CHAPTER 24
Venous Oximetry

COLIN L. DOYLE, MICHAEL S. HAYASHI, EMANUEL P. RIVERS, RONNY OTERO, JOSEPH A. GARCIA, KONRAD REINHART, ARTURO SUAREZ, and MIHAE YU

INTRODUCTION

During initial management of the critically ill patient, physiologic variables such as blood pressure (BP), heart rate (HR), urine output (UOP), cardiac filling pressures, and cardiac output (CO) are used to guide resuscitative efforts. Despite normalization of these variables, significant imbalances between systemic oxygen delivery (DO₂) and demand result in decreases in central (ScvO₂) and mixed (SvO₂) venous O₂ saturation levels and global tissue hypoxia (1–3). This global tissue hypoxia, if left untreated, leads to anaerobic metabolism, lactate production, and O₂ debt. The magnitude and duration of O₂ debt have been implicated in the development of the inflammatory response, multisystem organ failure (MSOF), and increased mortality (4–8). Early restoration of global tissue normoxia, aided by venous O₂ saturation monitoring has resulted in a reduction in inflammation, morbidity, mortality, and health care resource consumption (9,10). Herein we will review the physiologic principles and clinical utility of (SvO₂) in the management of the critically ill patient.

MAJOR PROBLEMS

Patient Selection for Continuous Venous Oximetry

Continuous venous oximetry is likely most useful in patients at greatest risk of developing global tissue hypoxia. This includes patients with significant acute or chronic cardiopulmonary disease undergoing major surgical procedures and undergoing therapy that may interfere with their ability to increase O₂ delivery during times of stress. It is also useful in patients who require hemodynamic and ventilator support (11).

Goals of Venous Oximetry Monitoring

The goals of continuous venous oximetry vary depending on the initial condition of the patient. Venous oximetry can be used as an end point in early resuscitation, or as a monitoring device for high-risk patients at risk for developing global tissue hypoxia. The common goal is to ensure a balance between systemic O₂ delivery and demand. A stable and normal value for the SVO₂ may indicate that further measurements are unnecessary. However, an abrupt decrease in SVO₂ becomes a warning that investigation of oxygen delivery (comprised of CO, arterial oxygen saturation [SaO₂], and hemoglobin [Hgb] concentration), and systemic oxygen consumption VO₂ is needed so that specific therapy may be directed toward the underlying disorder (Table 24.1) (12).

ESSENTIAL POINTS

1. A normal SvO₂ range is 65% to 75% (0.65 to 0.75) and suggests that the O₂ supply is meeting the demands of the tissues, though some have suggested ranges 2% to 3% higher (13). Since SVo₂ is a global value, a normal value does not guarantee the absence of ischemic tissues.

2. There are four determinants of SvO₂: CO, Hgb concentration, CaO₂, and VO₂. In the critically ill patient, an abrupt change in SvO₂ indicates that a change in O₂ transport–demand balance has occurred but does not identify which determinant has changed.

3. A decrease in SVO₂ may be caused by a decrease in CO, Hgb concentration, or CaO₂, or an increase in VO₂.

4. An increase in SVO₂ is more difficult to interpret. It may indicate distal migration of the catheter which is easy to check by determining catheter position (see below). Patients may have a high CO, VO₂, or CaO₂, especially during anesthesia or mechanical ventilation. If this is associated with persistent elevation of lactate levels, it is an ominous sign. In patients with cirrhosis, sepsis, and peripheral shunts, an abnormal distribution of peripheral blood flow may impair oxygen uptake so that SVO₂ remains high. In cirrhosis, there is pathologic shunting between the arterial and venous systems in the liver causing a high CO and high SVO₂.

5. Pulse oximetry and mixed venous oximetry can be combined into a tool of continuous cardiac and pulmonary monitoring.

6. The difference between arterial and venous saturation (SaO₂–SvO₂) is an estimation of arterial and venous O₂ content difference, and is inversely proportional to CO and directly proportional to O₂ consumption.

7. The ventilation/perfusion index (V/Q) gives an estimate of intrapulmonary shunt. Using saturation as an inference of O₂ content, respiratory dysfunction (V/Q) can be estimated from the equation (1 – SaO₂)/(1 – SvO₂).

ESSENTIAL TROUBLESHOOTING PROCEDURES

1. Continuous SVO₂ measurements may drift and require daily calibration using laboratory co-oximetry.
2. Calibration should also be verified anytime the optical module is disconnected, or whenever the measurement is thought to be erroneous.

3. Distal migration of the pulmonary artery catheter (PAC) tip may cause a higher $Sv\_O2$ reading due to proximity to pulmonary capillary blood, which is approximately 100% saturated. The catheter should be positioned in a large enough segment of the pulmonary artery to require no more than 1.25 mL of air in the balloon to occlude that segment.

4. Infusion of fluids or blood through the distal port of the catheter may alter the light signal and the reading.

5. Decreased light intensity signal or damping of the pulmonary artery (PA) tracing may indicate migration distally or fibrin around the optic bundles. If irrigation of the catheter does not correct the artifact, the catheter should be withdrawn and repositioned.

6. A change in $Sv\_O2$ of greater than 10% in either direction requires investigation.

### INITIAL THERAPY

1. If $Sv\_O2$ is low in association with a low CO, optimization procedures with fluids or inotropic agents should occur immediately. When titrating inotropic infusions, a lack of response ($Sv\_O2$ does not increase) suggests inadequate therapy. CO should be reassessed and treatment augmented.

2. In cases of respiratory dysfunction, arterial saturation ($Sa\_O2$) should respond to therapies such as increased fraction of inspired oxygen ($FiO2$) and positive end-expiratory pressure (PEEP) within 8 to 10 minutes. If $Sa\_O2$ does not increase or if $Sv\_O2$ decreases, either respiratory therapy is ineffective or CO may be compromised.

3. After improvement in respiratory function, if the patient is receiving a high $FiO2$, the $FiO2$ may be decreased every 10 to 20 minutes if arterial and venous saturation remain stable. Increased difference in ($Sa\_O2 - Sv\_O2$) usually correlates with a sudden decrease in CO.

4. A decrease in the arterial-venous oxygen concentration difference ($Sa\_O2 – Sv\_O2$) in response to measure to alter CO indicates a successful intervention.

---

### PHYSIOLOGY OF OXYGEN TRANSPORT

The process of O2 transport includes loading O2 into the red blood cells (hemoglobin) and delivering it to the tissue by the heart (CO), as well as utilization of the O2 in the periphery and the return of deoxygenated blood to the right side of the heart. Several terms must be defined to understand the components of O2 transport (absolute values should be indexed to body surface area):

- **Oxygen delivery ($DO2$)** is the volume of oxygen delivered (mL/min) from the left ventricle each minute:

  $$DO2 = CO \times CaO2 \times 10$$

  Arterial content of oxygen ($CaO2$) is the mL of O2 in 100 mL of arterial blood:

  $$CaO2 = (Hgb \times 1.34 + 1.39 mL O2/g of Hgb \times SaO2) + (0.0031 \times PaO2)$$

  Mixed venous content of oxygen ($CvO2$) is mL of O2 in 100 mL of mixed venous blood:

  $$CvO2 = (Hgb \times 1.34 + 1.39 mL O2/g of Hgb \times SvO2) + (0.0031 \times PvO2)$$

  where $PvO2$ is the mixed pulmonary venous oxygen partial pressure.

- **Oxygen demand is the cellular O2 requirement to avoid anaerobic metabolism. Oxygen demand is the amount of O2 required by the body tissues to function under conditions of aerobic metabolism. Because $O2$ demand is determined at the tissue level, it is difficult to quantify clinically.**

- **Oxygen consumption ($VO2$)** is the amount of O2 consumed by the tissue, usually calculated by the Fick equation (Table 24.2):

  $$VO2 = (CaO2 - CvO2) \times CO \times 10$$
230

SECTION 3  MONITORING

TABLE 24.2 Derivation of $S\nu_O_2$ from Fick Equation

<table>
<thead>
<tr>
<th>Step</th>
<th>Equation</th>
<th>Simplification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>$\nu_O_2 = C(a - \nu_O_2) \times CO \times 10$</td>
<td>Fick equation</td>
</tr>
<tr>
<td>2</td>
<td>$\nu_O_2/(CO \times 10) = C(a - \nu_O_2)$</td>
<td>Divide by CO $\times 10$</td>
</tr>
<tr>
<td>3</td>
<td>$\nu_O_2/(CO \times 10) = C(a - \nu_O_2) - CV_O_2$</td>
<td>Definition of C(a - $\nu_O_2$)</td>
</tr>
<tr>
<td>4</td>
<td>$\nu_O_2/(CO \times 10) - C(a - \nu_O_2) = CV_O_2$</td>
<td>Subtract CO $\times 10$</td>
</tr>
<tr>
<td>5</td>
<td>$C(a - \nu_O_2) = CV_O_2$</td>
<td>Definition of $CV_O_2$</td>
</tr>
<tr>
<td>6</td>
<td>$\nu_O_2/(CO \times 10) - C(a - \nu_O_2) = -CV_O_2$</td>
<td>Subtract $C(a - \nu_O_2)$</td>
</tr>
<tr>
<td>7</td>
<td>$CV_O_2 = C(a - \nu_O_2)/(CO \times 10 \times C(a - \nu_O_2))$</td>
<td>Multiply by $-1$</td>
</tr>
<tr>
<td>8</td>
<td>$S\nu_O_2 = 1 - \nu_O_2/D\nu_O_2$</td>
<td>Definition of $S\nu_O_2$</td>
</tr>
</tbody>
</table>

$CO$, cardiac output.

