fig. 15.2). Till this critical point, venous saturations should decrease proportionally to that of DO_2. On the steep part of the curve, cells switch to anaerobic metabolism; hence, lactate production increases. If urgent interventions are delayed, tissue hypoxia and organ dysfunction can develop.

It is important to note that during resuscitation – i.e., on the steep or DO_2-dependent part of the curve – when interventions are applied to increase DO_2, there is also an increase of VO_2; hence, there is little if any change in venous oxygen saturations, which may remain "low" and will only increase dramatically when VO_2 becomes DO_2 independent (i.e., when the patient reaches the flat part of the curve shown in Fig. 15.2).

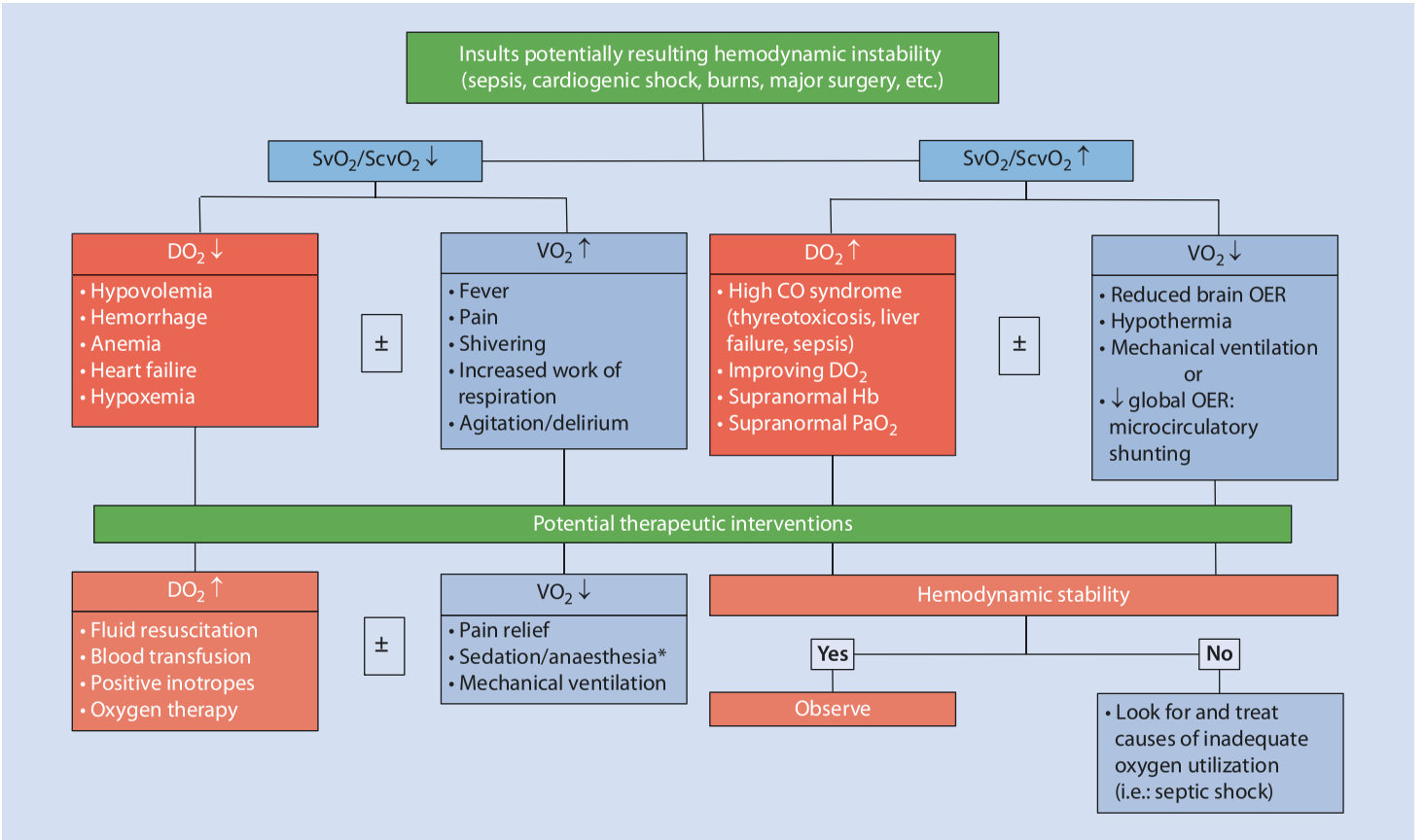

Fig. 15.1 The relationship between venous saturations and DO_2 and VO_2. DO_2 oxygen delivery, VO_2 oxygen consumption, OER oxygen extraction ratio. * – Although sedation can decrease VO_2, however, this should be a delicate option as this may also cause decreased cardiac output; hence, it may worsen the situation by decreasing DO_2. For further explanation, see main text

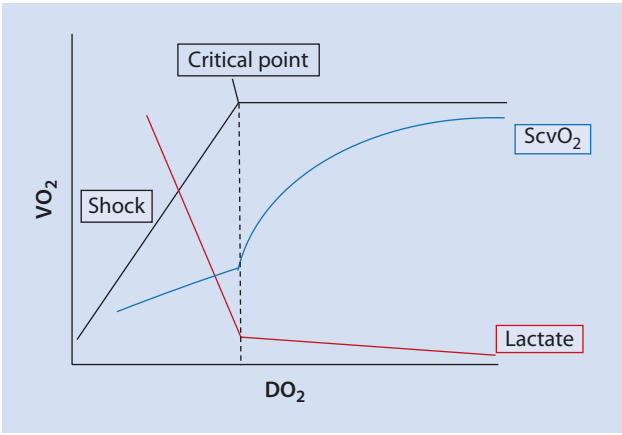

Fig. 15.2 Relationship between oxygen delivery and consumption. DO_2 oxygen delivery, VO_2 oxygen consumption, $ScvO_2$ central venous oxygen saturation. For details, see main text. Of note, this is a simplified diagram to show the rough tendency how these parameters are related. However, due to the irregular redistribution of blood flow as a compensatory mechanism to centralize circulation, certain organs may start anaerobic metabolism earlier than others; therefore, lactate may increase sooner and can be detected