$\nu_O_2$ is a mechanism by which the body “protects” the $O_2$ demand created at the tissue level. Increased $\nu_O_2$ in early stages of shock is associated with increased survival. Oxygen consumption may increase by increasing CO, widening the arterial–venous $O_2$ content difference, or both. In the normal state, both CO and arterial–venous $O_2$ difference may increase by about threefold, providing a total increase of $\nu_O_2$ during times of stress to about ninefold above the resting state. Normally, $\nu_O_2$ and $O_2$ demands are equal; however, in times of great $O_2$ demand or times in which either CO or arterial–venous $O_2$ content difference cannot increase to meet the $O_2$ demand of the cells, demand may exceed $\nu_O_2$. When this occurs, an $O_2$ debt accumulates and anaerobic metabolism and lactic acidosis ensue (15).

- Oxygen uptake is the measured volume of $O_2$ removed from inspired gas each minute (using indirect calorimetry/metabolic gas monitor). Oxygen uptake differs slightly from $\nu_O_2$ in that the latter is a calculated value (from the Fick equation) and the former is the measured volume of $O_2$ taken up by the patient each minute. Oxygen uptake is measured by analyzing inspired and expired gas concentrations and inspired and expired volumes. Measurement of $O_2$ uptake may be useful for metabolic studies in assessing variations in $\nu_O_2$ as well as determining caloric needs.

- Oxygen utilization coefficient (OUC) or extraction ratio ($O_2ER$) is the fraction of delivered $O_2$ that is consumed:
  \[
  \text{OUC or } O_2ER = \frac{\nu_O_2}{D\nu_O_2}
  \]

  Therefore, the OUC defines the balance between $O_2$ supply (delivery) and demand (consumption) (Fig. 24.1).

- Oxygen transport is the process contributing to $O_2$ delivery and oxygen consumption.

FIGURE 24.1 The physiology of oxygen transport and utilization.

ASSESSMENT OF OXYGEN TRANSPORT BALANCE

Oxygen transport balance may be assessed on several levels. First, examination of the patient may reveal signs of hypoperfusion, including altered mentation, cutaneous hypoperfusion, oliguria, tachycardia, and, when all compensatory systems have failed, hypotension. Unfortunately, these clinical signs are often late, nonspecific, and at times uninterruptible in critically ill patients. A more physiologic approach is to assess the determinants of $O_2$ transport balance individually by using the Fick equation. The arterial–venous $O_2$ content difference may be used to assess the relative balance between CO and $\nu_O_2$. An increase in the arterial–venous $O_2$ content difference indicates that either flow is decreased or consumption is increased.

When the Fick equation is solved for $S\nu_O_2$ (see Table 24.2), it becomes apparent that an inverse linear relation exists between $S\nu_O_2$ and $O_2$ utilization coefficients (11) if $SaO_2$ is maintained constant. $S\nu_O_2$ measured continuously is, therefore, an online indicator of the adequacy of the $O_2$ supply and of the demand in perfused tissues. The determinants of $S\nu_O_2$ are $\nu_O_2$, Hgb, CO, $SaO_2$, and, to a small degree, $PaO_2$. $S\nu_O_2$ represents the flow-weighted average of the venous $O_2$ saturations from all perfused tissues (Fig. 24.2).

Therefore, tissues that have high blood flow but relatively low O₂ extraction (kidney) will have a greater effect on SvO₂ than will tissues with low blood flow, although the O₂ extraction of these tissues may be high (myocardium) (16,17).

The interpretation of SvO₂ requires consistent and intact vasoregulation (5). When vasoregulation is altered—as in sepsis—O₂ uptake may be severely altered, causing a marked increase in SvO₂. Septic patients can have a normal SvO₂ while the hepatic venous saturation can be up to 15% lower (18,19). This reduced O₂ saturation was noted to arise from an increased regional metabolic rate rather than reduced perfusion. Flow-limited regional O₂ consumption may potentially exist despite the presence of a normal SvO₂. Therefore, a normal SvO₂ should not be considered as sole criteria to ensure optimal O₂ delivery in critically ill patients (Fig. 24.3) (20,21).

Although O₂ demand cannot be measured, the relative balance between consumption and demand is best indicated by the presence of excess lactate in the blood. Lactic acidosis implies that demand exceeds consumption, or O₂ supply dependency, and anaerobic metabolism is present (Fig. 24.4) (15,22,23). The relative balance between O₂ supply and demand can be assessed by the OUC (1). Calculation of this coefficient, however, requires the measurement of CO, Hgb, SaO₂, PaO₂, SvO₂, and PVO₂; the latter, a reflection of both PaO₂ and CO, is a better predictor of hyperlactatemia and death than either arterial PaO₂ or CO alone. A PVO₂ below 28 mmHg is usually associated with hyperlactatemia and increased mortality (24). Blood lactate concentrations greater than 4 mmol/L are unusual in normal and noncritically ill hospitalized patients and warrant concern. In hospitalized, non-ICU, nonhypotensive subjects, as well as in critically ill patients, a blood lactate concentration greater than 4 mmol/L portends a poor prognosis (25). Since serum lactate is a global measurement, a normal lactate is not a guarantee that all tissue beds are adequately perfused.

**ARTERIAL VENOUS OXYGEN CONTENT DIFFERENCE**

From the Fick principle, we learned that CO was equal to O₂ consumption divided by arterial venous O₂ content difference (CaO₂ − CvO₂). Even in the critically ill patient, it is unlikely that Hgb or total body O₂ consumption can change sufficiently minute to minute to affect the calculations. Therefore, (Ca − v)O₂ usually reflects changes in CO. In addition, immediate response to therapy—or lack thereof—can help tailor therapy more precisely and rapidly (26). Since the contribution of dissolved O₂ is minute (0.0031 × PaO₂), and the factor (Hgb × 1.39 mL O₂/g Hgb) occurs in both sides of the equation, (Ca − v)O₂ can be estimated by subtracting the values of pulse oximetry and continuous mixed venous oximetry (SaO₂ − SvO₂).

**INTRAPULMONARY SHUNT**

Although PaO₂ is affected by changes in respiratory function (intrapulmonary shunt), PaO₂ is also affected by changes in CO if there is a moderate intrapulmonary shunt (≥20%). For example, if 20% of CO is not involved with gas exchange—a shunt—and blood goes to the left side of the heart deoxygenated, any decrease in SvO₂ will decrease PaO₂. Thus, although no change in pulmonary function has occurred, a decrease in CO—or even any factor that decreases venous O₂ content—lowers PaO₂ and increases the alveolar-to-arterial O₂ tension gradient (27). This nonpulmonary effect on PaO₂ is important to understand since treatment of intrapulmonary shunt is to increase PEEP, which would be disastrous if low CO was the cause for low PaO₂. The equation for intrapulmonary shunt is as follows:

\[ \frac{\dot{Q}_{sp}}{\dot{Q}_t} = \frac{C_c - C_a}{C_c - C_v} \]

where \( \dot{Q}_{sp}/\dot{Q}_t \) is physiologic shunt (% of cardiac output), Cc is capillary oxygen content, Ca is arterial oxygen content, and Cv is venous oxygen content. We can simplify the shunt equation by ignoring the calculation of Hgb-carried oxygen by dropping (Hgb × 1.39) and substituting saturations of 100% for the pulmonary capillary saturation, pulse oximetry for arterial content, and mixed venous oximetry for venous content. The entire equation for pulmonary capillary content can be replaced by the term 1 (or 100% Hgb saturation). Because we have already substituted Sa for arterial content and Sv for venous content, this estimation of physiologic shunt (the \( \dot{V}/\dot{Q}_I \)) can be represented by Eq. [28]:

\[ \dot{V}/\dot{Q}_I = \frac{1 - SaO_2}{1 - SvO_2} \]
For instance, if arterial saturation were 90% (or 0.9) and venous saturation were 60% (or 0.6), the $Q_s/Qt$ calculation would be

$$1 - 0.9 \quad 1 - 0.6 \quad = \quad 0.1 \quad 0.4 \quad = \quad 25\%$$

This estimation does not reflect the severity of respiratory failure as judged by the need to use, potentially, a higher FiO$_2$; thus, the equation needs to specify the patient’s FiO$_2$ to be meaningful.

**THE CONSEQUENCES OF TISSUE HYPOXIA**

When compensatory mechanisms such as increased systemic O$_2$ extraction are exceeded, tissue hypoxia results with pathologic significance not only seen in vitro (4); low SVO$_2$ is associated with the generation of inflammatory mediators and the impairment of mitochondrial O$_2$ use (29). The accumulation of global tissue hypoxia over time leads to O$_2$ deficits; the magnitude and duration of this O$_2$ debt has been associated with the generation of inflammatory biomarkers, morbidity, and mortality (Fig. 24.5) (8,29–33). Monitoring the SVO$_2$, therefore, provides early and continuous data that may be acted upon immediately rather than waiting for laboratory results or markers of tissue hypoxia that signify an insult which has already occurred.

**MONITORING OXYGEN TRANSPORT**

Critically ill patients in the emergency department (ED), operating room (OR), and intensive care units (ICUs) may be grouped into three categories. Category 1 consists of patients requiring intensive observation or monitoring. These patients may have major risk factors or may be admitted because of the nature of their illness or the nature of the therapy they are receiving. Category 2 patients require intensive nursing care and often specialized technology and care facilities to direct therapy for major systemic illness. Category 3 patients need continuous physician intervention for hemodynamic and other instabilities. Continuous venous oximetry may have clinical applications in each of these broad classes of patients. The three major objectives of monitoring critically ill patients are to ensure that the patient is stable, to provide an early warning system regarding untoward events, and to evaluate the efficiency and efficacy of interventions performed.

Category 1 patients undergoing hemodynamic and O$_2$ transport monitoring only because of underlying risk factors, who have a normal and stable SVO$_2$, have an intact balance between O$_2$ supply and demand. Further assessment of CO and arterial and mixed venous blood gas analysis to reach that conclusion can be eliminated, and there is “safety in no (other) numbers.” If the patient becomes unstable as manifested by a decreasing SVO$_2$, the monitoring system will meet the second objective by providing an early warning of the imbalance in O$_2$ supply and demand. In this situation, although an alert has been given, the cause of the O$_2$ transport imbalance is not necessarily clear. The change in SVO$_2$ is sensitive but not specific. In this clinical situation, it may be necessary to measure CO, SaO$_2$, and Hgb. When the cause of the imbalance is identified, specific therapy may be instituted to restore the O$_2$ supply-demand balance. While interventions are applied, the continuous assessment of supply-demand balance may be used to evaluate the efficacy of the intervention with instant feedback. Continuous CO methodology should supplement but not supplant mixed venous oximetry. This is particularly important in critical illness, defined as a nonsteady state, when changes in all elements of O$_2$ transport and use can be expected (33).

**CONTINUOUS MIXED VENOUS MONITORING**

SVO$_2$ can be monitored continuously using infrared oximetry, based upon reflection spectrophotometry. Light is transmitted into the blood, and reflected off red blood cells and read by a photodetector in the receiving fiber-optic bundle (11). The amount of light reflected at different wavelengths varies depending on the concentration of oxyhemoglobin and hemoglobin (Fig. 24.6). The microprocessor uses the relative reflectances to calculate the oxyhemoglobin and total Hgb,
the fraction of which represents $\text{SvO}_2$. The catheter used to measure venous $O_2$ saturation can be a pulmonary artery or a modified central venous catheter.

The continuous oximetry system must be calibrated before use by a co-oximetry measured sample (34). This may be done in vitro by positioning the catheter tip next to a target that reflects the transmitted light in such a manner that the microprocessor can be calibrated. After in vitro calibration, the $O_2$ saturation of the central venous system, right atrium, right ventricle, and PA can be measured while the catheter is being floated into the proper position. These measurements during the insertion of the catheter may be useful to rule out intracardiac left-to-right shunts.

Once the PAC—if this is the tool used—is in proper position, blood may be sampled through the distal port to calibrate or to verify the calibration of the system. The first in vivo calibration is usually done at 24-hours post-PAC insertion. A mixed venous sample is withdrawn and analyzed by laboratory co-oximetry. Blood drawn from the PA should be aspirated slowly (1 mL over 20 seconds) to prevent contamination by the highly oxygenated pulmonary capillary blood. The value obtained by the microprocessor at the time the blood sample is drawn is retained by the system. This may be compared against the value obtained from the laboratory sample, and, if a significant (>2%) difference exists, the instrument may be recalibrated to the laboratory co-oximeter value. The calibration should be verified at any time the optical module is disconnected from the catheter, whenever the measurement is suspected of being erroneous, and every 24 hours to ensure stability of the system.

Because it is crucial that red blood cells be flowing past the tip of the catheter, proper positioning in the PA is necessary. Distal migration of the PA catheter tip is a common source of error. When the catheter tip advances into the distal segments of the PA, a high or increased $\text{SvO}_2$, a decreased light intensity signal, or damping of the PA tracing may become evident. If these signs are encountered, the distal lumen of the catheter should be irrigated with flush solution to remove fibrin on the catheter tip. If the pressure waveform is not restored to a proper PA tracing by irrigation, the catheter should be slowly withdrawn until the PA pressure tracing is restored. At this point, the PAC balloon may be slowly inflated until the pulmonary artery occlusion pressure (PAOP) tracing is observed. If this tracing is not produced by inflation of the balloon to maximum volume (1.5 mL), the catheter should be slowly advanced until an occlusion pressure tracing is observed. At that point, the balloon can be deflated again and then slowly reinflated until a PAOP tracing occurs. The volume required to restore this tracing should be at least 75% of the total capacity of the balloon. Using the maximum balloon volume to attain a PAOP tracing ensures that the catheter is in the proximal section of the PA and is, in fact, a physiologic confirmation of the catheter tip position.

Distal migration of the PAC may cause artifactual high $O_2$ saturation because highly saturated (approximately 100%) pulmonary capillary blood is sampled. The catheter tip may be lodged against a vessel wall or bifurcation, causing an alteration in the light intensity received by the fiber-optic bundles. A low-light intensity alarm must be corrected before the venous saturation measurement is considered reliable or before the system is recalibrated. Large fluctuations in the light intensity signal may indicate that the catheter tip is malpositioned but also may indicate a condition of intravascular volume deficit that allows compression or collapse of the pulmonary vasculature, especially during positive pressure ventilation (35).

### CONTINUOUS CENTRAL VENOUS MONITORING

Early management of the critically ill patient is frequently performed outside the ICU. The time between the onset of critical illness and definitive ICU intervention may be prolonged and have outcome implications (36–38). Measurement of $\text{SvO}_2$ requires placement of a PAC, which may not be feasible early in the resuscitation of adult, pediatric, and neonatal patients. However, central venous assess can be obtained in both ICU and non-ICU settings, making continuous ScvO$_2$ monitoring a convenient surrogate for $\text{SvO}_2$.

Numerous animal and human models have examined the relationship between $\text{SvO}_2$ and ScvO$_2$ obtained from the superior vena cava (SVC) and right atria (RA) (Fig. 24.7). SVC ScvO$_2$ is slightly lower and more accurately reflects $\text{SvO}_2$ when patients are not in shock (39,40). The lower value of ScvO$_2$ in a nonstressed state can be explained by the low $O_2$ extraction of the kidneys which drain into the IVC and contribute to the $\text{SvO}_2$ but not the ScvO$_2$ (41). RA ScvO$_2$ has a better correlation than SVC saturation and is not significantly different from $\text{SvO}_2$ whether in shock or not (39). In patients in shock, a consistent reversal of this relationship occurs, with the ScvO$_2$ being greater than $\text{SvO}_2$. This difference can range from 5% to 18% (39,40,42). Redistribution of blood flow away from the splenic, renal, and mesenteric bed toward the cerebral and coronary circulation, including more desaturated blood (<30%) from the coronary sinus contribute to this observation (39). Thus, ScvO$_2$ will consistently overestimate the true $\text{SvO}_2$ under shock conditions. An interesting variation on this concept was recently demonstrated in liver transplant patients. A study of 30 patients undergoing liver transplant, in which samples from the RA and pulmonary artery were taken at several points during the procedure, found that the central venous and mixed venous $O_2$ saturation measurements were only concordant during the hepatectomy phase of the procedure and differed once the graft was in place. The proposed mechanism behind this finding is that increased VO$_2$ of the...
Pulmonary artery

\[ \text{ScVO}_2 \]

\[ \text{SvO}_2 \]

graft after reperfusion, causes the \( \text{SvO}_2 \) to decrease relative to the \( \text{ScVO}_2 \), which is relatively unaffected by this change (43).