in the serum as compared to what is indicated in this figure as the "critical point." Regarding $ScvO_2$, its decrease and increase during resuscitation may not be that dramatic, as it depends on the relationship between VO_2 and DO_2. If VO_2 increases parallel with DO_2, this should cause hardly any change in $ScvO_2$ during resuscitation. However, if DO_2 increases faster than VO_2, then $ScvO_2$ will also increase rapidly

Another problem when interpreting venous saturations is that "high" values can indicate improvement but may also indicate inadequate oxygen uptake [1]. Similar to fluid therapy, this is also reflected in morbidity and mortality, as both high and low venous saturations are accompanied by increased morbidity and mortality (Fig. 15.3). Therefore, despite the high values, further interventions may be required (fluid resuscitation, positive inotropic agents, etc.).

Under these circumstances, when venous oxygen saturations are difficult to interpret, the central venous-to-arterial pCO_2 gap [2] and/or detailed invasive hemodynamic monitoring may serve as complementary tools to assess the hemodynamic status [3]. These will be discussed in other chapters.

15.4 SvO_2 or $ScvO_2$?

Nowadays, measurement of SvO_2 has become a rarity in the everyday clinical practice, because for sampling, a pulmonary artery catheter must be placed, which is a time-consuming, complicated procedure with significant risks [4]. On the contrary, central venous catheters are part of routine monitoring; hence, central venous oxygen saturation ($ScvO_2$) measurement is readily available. It has been shown that oxygen saturation measured in the superior vena cava is a good alternative of SvO_2 [5].

Accurate measurement requires that the tip of the catheter is positioned at the superior vena cava a couple of centimeters above the right atrium. The normal value of $ScvO_2$ ranges between 67% and 77% which is 5–8% higher compared to SvO_2 [6]. Although the absolute values are not interchangeable, their trends show good correlation in various disease states [7].

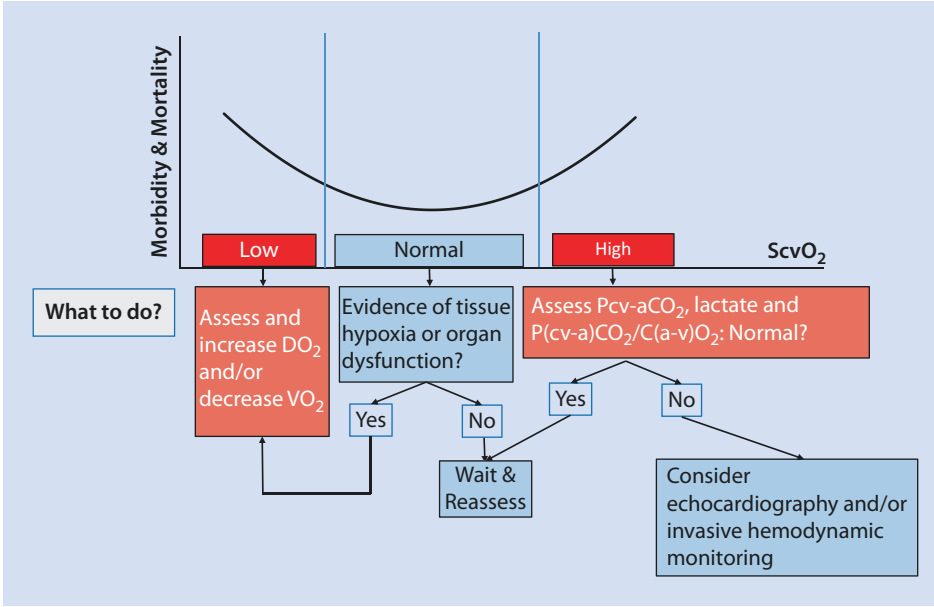

Fig. 15.3 The relationship between $ScvO_2$ and morbidity and mortality. DO_2 oxygen delivery, VO_2 oxygen consumption, $ScvO_2$ central venous oxygen saturation, Pcv-aCO_2 central venous-to-arterial CO_2 gap, C(a-v)O_2 arterial and venous oxygen content difference. This figure indicates that regardless of the actual value of $ScvO_2$, whether it is considered low, normal, or high, careful assessment of the full clinical picture is necessary to best interpret results and to commence appropriate interventions in time

However, as $ScvO_2$ reflects the oxygen consumption mainly of organs draining blood into the superior vena cava, one has to take into account that the biggest consumer of those is the brain. Therefore, during circumstances when brain oxygen uptake is affected (i.e., anesthesia, diffuse brain damage, etc.), $ScvO_2$ may be misleading or at least difficult to interpret.

Nevertheless, by and large these two parameters can be discussed in a similar manner; therefore, to avoid unnecessary citations of both, in the coming paragraphs, we will mainly quote $ScvO_2$, which is the most readily available of the two, unless indicated otherwise.

15.5 The Current Place of $ScvO_2$ in Clinical Practice

15.5.1 $ScvO_2$ in Sepsis and Septic Shock

Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection [8]. Organ dysfunction is most likely the result of inadequate tissue perfusion causing cellular hypoxia. Therefore, treatment strategies that are aimed to restore tissue perfusion by improving the balance between DO_2 and VO_2 may prevent the development of organ dysfunction syndrome and thus improve the outcome of septic patients.