There has been considerable debate regarding whether \( \text{ScVO}_2 \) is a satisfactory substitute for \( \text{SvO}_2 \), particularly in ranges above 65% (44–53). Although the absolute values of \( \text{Scvo}_2 \) and \( \text{SvO}_2 \) differ, studies have shown close and consistent tracking of the two sites across a wide range of hemodynamic conditions (Figs. 24.8 and 24.9), thus making it clinically useful (46,54–66). The clinical utility or value of \( \text{SVO}_2 \)/\( \text{ScVO}_2 \) is in the lower ranges. The presence of a pathologically low \( \text{ScVO}_2 \) value—implying an even lower \( \text{SvO}_2 \)—is more clinically important than whether the values are equal. Goldman et al. (54) found that \( \text{ScVO}_2 \) below 60% showed evidence of heart failure or shock or a combination of the two. Hyperdynamic septic shock ICU patients seldom exhibit \( \text{SvO}_2 \) levels <60% to 65%, which, if present and sustained, are associated with increased mortality (12,67). Studies examining the clinical utility of \( \text{ScVO}_2 \) early in the course of disease presentation routinely encounter values less than 50%, which are considered critical (3,68,69). At these values, venous saturations are actually 5% to 18% lower in the pulmonary artery (39,42) and 15% lower in the splanchnic bed (20). Thus, although not numerically equivalent, these ranges of values have similar pathologic implications (54) and are associated with high mortality (24). Conversely, it has also been reported that patients with an \( \text{ScVO}_2 \) > 90% also had increased mortality (70); this can be explained by cellular injury resulting in lack of oxygen consumption.

The clinical utility of an end point of resuscitation is determined by whether it changes clinical practice and morbidity or mortality. Irrespective of whether the \( \text{ScVO}_2 \) equals \( \text{SvO}_2 \), the presence of a low \( \text{ScVO}_2 \) in early sepsis portends increased mortality and correcting this value by a treatment algorithm (71) improves morbidity and mortality. The concept of the approximately 5% numeric difference between \( \text{SVO}_2 \) and \( \text{ScVO}_2 \) (13) prompted the Surviving Sepsis Campaign to recommend reaching a \( \text{SvO}_2 \) of 65% and/or \( \text{ScVO}_2 \) of 70% goal in the resuscitation portion of its severe sepsis and septic shock bundle (72,73). An \( \text{ScVO}_2 \) value of less than 70% has also been demonstrated to suggest a need for blood transfusion when accompanied by a hemoglobin of less than 10 g/dL and a central venous pressure (CVP) of 8 to 12 mmHg (13).

**INTERPRETATION OF VENOUS OXYGEN SATURATION**

The algorithm is presented in Figure 24.10. Mixed venous \( O_2 \) saturation values within the normal range (65% to 75%) indicate a normal balance between \( O_2 \) supply and demand, provided that vasoregulation is intact and a normal distribution of peripheral blood flow is present. Dysoxia usually develops when \( \text{SvO}_2 \) decreases to 40% to 50%, though it may occur at higher values if oxygen extraction is impaired (13). Values of \( \text{SvO}_2 \) greater than 75% indicate an excess of \( \text{DO}_2 \) over \( \text{VO}_2 \) and are most commonly associated with syndromes of vasoderegulation, such as cirrhosis and sepsis. High values also are seen in states of low \( \text{VO}_2 \)—hypothermia, muscular paralysis, sedation, coma, hypothyroidism, or a combination of these factors—hyperoxygenation, high \( CO \), inability to consume \( O_2 \) and, rarely, cyanide toxicity.

Uncompensated changes in any of the four determinants of \( \text{SvO}_2 \) may result in a decrease in the measured value, but in

---

**FIGURE 24.7** Central versus mixed venous oxygen saturation.

complex, critically ill patients, the correlation between changes in \( \text{SvO}_2 \) and changes in any of the individual determining factors is low (74). In a study of the patients in a surgical ICU, no statistical correlation existed between changes in either \( \text{PaO}_2 \) or \( \text{SaO}_2 \) and \( \text{SvO}_2 \). Although there was a statistically significant correlation between changes in \( \text{SvO}_2 \) and CO and \( \text{DO}_2 \), the coefficients of determination (\( r^2 \)) were too low to allow prediction of \( \text{CO} \), \( \text{VO}_2 \), or \( \text{DO}_2 \), from \( \text{SvO}_2 \). Also, no statistical correlation existed between \( \text{SvO}_2 \) and either arterial–venous \( \text{O}_2 \) content difference or calculated \( \text{VO}_2 \). There was a significant inverse correlation between \( \text{SvO}_2 \) and \( \text{O}_2 \) utilization coefficients, confirming the accuracy of the measurement and the reliability of \( \text{SvO}_2 \) as an estimation of the \( \text{O}_2 \) utilization ratio—as long as \( \text{SaO}_2 \) is near 100%. The determinants of \( \text{SvO}_2 \) are multifactorial, and, in critically ill patients, the degree of compensation for changes in one variable cannot be predicted (74). Patients with chronically impaired \( \text{O}_2 \) transport appear to tolerate very low \( \text{SvO}_2 \) values better than acutely ill patients, presumably due to adaptive changes in the former group. Delayed lactate presentation may be seen in this group of patients (75,76).

It is useful, however, to appreciate the magnitude of change in \( \text{SvO}_2 \) that would occur with an isolated change in any of the individual determinants. The relationship between the variables that contribute to \( \text{SvO}_2 \) may not be linear (77). If no compensatory changes occur in \( \text{VO}_2 \) or \( \text{CO} \), \( \text{Hgb} \) must decrease by almost 50% (13 to 7.5 g/dL) before \( \text{SvO}_2 \) decreases below the lower limit of the normal range (Table 24.3). The \( \text{SvO}_2 \) changes would be even smaller because \( \text{CO} \) should increase in response to the acute anemia. However, if \( \text{CO} \) is fixed because of underlying cardiovascular disease, a decrease in \( \text{Hgb} \) will be reflected by a decrease in \( \text{SvO}_2 \). Similarly, a small change in a patient with a low \( \text{CO} \) will have a greater effect on \( \text{SvO}_2 \) than a larger change in a patient with a high \( \text{CO} \) (77).

The effect of arterial \( \text{O}_2 \) tension on \( \text{SvO}_2 \) in the absence of other compensatory changes is demonstrated in Table 24.4. As long as \( \text{SaO}_2 \) is maintained in a relatively normal range, the direct effect on \( \text{SvO}_2 \) is minimal. However, when there is sufficient arterial hypoxemia to produce arterial desaturation, the \( \text{SvO}_2 \) falls in direct proportion to the change in \( \text{SaO}_2 \). Similarly, changes in \( \text{CO} \) (Table 24.5) and \( \text{VO}_2 \) (Table 24.6) may...
be shown to affect SvO₂, although the magnitude of change in any of these individual parameters does not predict the magnitude of change in SvO₂ because compensatory factors are usually involved. A decrease in SvO₂ greater than 10% is likely to be clinically significant regardless of the initial value. A change from 70% to 60% may be associated with a large fractional change in CO if other factors did not change. On the other hand, a change from 60% to 50% is associated with a much smaller fractional change in CO but in the range of limited O₂ transport reserve and should raise more concern (Table 24.7).

When demand exceeds consumption, anaerobic metabolism must occur, and the eventual result is lactic acidosis. The lactate level, therefore, defines the balance between VO₂ and O₂ demands. An elevated lactate implies either ongoing anaerobic metabolism (shock) or prior anaerobic metabolism and O₂ debt. A normal SvO₂ implies the latter and a low SvO₂, the former, in states of lactic acidosis, except in situations in which unloading cellular uptake or mitochondrial utilization are impaired.

### CLINICAL USES OF MONITORING

SvO₂ values have been used extensively in various clinical scenarios in critically ill patients. These include during and after cardiac arrest (CA) (78,79), in cardiac surgery patients (80), during and after cardiac failure (81), shock (82), acute myocardial infarction (54,84), general medical ICU conditions (85–87), postoperative cardiovascular procedures (87), trauma (88–90), vascular surgery (91,92), septic shock (9,12,67), hypovolemia (93,94), pediatric surgery (81), in neonates (95), lung transplantation (96), liver transplant (43), cardiogenic shock (97,98) and ECMO (99).