Rivers and colleagues reported in a landmark paper that in patients with severe sepsis, early goal-directed intervention guided by continuous monitoring of $ScvO_2$, central

venous pressure, and mean arterial pressure (MAP), with target values of CVP 8–12 mmHg, MAP > 65 mmHg, and $ScvO_2$ > 70%, reduced mortality from 46.5% to 30.5% at the 28th day [9].

Consequent studies applying early goal-directed therapy (EGDT) with these clinical endpoints suggested that incorporation of $ScvO_2$ in the treatment algorithm and compliance with the algorithm are beneficial in septic patients [10–12]. On the contrary, two large randomized trials, the ProCESS and the ARISE trials, could not show any benefit of the "protocol-based standard therapy" and "usual care" groups. They found no significant difference in 90-day mortality, 1-year mortality, or the need for organ support [13, 14].

The controversy around the usefulness of the "Rivers' EGDT protocol" has been going on for years. Detailed evaluation of these studies is well beyond the scope of this chapter. However, there are some other issues worth discussing in this context.

During the aforementioned studies, "low" $ScvO_2$ was a warning sign that intervention is needed; however, recent data suggest that high $ScvO_2$ values may also have adverse outcomes in septic patients [15]. Due to impaired oxygen utilization, normal or supraphysiological $ScvO_2$ values may thus represent an inability of the cells to extract oxygen or microcirculatory shunting in sepsis [16]. This underscores that some of these patients can be fluid responsive; in other words, their DO_2 can be further increased despite high $ScvO_2$ [1]. In patients with $ScvO_2$ > 70% complimentary parameters, such as elevated venous-to-arterial CO_2 gap (dCO_2) (>6 mmHg), serum lactate levels could help the clinicians to identify tissue hypoxia. In a retrospective analysis, septic patients with physiological $ScvO_2$ and abnormal dCO_2 mortality were significantly higher compared to patients with physiological values (56.1% vs. 16.1%; $p < 0.001$) [17].

15.5.2 $ScvO_2$ in Cardiogenic Shock

Based on the previous physiological notes, it follows a simple logic that acute heart failure which caused low cardiac output, irrespective from the underlying pathophysiology, can cause VO_2/DO_2 imbalance that could be detected by low $ScvO_2$ [18].

Indeed, it has been shown in one of the earliest papers in this field that after myocardial infarction in patients with heart failure and cardiogenic shock, SvO_2 was 43%, while in patients with heart failure without shock, it was 56% compared to patients without heart failure with an SvO_2 of 70% [19].

Treatment effectiveness may also be supported by changes in $ScvO_2$. When cardiogenic shock patients were treated with fluids and inotropes, improvement of DO_2 resulted in an increase in SvO_2 suggesting better tissue oxygenation [20]. It may also be useful in patients with cardiogenic shock requiring the support by intra-aortic balloon counter pulsation. In a study, intra-aortic balloon pump assist ratio was decreased gradually from 1:1 to 1:3. In the weaning failure group decreased support was accompanied by a drop in $ScvO_2$, while it remained constant in the successful group [21].

Even in patients with chronic heart failure, $ScvO_2$ has important predictive values. In these patients, the $ScvO_2$ can be chronically low. However, during acute decompensation, major cardiac events were observed in 81% of patients with $ScvO_2 \leq 60\%$ at 24 h, while it was only 13% in patients with higher $ScvO_2$ [22].

15.5.3 $ScvO_2$ to Predict Successful Extubation

During the weaning procedure, there can be an increase of VO_2 due to the increased respiratory muscle activity and increased alertness. If DO_2 is inadequate, then an imbalance can occur between the VO_2/DO_2. Theoretically, this can be picked up by low or at least decreasing $ScvO_2$ values. In a recent clinical trial, $a > 4\%$ drop in $ScvO_2$ after a 30-min spontaneous breathing trial indicated extubation failure with high sensitivity and specificity [23].

15.5.4 $ScvO_2$ as a Physiological Transfusion Trigger

One of the most common causes of impaired DO_2 in critically ill patients is anemia requiring red blood cell transfusions [24]. Large multicenter trials (TRICC, TRISS) suggest that patients with hemoglobin levels above 10 mg/dl usually do not require transfusion, while red blood cell administration is usually beneficial if the hemoglobin level is below 7 mg/dl [25, 26]. However, there is a gray zone between 7 and 9.5 mg/dl where physicians have to rely on clinical signs like mental status, tachycardia, tachypnea, blood pressure, and diuresis.

In this gray zone, $ScvO_2$ may offer an easily obtainable tool to detect a low hemoglobin-related altered O_2ER and hence may serve as a physiological trigger for blood transfusion [27]. It was found during hemorrhage in animal and human experimental models that $ScvO_2$ may be useful for the identification of patients with occult or ongoing clinically significant blood loss [28]. In a human study, acute isovolemic anemia of hemoglobin of 50 g/l in conscious healthy resting humans did not produce hemodynamic instability, but oxygen imbalance was accompanied by a significant drop in SvO_2 [29]. These results were reinforced by a retrospective analysis of a prospective observational study in which $ScvO_2$ was found to be a good indicator of transfusion [30]. The results of our animal study on isovolemic hemodilution gave further evidence that anemia-induced change in VO_2/DO_2 showed significant negative correlation with changes of $ScvO_2$ [31].

15.6 $ScvO_2$ and Major/High-Risk Surgery

In addition to the acutely ill, the high-risk surgical patients may also develop an imbalance between VO_2 and DO_2 in the perioperative period. Therefore, monitoring $ScvO_2$ may have a rationale during both the intraoperative and postoperative management.

It has been shown that low $ScvO_2$ values are good indicators of complications and poor prognosis in the postoperative period [27]. We reported in a small, single-center prospective randomized study that an $ScvO_2$-assisted intraoperative hemodynamic optimization resulted in less organ dysfunction and better outcome after major abdominal surgery [32]. This was in accord with the results of an earlier single-center study, where patients in the $ScvO_2$-directed group had fewer postoperative complications and had shorter length of hospital stay compared to patients in the control group [28].

However, there are some special considerations when interpreting $ScvO_2$ in the perioperative setting. Firstly, in an anesthetized, mechanically ventilated patient, "normal" values of $ScvO_2$ are 5–10% higher (i.e., 75–80%) than in an awake or sedated intensive care patient or in a normal subject. Secondly, it is important to note that while fluid therapy on the one hand improves cardiac output, on the other hand, it can also cause hemodilution.

In our experimental stroke volume-guided hemorrhage and fluid resuscitation animal model, $ScvO_2$ normalized at the end of resuscitation but returned to a significantly lower level (with a mean of 5%) due to the hemodilution which caused significant drop in hemoglobin levels [33].

Goal-directed therapy is also a controversial issue in surgical patients. However, according to a recent meta-analysis, while goal-directed therapy had no significant effect in the low-risk surgical population, both mortality and morbidity were significantly better in the goal-directed group among the high-risk subgroups [34]. In our view, $ScvO_2$ is an important element of this complex perioperative multimodal monitoring-based concept, including advanced hemodynamic monitoring and assessment of VO_2/DO_2, what we call the individualized, multimodal approach [35].

15.7 Pitfalls of $ScvO_2$

$ScvO_2$ is the net result of the complex physiological and pathophysiological interactions of DO_2 and tissue VO_2. Low values strongly suggest inadequate DO_2; however, in patients with chronic heart failure, chronic anemia, etc., with a "compensated" state, low levels should be considered as "normal" but at least accepted. Not acknowledging this may result in unnecessary and potentially harmful interventions like overzealous fluid resuscitation.