### Cardiac Arrest

Management of the CA patient by advanced cardiac life support (ACLS) guidelines include physical examination (i.e., palpation of a pulse) and electrocardiographic monitoring. SvO₂ monitoring during CA has been shown to be a diagnostic and therapeutic adjunct (100–102); CA patients routinely have SvO₂ values of 5% to 20% during cardiopulmonary resuscitation (CPR). Failure to reach an SvO₂ of at least 40% during the management of CA carries a 100% mortality even if the patient has an intermittent measurable blood pressure. These values are consistent with animal models (SvO₂ < 43%) using cardiopulmonary bypass (103). SvO₂ has also been used to confirm the presence or absence of sustainable cardiac activity during electromechanical dissociation (EMD) or a pulseless idioventricular rhythm where over 35% of these patients have been shown to have spontaneous cardiac activity (pseudocEMD) (104). If the SvO₂ is greater than 60% during CPR, return of spontaneous circulation (ROSC) is likely, and the pulse should be frequently rechecked if EMD was present. Between SvO₂ values of 40% and 72%, there is a progressive increase in the rate of ROSC. When an SvO₂ greater than 72% is obtained, ROSC has likely occurred. Continuous SvO₂ monitoring also provides an objective measure to confirm the adequacy or inadequacy of CPR in providing DO₂.

### Postcardiac Arrest Care

In the immediate postresuscitation period, patients are frequently hemodynamically unstable and have a high frequency of rearest. Blood pressure (1,101) may be rendered insensitive in the measurement of CO or DO₂ secondary to high systemic vascular resistance induced by catecholamine therapy. An abrupt or gradual decrease in SvO₂ to less than 40% to 50%, indicates likelihood for rearest. An SvO₂ greater than 60% to 70% indicates hemodynamic stability. A study by Ameloot et al. (105) in postarrest patients who were intubated, placed in a coma, paralyzed, and cooled showed an optimal SvO₂ range of 67% to 72%, with an odds ratio of 8.23 for mortality of patients outside of this range. A sustained extreme elevation of SvO₂ greater than 80%, or venous hyperoxia, in the presence of a low DO₂ and increased lactate levels carries a poor prognosis because it indicates an impairment of systemic O₂ utilization. This has been attributed to long periods of arrest and the use of large doses of vasopressors (106). If this derangement...
evaluated whether maintaining normal levels of SvO₂ in maintaining above-normal levels of O₂ transport. For patients with multiple injuries is more relevant to survival than the immediate postoperative periods. SvO₂ was a valuable predictors of survival and was a helpful parameter to monitor during the resuscitative, operative, and immediate postoperative periods. Studies have shown that vital signs are insensitive end points of resuscitation and outcome predictors in hemorrhage and trauma resuscitation (1,109). Scalea et al. (109) and Kowalenko and colleagues (110) have shown that patients presenting with trauma and hemorrhage required additional resuscitation or surgical procedures if the ScvO₂ remained less than 65%. Kremzar et al. (88) evaluated whether maintaining normal levels of SvO₂ in patients with multiple injuries is more relevant to survival than maintaining above-normal levels of O₂ transport. For patients with multiple injuries, maintaining normal SvO₂ values and increasing DO₂ only if required are more relevant for survival than routine maintenance of above-normal O₂ transport values. In a series of 10 seriously injured patients requiring resuscitation and definitive operative control of hemorrhage, Kzarzarian and Del Guercio (89) found that improvement of the SvO₂ was associated with improved survival. In this study, SvO₂ was a valuable predictors of survival and was a helpful parameter to monitor during the resuscitative, operative, and immediate postoperative periods.

### Severe Sepsis and Septic Shock

SvO₂ in sepsis is commonly referred to as an end point of low impact in clinical decisions because of the common perception that SvO₂ is always increased in septic ICU patients. In septic shock the SvO₂ is more difficult to interpret, as the O₂ demand may exceed VO₂, meaning that SvO₂ will not accurately reflect the relationship between VO₂ and DO₂ (116). Microcirculatory shunting is thought to explain the phenomenon of normal SvO₂ in septic shock with multisystem organ failure from local tissue dysoxia (116). However, there are fundamental issues that render this modality clinically useful when applying it to the early stages of the supply-dependent phase of sepsis (global tissue hypoxia) where saturation is low in both animal (117,118) and human models of sepsis (111). During this phase, SvO₂ is inversely correlated with lactate concentration \((r = -0.87, p < 0.001)\). These data suggest that cellular O₂ utilization is largely maintained during rapidly fatal septic shock (119,120). These findings highlight the importance of early assessment and intervention. Identifying sudden episodes of supply dependency in septic ICU patients, that is, a sudden decrease in SvO₂, has diagnostic and prognostic significance (10,12,67). Previous studies have examined SvO₂-guided goal-directed therapy after ICU admission and have found no outcome benefit in general ICU patients (86). However, in a study evaluating early goal-directed therapy (EGDT) using multiple hemodynamic end points including SvO₂ in the most proximal stages of hospital admission, patients presenting with severe sepsis and septic shock were randomized to 6 hours of EGDT or standard therapy before ICU admission. Both groups were resuscitated to a CVP higher than 8 mmHg and mean arterial pressure (MAP) over 65 mmHg; however, the treatment group was resuscitated to an ScvO₂ above 70% using additional therapies such as red cell transfusion, inotropes, and mechanical ventilation to reach this end point (Fig. 24.11). Over the initial 72 hours, there was a higher ScvO₂, lower lactate, lower base deficit, and higher pH in the EGDT versus the control group indicating more definitive resolution of global tissue hypoxia. Organ dysfunction, vasopressor use, duration of mechanical ventilation, and mortality were significantly reduced (9). This concept of EGDT has been reproduced in multiple studies and is one of the cornerstones of the resuscitation bundle recommended by the Surviving Sepsis Campaign (72). More recent studies have

### Traumatic and Hemorrhagic Shock

The standards of Advanced Trauma Life Support (ATLS) focus on normalization of vital signs (108). Studies have shown that vital signs are insensitive end points of resuscitation and outcome predictors in hemorrhage and trauma resuscitation (1,109). Scalea et al. (109) and Kowalenko and colleagues (110) have shown that patients presenting with trauma and hemorrhage required additional resuscitation or surgical procedures if the ScvO₂ remained less than 65%. Kremzar et al. (88) evaluated whether maintaining normal levels of SvO₂ in patients with multiple injuries is more relevant to survival than maintaining above-normal levels of O₂ transport. For patients with multiple injuries, maintaining normal SvO₂ values and increasing DO₂ only if required are more relevant for survival than routine maintenance of above-normal O₂ transport values. In a series of 10 seriously injured patients requiring resuscitation and definitive operative control of hemorrhage, Kzarzarian and Del Guercio (89) found that improvement of the SvO₂ was associated with improved survival. In this study, SvO₂ was a valuable predictors of survival and was a helpful parameter to monitor during the resuscitative, operative, and immediate postoperative periods.

### Acute and Chronic Heart Failure and Pulmonary Hypertension

Cardiogenic shock is characterized by decreased DO₂, decreased SvO₂, increased O₂ER, and evidence of tissue hypoxia—lactic acidosis and end-organ dysfunction—secondary to acute myocardial dysfunction (97,98). While SvO₂ has been shown to have therapeutic and prognostic utility in patients with acute myocardial infarction (84,98,111,112), prospective outcome studies have not validated its clinical use in this patient population (113). Ander et al. (68) examined patients who presented with decompensated chronic severe heart failure (ejection fraction < 30%) who were stratified into normal and elevated lactate (≥2 mmol/L) groups. There was a significant prevalence of “occult cardiogenic shock” (ScvO₂ 26.4% to 36.8%) in the presence of normal vital signs. Using a goal-oriented approach of preload, afterload, contractility, coronary perfusion, and heart rate optimization, these patients required additional therapy compared to their counterparts with normal lactate levels. ScvO₂ and brain natriuretic peptide (BNP) level predict hemodynamics associated with lower survival rates and may be useful as noninvasive markers of prognosis in epoprostenol-treated pulmonary arterial hypertension (PAH) patients (114).
examined the relationship between SV\textsubscript{O}\textsubscript{2} and fluid responsiveness, and have shown that baseline SV\textsubscript{O}\textsubscript{2} in septic patients is a poor predictor of fluid responsiveness, measured as change in CI or change in SVI after a fluid challenge (116).

**Pulmonary Embolus**

Patients with massive pulmonary embolism (PE) and obstructive shock usually require hemodynamic stabilization, thrombolytics, and mechanical interventions. Krivec and colleagues (121) examined 10 consecutive patients hospitalized in the ICU with obstructive shock following massive PE in a prospective observational study. During hemodynamic optimization and infusion of thrombolytic therapy, heart rate, CVP, mean pulmonary artery pressure, and UOP remained unchanged, but the relative change of SV\textsubscript{O}\textsubscript{2} at hour 1 was higher than the relative changes of all other studied variables \(p < 0.05\). Serum lactate on admission and at 12 hours correlated inversely with SV\textsubscript{O}\textsubscript{2} \(r = -0.855, p < 0.001\). In obstructive shock after massive PE, SV\textsubscript{O}\textsubscript{2} changes more rapidly than other standard hemodynamic variables.