The interpretation of "high" values of $ScvO_2$ is even more challenging. Under physiological circumstances, dissolved oxygen is negligible in DO_2. In an elegant trial on mechanically ventilated ICU patients, after increasing FiO_2 from 40% to 100%, PaO_2 increased from 100 mmHg to almost 400 mmHg: Without any change in cardiac output or hemoglobin, $ScvO_2$ rose from 71% to 84% [36]. Therefore, and this holds true for all the above mentioned examples, relatively stable conditions are desirable for the appropriate assessment. When there are too many changes occurring within a relatively short period time, this can make interpretation of $ScvO_2$ even more difficult.

During circumstances when brain oxygen uptake is affected (i.e., anesthesia, diffuse brain damage, etc.), $ScvO_2$ may be misleading or at least difficult to interpret. Data are lacking, but for these special situations, multimodal monitoring of depth of anesthesia (bispectral index, entropy) and brain oxygen consumption (near-infrared spectroscopy) may be useful and also another step to individualize our treatment for the given patient's actual needs..

Practical Implications

Venous oxygen saturation can be determined from either obtaining blood from the pulmonary artery (SvO_2) or from the superior vena cava ($ScvO_2$). Both can provide useful information about the balance between VO_2 and DO_2 and may also help monitoring the effectiveness of hemodynamic stabilization.

1. In sepsis, impaired oxygen utilization can result in normal or supraphysiological $ScvO_2$ values, which may represent the inability of cells to extract oxygen most likely due to microcirculatory shunting [16]. In the complex pathology of sepsis, treating one single parameter – Let it be $ScvO_2$, lactate, MAP, cardiac output, or else – Can certainly be misleading. Putting easily obtainable clinical and laboratory data including arterial and venous blood gas-driven parameters into context may help to recognize oxygen debt early and may also help to identify those patients

who will require advanced invasive hemodynamic monitoring [3]. This also forms the basis of multimodal, individualized patient management.
2. It has been shown by several studies that in acute left ventricular failure, low SvO_2/$ScvO_2$ is an important sign of severe imbalance in the VO_2/DO_2 relationship, and this parameter also has an important prognostic value [19, 22].
3. Following the changes of $SvO_2/ScvO_2$ over time may be used for weaning patients from cardiac support both pharmacological and assist devices [21], and during spontaneous breathing trials, changes may also provide a good prognosticating factor for extubation success or failure [23].
4. In otherwise stable but anemic patients, $SvO_2/ScvO_2$ may serve as physiologic transfusion trigger [30, 31], although no precise recommendation can be made.
5. In high-risk surgical patients, intraoperative evaluation of $ScvO_2$ can be a very useful tool both for diagnosing and monitoring VO_2/DO_2 imbalance as described in other clinical scenarios, as part of the multimodal monitoring approach [35].

Conclusion

Assessing oxygen consumption requires detailed hemodynamic assessment, which is not always feasible. Measurement of venous oxygen saturations – especially $ScvO_2$ – may serve as a simple, easily and readily available tool for assessing oxygen debt at the bedside. When interpreting the cellular well-being of the high-risk intensive care or surgical patient, $ScvO_2$ can play a very useful role. On its own it can be an important alarming signal of inadequate oxygen delivery, but to see the full picture, it should be incorporated into the complex of the hemodynamic puzzle.

Take-Home Messages

- Venous oxygen saturations are important tools to assess VO_2/DO_2 at the bedside.
- $ScvO_2$ is an easily obtainable and useful alternative of SvO_2.
- Low venous saturations should be considered as an important alarming signal of VO_2/DO_2 imbalance, and causes of low DO_2 – Such as hypovolemia, heart failure, bleeding, anemia, and hypoxemia – Should be looked for.
- High or even normal venous saturations should be interpreted with caution especially in patients who require moderate or high level of hemodynamic support, as they may indicate impaired oxygen uptake.
- In general, but especially under circumstances when interpretation of venous saturation is not straightforward, instead of targeting a given value of SvO_2/$ScvO_2$ (i.e., 65–70%), complimentary parameters, such as venous-to-arterial CO_2 gap, lactate levels, echocardiography, and/or invasive hemodynamic monitoring provided indices, should be put into context in order to individualize hemodynamic support.

15.8 Case Studies

Clinical Case 1

A 35-year-old man suffered acute myocardial infarction. During percutaneous coronary angioplasty, he developed cardiogenic shock and required continuous infusion of norepinephrine (NE) and endotracheal intubation. At the end of the intervention, due to the persistent shock, intra-aortic balloon pump (IABP) was placed to support coronary flow. On arrival to the ICU, he required 75 µg/min NE to maintain a blood pressure of 98/51(73) mmHg. He was ventilated at 60% FiO_2, 10 of PEEP, in BiPAP mode.

The IABP was set to a 1:1 support mode and a control arterial and central venous blood gases were taken.

	Arterial blood gas	Central venous blood gas
pH	7.41	7.35
pCO_2 (mmHg)	42	53 ($Pcv\text{-}aCO_2$-gap: 11)
pO_2 (mmHg)	103	46
BE (mmol/L)	1.3	–
HCO_3 (mmol/L)	26.0	–
SO_2 (%)	98	77
Lactate (mmol/L)	1.4	1.3

These results indicate remarkable oxygenation, ventilation, and acid-base homeostasis, as far as pH, HCO_3, and lactate are concerned. However, central venous blood gas results, taken at the same time, revealed a completely different picture.

$ScvO_2$ could be considered as "normal" or "high." However, the elevated CO_2 gap suggests that cardiac output may be low. An echocardiography was performed, which revealed poor left ventricular function (EF, 35%) with dilated ventricles (135 mL). The IABP was then stopped for 5 min and blood gases were repeated.

	Arterial blood gas	Central venous blood gas
pH	7.39	7.36
pCO_2 (mmHg)	44	51 ($Pcv\text{-}aCO_2$-gap: 7)
pO_2 (mmHg)	87	46
BE (mmol/L)	10.8	–
HCO_3 (mmol/L)	26.0	–
SO_2 (%)	97	81
Lactate (mmol/L)	1.3	1.3

Interpretation

Stopping the IABP for 5 min caused an increase in $ScvO_2$ by 4% and a decrease in CO_2 gap to 7 mmHg, indicating a possible improvement in cardiac output. For more information, invasive hemodynamic monitoring was commenced with transpulmonary thermodilution, which revealed elevated end-diastolic volume (GEDVI) of 1043 ml/m^2 (normal, 600–800 ml/m^2) and increased

extravascular lung water (EVLWI) of 21 ml/kg (normal, less than 10 ml/kg), indicating gross fluid overload; hence, fluid removal was decided, initially with furosemide, and then later with continuous veno-venous hemofiltration.

Conclusion

Arterial blood gas analysis on its own is not enough to assess the hemodynamic situation – in fact it may show a false-positive picture – unless there is already severe metabolic acidosis with low pH, HCO_3, and high lactate levels. Including the central venous blood gas results in the assessment, an early warning sign was revealed indicating that the patient is still unstable, and further information and intervention may be required.

Clinical Case 2

An 83-year-old woman with urinary tract infection was treated on a medical ward and was asked to be reviewed due to respiratory distress and hypotension. On assessment she looked frail, she was tachypneic (30/min), and her blood pressure was 90/40(57) mmHg. The attending ICU resident immediately started oxygen supplementation via face mask and after inserting a large bore (14G) peripheral venous catheter ordered a fluid bolus of 500 mL balanced crystalloid solution to be infused. At the same time, an arterial blood gas was sent to the ICU.

	Arterial blood gas
pH	7.19
pCO_2 (mmHg)	28
pO_2 (mmHg)	64
BE (mmol/L)	−16.4
HCO_3 (mmol/L)	10.5
SO_2 (%)	88
Lactate (mmol/L)	6.9

Based on these results, the patient was immediately transferred to the ICU.