**Respiratory Failure**

In 9 of 13 patients with hypoxemic respiratory failure requiring PEEP, there was a strong correlation \(r = 0.88\) between \(\text{DO}_{2}\) and \(\text{SV}_{\text{O}2}\). Of the four patients not showing a good correlation, two had sepsis and two had nearly normal values of \(\text{SV}_{\text{O}2}\) and \(\text{O}_{2}\) delivery at all levels of PEEP studied. Continuous measurement of \(\text{SV}_{\text{O}2}\) improves monitoring of patients, facilitates titration of respiratory therapies, detects abrupt changes in tissue \(\text{O}_{2}\) consumption, and identifies levels of PEEP associated with greatest \(\text{O}_{2}\) delivery (123).

**Postoperative Thoracic and Cardiac Surgery Patients**

Continuous \(\text{SV}_{\text{O}2}\) monitoring was examined in 19 patients as to its predictive value during the postoperative course after thoracotomy for a time period up to 60 hours. In all but 1 of the 10 patients with \(\text{SV}_{\text{O}2}\) less than 65\% for at least 1 hour, complications occurred. A fall of \(\text{SV}_{\text{O}2}\) greater than 5\% or a value below 60\% predicted a period of hypotension.
in six patients. In two of them, this coincided with a period of ventricular dysrhythmias. In those with SvO2 above 65%, no postoperative complications such as arrhythmias, shock, respiratory dysfunction, or oliguria took place (82). Cardiac surgical patients are at risk of inadequate perioperative O2 delivery caused by extracorporeal circulation and limited cardiovascular reserves (123,124). Four hundred and three elective cardiac surgical patients were enrolled in the study and randomly assigned to either the control or the protocol group. Goals of the protocol group were to maintain SvO2 over 70% and a lactate concentration of 2.0 mmol/L or less from ICU admission and up to 8 hours thereafter. The median hospital stay was shorter in the protocol group (6 vs. 7 days, \( p < 0.05 \)), and patients were discharged faster from the hospital than those in the control group (\( p < 0.05 \)).

Discharge from the ICU was similar between groups (\( p < 0.01 \)). Morbidity was less frequent at the time of hospital discharge in the protocol group (1.1% vs. 6.1%, \( p < 0.01 \)). Venous oximetry has also been shown to have clinical utility in weaning patients from ventricular assist devices (126,127).

There have been efforts to replace SVO2 with ScvO2 in the cardiac surgery population, but these have shown that the two methods are not interchangeable. A study of 15 consecutive patients undergoing cardiopulmonary bypass for cardiac surgery using continuous fiber-optics to obtain 9,267 paired intraoperative and postoperative data points demonstrated that while concordance in the measurements and pattern of changes was seen in some patients, in others significant changes in SVO2 were not accompanied by changes in the ScvO2 (128). These findings suggest that ScvO2 is not a suitable replacement for SVO2 in cardiopulmonary bypass patients.

There may be utility to measuring ScvO2 during cardiac surgery, however. Suehiro et al. (129) looked at 102 patients undergoing cardiac surgery with cardiopulmonary bypass and showed that the discrepancy between SVO2 and ScvO2 intraoperatively was a better predictor of postoperative ICU stay and ventilator dependence than either marker alone, with a difference of 12% indicative of worse outcome. Similarly, a negative ScvO2 – SVO2 gradient on postoperative day 1 in 156 cardiac surgery patients having undergone cardiopulmonary bypass had higher serum lactate levels, longer ischemia times, were older and considered higher operative risk. Patients with a negative gradient preoperatively also had lower SVI (41). The patient's cardiac function may have an impact on the relationship between ScvO2 and SVO2, however. Gasparovic and colleagues (41) studied patients undergoing cardiac surgery with cardiopulmonary bypass and showed that ScvO2 was strongly correlated with SVO2 in patients with a CI above 2 L/min/m2 (\( r^2 = 0.73 \)), but was weakly correlated in patients with a lower CI (\( r^2 = 0.37 \)). Shahbazi et al. (130) demonstrated in 62 patients undergoing cardiac surgery with cardiopulmonary bypass that simultaneous measurements of SVO2 and lactic acid drawn from an arterial line did not correlate, stressing the importance of following SVO2 rather than relying of laboratory markers. Interestingly, this study did show a correlation between SVO2 and ScvO2, though it used far fewer data points than the study by Lequeux and colleagues (128), mentioned above. The relationship between fluid responsiveness and SVO2 in cardiac or vascular surgery patients may differ from that of septic patients, described above. A recent study of patients undergoing cardiac and major vascular surgeries demonstrated a greater response in SVO2 among patients categorized as fluid responders based on increase in CI or SVI in response to a fluid bolus (131).

**Vascular Surgery**

In patients undergoing elective operations for aortic aneurysms (\( n = 25 \)) and aortoiliac occlusive disease (\( n = 6 \)), SVO2 was recorded throughout the operation. In all patients, unclamping the aorta resulted in a marked reduction of mean SVO2, with no change in the CO or SaO2. The unclamping of tube grafts was associated with a significant reduction in arterial pH (\( p < 0.01 \)) and in SVO2 (\( p < 0.001 \)) when compared with unclamping of bifurcation grafts. Despite a longer clamp time, unclamping the second limb of a bifurcation graft resulted in a smaller decrease in SVO2 when compared with that observed after unclamping the first limb (12% vs. 6%, \( p < 0.01 \)). The change in SVO2 after unclamping the second limb was only 2% in aortobifemoral grafts and 9% in aortobifemoral grafts. Reperfusion via extensive pelvic and lumbar collaterals in patients with aortoiliac occlusive disease reduces the degree of SVO2 decrease after aortic unclamping. Monitoring the changes in SVO2 during different types of aortic reconstruction helps to define precisely the physiologic alterations that occur in the course of these operations (91,92).

**Postoperative High-Risk Patients**

ScvO2 and other biochemical, physiologic, and demographic data were prospectively measured for 8 hours after major surgery. Data from 118 patients were analyzed; 123 morbidity episodes occurred in 64 of these patients. The optimal ScvO2 cutoff value for morbidity prediction was 64.4%. In the first hour after surgery, significant reductions in ScvO2 were observed, but there were no significant changes in CI or DO2 during the same period. Significant fluctuations in SVO2 occur in the immediate postoperative period and are not always associated with changes in DO2, suggesting that O2 consumption is also an important determinant of ScvO2. Reductions in ScvO2 are independently associated with postoperative complications (132–135).

**Patients On ECMO**

In patients being placed on extracorporeal membrane oxygenation (ECMO) devices, SVO2 has been shown to have prognostic importance. Preoperative SVO2 is predictive of the mortality of patients being placed on V-A ECMO, with 79.3% being the median SVO2 for survivors and 53.0% the median value for nonsurvivors in an observational study of 80 patients with cardiac failure, including adults and children (99). This finding is intriguing as the mean SVO2 of survivors exceeds the normal physiologic range; in this setting, it may prove useful in predicting who will benefit from such a resource-intensive intervention.

**Positioning Patients and Postural Changes**

The effects of changes in positioning on SVO2 in critically ill patients with a low EF (≤30%) and the contribution of (DO2) and (VO2) variables to the variance in SVO2 were examined.
An experimental two-group repeated-measures design was used to study 42 critically ill patients with an EF of ≤30%. Patients were assigned randomly to one of two position sequences: supine, right lateral, left lateral; or supine, left lateral, right lateral. Data on Sv\textsubscript{O}2 were collected at baseline, each minute after position change for 5 minutes, and at 15 and 25 minutes. A difference in Sv\textsubscript{O}2 among the three positions across time was significantly different \((p < 0.0001)\), with the greatest differences occurring within the first 4 minutes and in the left lateral position. VO\textsubscript{2} accounted for a greater proportion of the variance in Sv\textsubscript{O}2 with position change than did DO\textsubscript{2} (136,137). Similar findings have been noted in Sv\textsubscript{O}2 with orthostatic positioning and its superiority in reflecting central blood volume over CVP (93).