By the time of arrival, her blood pressure and oxygenation already improved, and she felt better in general. An indwelling arterial catheter was inserted into the left radial artery, and another blood gas was taken. In the meantime she received another bolus of 500 mL crystalloid.

	Arterial blood gas
pH	7.27
pCO_2 (mmHg)	27
pO_2 (mmHg)	92
BE (mmol/L)	−13.1
HCO_3 (mmol/L)	12.5
SO_2 (%)	96
Lactate (mmol/L)	3.7

These results indicate improvement, but metabolic acidosis is still present; hence, a central venous catheter was inserted into the right internal jugular vein, and in the meantime a transthoracic echocardiography was also performed. The latter revealed good ventricular function and small ventricular diameters; therefore, fluid administration was continued, and another 500 mL of bolus crystalloid was administered. The patient's blood pressure hasn't changed and remained anuric; hence, norepinephrine was also commenced into a peripheral vein at a rate of 5 µg/min. After inserting the central venous catheter, arterial and central venous blood gases were taken at the same time.

	Arterial blood gas	Central venous blood gas
pH	7.38	7.34
pCO_2 (mmHg)	39	52 (Pcv-aCO_2-gap: 13)
pO_2 (mmHg)	130	25
BE (mmol/L)	−5.1	–
HCO_3 (mmol/L)	20.5	–
SO_2 (%)	98	49
Lactate (mmol/L)	2.4	2.2

Interpretation

According to these results, there is still an imbalance between VO_2 and DO_2 as indicated by low $ScvO_2$, and the grossly elevated CO_2 gap also suggests the inadequacy of flow (cardiac output). Therefore, fluid resuscitation was continued, and after another two boluses of 500 mL of crystalloid, the patient's condition eventually improved, and both macrohemodynamics (blood pressure, urine output) and blood gases normalized.

Conclusion

Despite dramatic improvement in arterial blood gases, lactate, respiratory, and macrohemodynamic indices, central venous blood gas results revealed that serious hemodynamic instability is still present indicated by very low $ScvO_2$ and very high CO_2 gap. Putting both blood gases into context helped the decision to continue fluid resuscitation, which ended with positive results; hence, advanced monitoring and further intervention became unnecessary.

Clinical Case 3

A 67-year-old man required acute surgery due to a perforated colon diverticulum. From his previous medical history, controlled hypertension and mild ischemic heart disease are worth mentioning. In the postoperative period, he required some vasopressor support for 24 h, but by day 3 his condition improved, he felt well, he was without any pain, all vital signs were stable, and he started eating and drinking the day before; hence, he was considered as ready to be discharged. The only abnormal finding was a hemoglobin of 7.2 g/dL. These were his blood gases:

	Arterial blood gas	Central venous blood gas
pH	7.34	7.32
pCO_2 (mmHg)	46	52 (Pcv-aCO_2-gap: 6)
pO_2 (mmHg)	84	43

	Arterial blood gas	Central venous blood gas
BE (mmol/L)	−0.6	–
HCO_3 (mmol/L)	26.5	–
SO_2 (%)	98	73
Lactate (mmol/L)	1.9	2.0

Interpretation

Based on the stable macrocirculation, well-established oral intake of food and drinks, the normal $ScvO_2$, lactate, and CO_2 gap, we decided not to transfuse this patient. He was then discharged, and following him up, his hemoglobin started to increase gradually and did not require blood transfusion during his hospital stay.

Conclusion

Although most transfusion guidelines would recommend transfusing an elderly patient with previous medical history of ischemic heart disease, especially in the early postoperative period with a hemoglobin of 7.2 g/dL, but putting all available data into context, there was no evidence that this degree of anemia caused any instability to this particular patient; therefore, transfusion had no physiological indication; hence, it was put on hold, and transfusion – with all its potential side effects – was eventually avoided.

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