**Neonates and Pediatric Patients**

Sv\textsubscript{O}2 has been shown to be clinically useful in pediatric patients (138). However, the challenges of PAC placement make monitoring of the shock state with Sv\textsubscript{O}2 limited, making Scv\textsubscript{O}2 a convenient surrogate (81,94). In an experimental model of neonatal sepsis, Sv\textsubscript{O}2 significantly correlates with right atrial O\textsubscript{2} saturation \((r^2 = 0.88)\). Animal studies suggest that Scv\textsubscript{O}2 at the RA can be a sure, efficient, and easy alternative for the neonatal patient (139), particularly during therapeutic interventions such as mechanical ventilation and intravascular volume resuscitation (140); studies in patients have been less consistent. Simultaneous Scv\textsubscript{O}2 and Sv\textsubscript{O}2 values in children recovering from open heart surgery show Scv\textsubscript{O}2 is consistently lower than Sv\textsubscript{O}2. This difference may be secondary to residual intracardiac left-to-right shunting of blood or to altered distribution of systemic blood flow. The saturation difference between the two venous samples decreases during postoperative recovery, making a Scv\textsubscript{O}2 blood sample an inadequate substitute for Sv\textsubscript{O}2. Because Scv\textsubscript{O}2 was frequently subnormal while Sv\textsubscript{O}2 was in the normal range, monitoring of Sv\textsubscript{O}2 could not be reliably used to rule out O\textsubscript{2} supply/demand imbalance during the early postoperative period in these patients (138,141). To overcome these clinical inconsistencies, a regression formula was derived:

\[
Sv\textsubscript{O}2 = 3 \times SVC + \frac{HIVC}{4}
\]

where SVC is superior vena cava saturation and HIVC is high inferior vena cava saturation (64).

Validation of the clinical utility of Scv\textsubscript{O}2 in children has the same challenges as in adults. A sepsis trial reported significant survival benefit when Scv\textsubscript{O}2 was added to the pediatric model of septic shock. This study supports current recommendations by the American College of Critical Care Medicine for its use in neonatal and pediatric septic shock (Fig. 24.12) (142).

**FIGURE 24.12** Pediatric advanced life support (PALS). CI, chloride; CVP, central venous pressure; ECMO, extracorporeal membrane oxygenation; MAP, mean arterial pressure; PDE, phosphodiesterase; PICU, pediatric intensive care unit; Scv\textsubscript{O}2, central venous oxygen saturation. (Adapted from Carcillo JA, Fields AI. Clinical practice parameters for hemodynamic support of pediatric and neonatal patients in septic shock. Crit Care Med. 2002;30(6):1365-1378.)
COST-EFFECTIVENESS

Economic analysis of the technology of venous oximetry is complex. Because of its variable use in many clinical situations, the direct association with one single variable to outcome and health care resource consumption is not a simple one. In quantitating the economic impact, one must assess prevention of additional resource use such as venous blood gases and nursing time, hemodynamic life-threatening events, and decreased health care resource consumption through improved morbidity and mortality. Significant reductions in the number of venous blood gas analyses, cardiac output measurement, and charges have been observed (123,143,144). Several studies have suggested that the increased cost of the fiber-optic catheter is not justifiable in terms of cost savings (145,146). However, in the treatment of sepsis and cardiothoracic patients, significant reductions in morbidity, mortality, and health care resource consumption have been observed with goal-directed algorithms using venous oximetry (125,147).

COMBINED VENOUS AND PULSE OXIMETRY

Pulse oximetry and continuous mixed venous oximetry can be combined into a useful tool if we understand the underlying physiology that allows certain inferences to be made as well as the limitations. The two devices together provide the capacity to evaluate simultaneous changes in the patient’s cardiovascular and respiratory systems. Arterial oxygen tension and arterial oxygen saturation are related through the familiar oxyhemoglobin dissociation curve. SaO2 values in the range of 70 to 95 reflect changes in PaO2 and are useful in monitoring cardiorespiratory disease and directing therapy. Large changes in PaO2 (80 to 600 mmHg) can occur with minimum changes in SaO2. To maintain arterial oxygen delivery, we keep SaO2 values of blood. If PaO2 fell to 75 mmHg and SaO2 concomitantly dropped to 95%, Hgb-carried oxygen would be 18.07 times 0.95, or 17.17 mL. The dissolved oxygen would be 17.4 mL in 100 mL of blood. In the first example, total oxygen content was 18.38 mL per 100 mL of blood. If PaO2 fell to 75 mmHg and SaO2 concomitantly dropped to 95%, Hgb-carried oxygen would be 18.07 times 0.95, or 17.17 mL. The dissolved oxygen would be 17.4 mL in 100 mL of blood. In the first example, total oxygen content was 18.38 mL per 100 mL of blood. If the second oxygen content, 17.4 mL, is divided by 18.38 mL, the quotient is 0.95; thus, total oxygen content changed the same amount as did the arterial saturation. We can obtain the same information by comparing changes in SaO2 alone without following either PaO2 or calculating total oxygen content. The same is true for SvO2 and mixed venous oxygen content (28,148,150).

APPLICABILITY

There are many valuable bedside uses for simultaneous oximetry. For instance, if a patient’s respiratory function has improved, high FiO2 may be weaned quickly. We have found that changes can be made every 5 minutes. This contrasts to the usual clinical scenario using blood gases where after a change in FiO2 (15-minute equilibration period), drawing of blood is done. If patients have severely depressed oxygenation, PEEP therapy can be augmented much more rapidly by monitoring SvO2. In the case of cardiovascular collapse associated with low SvO2, the response to blood and other fluid infusions as well as vasoactive drugs can be judged rapidly. If the intervention does not increase SvO2 quickly (within a few minutes), it probably has not been effective. Increased CO may result in increased oxygen consumption without a change in SaO2 minus SvO2. This ability to judge the effectiveness of interventions quickly is certainly attractive and often gratifying to the clinician.

LIMITATIONS AND FUTURE QUESTIONS

In spite of studies questioning the value of ScvO2 in ICU patients (101,141,146,150), there is considerable evidence that ScvO2 may have a beneficial role in the early management of critically ill adults, children, and neonates (95,140). The ability to access this information earlier in the phases of critical illness is now a reality, and further studies are now in progress to confirm that early recognition and treatment of out-of-normal-range ScvO2 values have significant outcome benefit.

CLINICAL EXAMPLES

Case 1

A 75-year-old male victim of a witnessed CA presents to the emergency department. After bystander CPR was performed, emergency medical services (EMS) initiates ACLS guidelines. He was found to be in ventricular fibrillation and was successfully defibrillated into normal sinus rhythm. He is admitted to the ICU.

Vital signs: Blood pressure (BP), 160/80; MAP, 106 mmHg; heart rate (HR), 130 beats per minute; respiratory rate (RR), 16 (bag/valve-mask); temperature, 36.4°C; SaO2, 98% on 100% FiO2; ScvO2, 85%.

Arterial blood gas (ABG) (21%):

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.20</td>
</tr>
<tr>
<td>PaCO2</td>
<td>31</td>
</tr>
<tr>
<td>PaO2</td>
<td>100</td>
</tr>
<tr>
<td>Hemoglobin (Hb)</td>
<td>10.5</td>
</tr>
<tr>
<td>Hematocrit (Hct)</td>
<td>31%</td>
</tr>
<tr>
<td>Platelets</td>
<td>400,000</td>
</tr>
</tbody>
</table>

Complete blood count (CBC): White blood cells (WBC), 15.1; hemoglobin (Hb), 10.5; hematocrit (Hct), 31%; platelets (PLT), 400,000.

PA catheter: CI, 1.2/min/m²; PAOP, 22 cmH2O; CVP, 26 cm/H2O; systemic venous resistance (SVR), 5,600 dynes/s·cm².

<table>
<thead>
<tr>
<th>Baseline</th>
<th>Therapy</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>ScvO2 (CPR) 15%</td>
<td>Nitroglycerin</td>
<td>60%</td>
</tr>
<tr>
<td>SPO2 90%</td>
<td>Rate control</td>
<td>3.2</td>
</tr>
<tr>
<td>Lactate 8.4 (mmol/L)</td>
<td>→</td>
<td>100%</td>
</tr>
<tr>
<td>SaO2 93%</td>
<td>EEP 5</td>
<td>40%</td>
</tr>
<tr>
<td>O2ER 10%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

What’s the Baseline?

This case (Fig. 24.13) illustrates several important elements. Namely, the interpretation of the SvO2 is limited without an
ABG since a near-normal SvO₂ value does not imply normal physiology. The oxygen extraction ratio (O₂ER) \((\alpha O_2 - \dot{V}O_2) \text{ difference/} \text{SaO}_2\) is only 10%. The value of SvO₂ is also confounded by the presence of mild anemia. Hypoxemia and circulatory arrest with resultant hypoperfusion leads to anaerobic metabolism represented by the presence of lactic acidosis.

**What's Happening?**

The O₂ER is very low, and in the setting of CA, can possibly relate to the effects of vasopressors used during ACLS or the cytotoxic damage of global tissue hypoxia and reperfusion. This impairment of systemic O₂ utilization is manifest by the presence of mixed venous hyperoxia. Global tissue hypoxia ensues as a consequence of decreased perfusion and impaired tissue uptake resulting in lactic acidosis (79). It is notable that as treatment progresses with vasodilators, the O₂ER increases to 40% and lactate decreases.

**What's the Interpretation?**

The postresuscitative phase of CA is characterized by a complex array of hemodynamic perturbations (Fig. 24.14). O₂ER can be up to 90% during CA, and the failure to reach a SvO₂ of 40% portends near 100% mortality (79). Once an ROSC is obtained, venous hyperoxia or an impaired O₂ER may be a temporary or permanent issue. The period immediately following multiple doses of vasopressors with ROSC is characterized by elevated circulating catecholamines and is termed the early postarrest phase. If efforts fail to decrease afterload, vasodilate the microcirculation, improve cardiac function to a VO₂ above 90 mL/min/m² within 6 hours after CA and persistent lactic acidosis, death is imminent within 24 hours (79).

Similar scenarios to the early phase of CA characterized by an elevated SvO₂ and lactic acidosis can also be seen with vasopressor-dependent shock, sepsis, severe thiamine deficiency, severe Paget disease, malaria, salicylate toxicity, and cyanide toxicity. The later post-ROSC phase demonstrating low SvO₂ and persistent lactic acidosis is comparable to hepatic failure, sepsis, anemia/hemorrhage, cardiogenic shock, and severe mesenteric ischemia.

**Case 2**

A 66-year-old woman with a history of chronic obstructive lung disease (COPD) presents to the ED with a chief complaint of shortness of breath with fever for the past 4 days. She has had a cough productive of yellowish–greenish sputum and is tachypneic and in obvious respiratory distress.

Vital signs: BP, 140/80; HR, 118; RR, 24; temperature, 38.0°C; SpO₂, 88% on room air, 93% on 2 L/min O₂.

**ED Course:** The patient is noticeably more tachypneic and lethargic, so the patient is ultimately intubated for airway protection. Chest x-ray (CXR) study demonstrates a right lower lobe (RLL) infiltrate, consolidation, and airspace disease.

**Monitoring in the ED:** CVP, 16 cm H₂O; ScvO₂, 44%; lactate, 1.9 mmol/L.

About 1 minute after intubation, SvO₂ monitoring begins to rise; climbing to 58%. The patient is suctioned and copious thick sputum is removed. The patient’s CVP improved to 8 cm

![Figure 24.13 Baseline for Case 1.](image-url)

![Figure 24.14 SVo₂ response during resuscitation. ACLS, advanced cardiac life support; ROSC, return of spontaneous circulation; SvO₂, mixed venous oxygen saturation; VF, ventricular fibrillation. (Adapted from Rivers E, Martin GJ, Smithline H, et al. The clinical implications of continuous central venous oxygen saturation during human CPR. Ann Emerg Med. 1992;21(9):1094–1101.)](image-url)
H₂O after administration of a vasodilator. Repeat lactate reading increases 4.7 mmol/L.

Baseline
ScvO₂ 44%
Lactate 1.9 (mmol/L)
SaO₂ 88%
O₂ER 50%

Therapy
Nitroglycerin intubation

Result
58%
4.7
100%
40%

What's the Baseline?

This patient has hypoxia, respiratory distress, and relatively stable vital signs (Fig. 24.15). The fever and clinical complaint in the presence of three systemic inflammatory response syndrome (SIRS) criteria makes pneumonia a likely inciting condition. The patient also exhibits hyperlactatemia and central venous hypoxia (low ScvO₂).

What's Happening?

The patient has symptoms consistent with pneumonia and hypoxemia with an O₂ER of 50%. This increased O₂ER despite a normal blood pressure with an elevated CVP would ordinarily imply normal or elevated intravascular volume, and in patients with a history of cardiac dysfunction, could lead the clinician to inappropriately administer a diuretic. In this case, a vasodilator was more appropriate to improve CO by reduction of afterload. Patients with longstanding cardiopulmonary disease may have low venous saturation with normal lactate until they become delivery dependent. These have been characterized as metabolic hibernators (76).

The presence of SIRS criteria should prompt the clinician to consider obtaining a lactate level to stratify the severity of her condition. In certain patients who do not present initially with an elevated lactate, their history of concurrent medical conditions can create a state of ischemic preconditioning, also termed metabolic hibernation. This early recognition and treatment of the hypoperfused state was originally described by Rivers et al. where a protocolized approach to severe sepsis significantly improved morbidity and mortality. Similar hemodynamic conditions to this patient's initial presentation include hypothermia, a regional hypoperfused state, or congestive heart failure/ cardiopulmonary disease.

Case 3

A 60-year-old male patient was brought to the ED from an assisted-living facility with a chief complaint of change in mental status. The patient has a past medical history significant for cerebral vascular accident (CVA), hypertension, schizophrenia, and diabetes. The patient was found slumped on a park bench. Initially, the patient is nonverbal and presents with the following vital signs: BP, 110/40; HR, 120; RR, 24; temperature, 32°C; SaO₂, 96% on 2L O₂; Glasgow coma scale score, 11.

Physical examination: Patient receives 1-L bolus of isotonic crystalloid with mild increase in BP. The patient is taken to the monitored area of the ED because the nurse notices the patient...
is very slow to respond. The patient's bedside glucose is <50 mg/dL. The patient is given an amp of 50% dextrose. The patient's mental status immediately improves.

**Labs:** Na, 158; K, 5.2; Cl, 100; CO₂, 24; BUN, 90; creatinine, 1.8; glucose, 44; β-hydroxybutyrate, 8.0.

**ABG:** pH, 7.30; PaCO₂, 44; PaO₂, 100; SaO₂, 96%; HCO₃⁻, 24; lactate, 2.0.

**Hemodynamics:** CVP, 1 cm H₂O; ScvO₂, 72%.

**Hospital Course**

<table>
<thead>
<tr>
<th>Baseline</th>
<th>Therapy</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>SaO₂: 96%</td>
<td>Fluids, thiamine, glucose</td>
<td>100%</td>
</tr>
<tr>
<td>ScvO₂: 72%</td>
<td></td>
<td>70%</td>
</tr>
<tr>
<td>O₂ER: 25%</td>
<td></td>
<td>30%</td>
</tr>
<tr>
<td>Lactate: 2.2% mmol/L</td>
<td></td>
<td>1.2 mmol/L</td>
</tr>
</tbody>
</table>

**What’s the Baseline?**

This patient's mental status is altered, probably due to the combination of hypoglycemia and hypothermia (Fig. 24.16). His initial presentation and hemodynamic measurements indicate severe volume depletion. The patient is maintaining a normal blood pressure but has evidence of progressing hemodynamic instability. Given his history, toxicologic and metabolic derangements may be responsible for his hemodynamic embarrassment.

**What’s Happening?**

The patient is exhibiting evidence of anion gap metabolic acidosis (which may be due to ketonemia [β-hydroxybutyrate] and mild lactic acidosis) as well as abnormal chemistry and blood gas data. His O₂ER is 25%, which is in the normal range. The patient is hypothermic, which may account for the central venous O₂ saturation in the normal range. His mental status may be accounted for by hypoglycemia.

**What’s the Interpretation?**

The patient's extraction ratio may be slightly higher than expected but may be explained by a depressed metabolic rate associated with hypothermia. The near-normal lactate level on presentation may also be explained by a depressed metabolic rate despite the lack of substrate (glucose). The higher-than-expected O₂ER should be noted, and a search for disturbances of O₂ utilization should be considered. Entities that impair the tissues’ ability to utilize O₂ consist of toxicologic and metabolic derangements including chronic thiamine deficiency, cyanide toxicity, and possibly severe acetaminophen toxicity.

**Key Points**

1. The mixed venous oxygen saturation may be represented as 1 – (systemic oxygen consumption/systemic oxygen delivery).
2. An imbalance between systemic oxygen delivery and consumption may result in either abnormally low values of mixed venous or central venous oxygen saturation when delivery is inadequate to meet demand, or abnormally high values when consumption decreases as a result of mitochondrial damage, altered vasoregulation, or with shunting. Both of these are associated with increased mortality.
3. Under normal conditions, central venous oxygen saturation will be higher than mixed venous oxygen saturation, but in a shock state, this relationship is consistently reversed. Regardless of which method is used, gross alterations in these values are ominous signs that require further investigation and/or intervention.
4. When oxygen demand is greater than consumption, a lactic acidosis will occur. This finding in combination with a low mixed venous oxygen saturation suggests a shock state, whereas if the saturation is normal, it implies a prior oxygen debt.
5. Because Hgb and oxygen consumption do not usually change rapidly, the difference between arterial oxygen content and venous oxygen content is usually indicative of a change in cardiac output.
6. Mixed venous oxygenation can be used to calculate the intrapulmonary shunt (1 – arterial saturation)/(1 – mixed venous saturation), which can be treated by increasing PEEP.

**FIGURE 24.16** Baseline for Case 3.
Specific nuances of mixed and central venous oxygen saturation monitoring in different patient populations exist and examples are highlighted in this chapter.

References


