Hypoperfusion is the most common event preceding the onset of multiple organ dysfunction syndrome during trauma resuscitation. Detecting subtle changes in perfusion is crucial to ensure adequate tissue oxygenation and perfusion. Traditional methods of detecting physiological changes include measurements of blood pressure, heart rate, urine output, serum levels of lactate, mixed venous oxygen saturation, and central venous oxygen saturation. Continuous noninvasive monitoring of tissue oxygen saturation in muscle has the potential to indicate severity of shock, detect occult hypoperfusion, guide resuscitation, and be predictive of the need for interventions to prevent multiple organ dysfunction syndrome. Tissue oxygen saturation is being used in emergency departments, trauma rooms, operating rooms, and emergency medical services. Tissue oxygen saturation technology is just as effective as mixed venous oxygen saturation, central venous oxygen saturation, serum lactate, and Stewart approach with strong ion gap, yet tissue oxygen saturation assessment is also a direct, noninvasive microcirculatory measurement of oxygen saturation. (Critical Care Nurse. 2016;36[3]:12-19,70)

Alterations in tissue perfusion and oxygenation at the level of the microcirculation can contribute to the development of organ dysfunction and poor outcomes. Insufficient oxygen to meet cellular demands leads to cellular ischemia, bacterial translocation, sepsis, worsening of shock, organ dysfunction, multiple system organ failure, and death. A balance must be maintained between oxygen delivery ($D_\text{O}_2$) and oxygen consumption ($V_\text{O}_2$) to minimize anaerobic metabolism, maintain normal metabolic processes, and meet the cellular requirements for the body. High baseline oxygen-extraction organs, such as the heart, are at high risk for ischemia if the $V_\text{O}_2$ just meets the oxygen demand. Adequate physiological oxygen reserves need to be available in order to cope with increases in oxygen demand.2

This article has been designated for CE contact hour(s). The evaluation tests your knowledge of the following objectives:
1. Compare mixed venous oxygen saturation, central venous oxygen saturation, serum lactate, Stewart approach with strong ion gap and near-infrared spectroscopy
2. Identify different venues where tissue oxygen monitoring technology is currently being used and the advantages of this technology
3. State 1 limitation of tissue oxygen monitoring technology

To complete evaluation for CE contact hour(s) for test #C1632, visit www.ccnonline.org and click the “CE Articles” button. No CE test fee for AACN members. This test expires on June 1, 2019.

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Adverse changes in oxyhemoglobin and deoxyhemoglobin levels in the peripheral tissues, such as during resuscitation in patients with hemorrhagic shock, indicate that peripheral blood flow is being redistributed to the vital organs. Hypoperfusion is the most common event preceding the onset of multiple organ dysfunction syndrome (MODS) during shock resuscitation. Detecting subtle changes in perfusion is crucial to ensure adequate tissue oxygenation and perfusion. Assessments of blood pressure, heart rate, arterial oxygen saturation, central venous pressure, blood gases, urine output, and hemoglobin concentration are used by the trauma team during resuscitation. However, normal values for these physiological parameters do not rule out tissue hypoxia or imbalances between whole-body oxygen supply and demand. As care providers, nurses must always determine if the tissue DO\(_2\) is sufficient to meet the oxygen demands at the cellular level.

### Venous Oximetry

Mixed venous oxygen saturation (S\(\text{vO}_2\)) is the percentage of oxygen remaining in the venous blood returning from the lower part of the body to the right side of the heart. The normal level of S\(\text{vO}_2\) is approximately 75%. In general, a low or a high S\(\text{vO}_2\) is critical and an early warning sign. Four main causes of low S\(\text{vO}_2\) are an increase in \(\dot{V}_\text{O}_2\), low cardiac output, arterial desaturation, and anemia. Increases in S\(\text{vO}_2\) can occur if blood is being shunted without releasing its oxygen, the uptake of oxygen by the tissues is decreasing, the DO\(_2\) is increasing, or the \(\dot{V}_\text{O}_2\) is decreasing. Adequate calibration and malposition of the catheter can cause an error in S\(\text{vO}_2\) measurements.

Central venous oxygen saturation (S\(\text{cVO}_2\)) is the oxygen saturation of venous blood coming from the head and the upper part of the body. S\(\text{cVO}_2\) is measured by using an invasive central venous catheter placed in the superior vena cava. The superior vena cava drains blood from the head and the upper part of the body to the heart. S\(\text{cVO}_2\) does not provide an accurate estimate of preload or a measurement of contractility.

The normal cardiovascular response to an increase in \(\dot{V}_\text{O}_2\) is to increase oxygen extraction and cardiac output. However, this mechanism has its limits. A decrease in DO\(_2\) can be induced by anemia, hypoxia, hypovolemia, or heart failure. Fever, pain, and stress can also decrease S\(\text{vO}_2\) or S\(\text{cVO}_2\) by increasing the whole-body \(\dot{V}_\text{O}_2\).

A decrease in S\(\text{vO}_2\) and S\(\text{cVO}_2\) indicates an increase in metabolic stress. A decrease in DO\(_2\) due to a decrease in arterial oxygen, cardiac output, or both can cause S\(\text{vO}_2\) and S\(\text{cVO}_2\) to decrease. In addition, S\(\text{vO}_2\) and S\(\text{cVO}_2\) can decrease because DO\(_2\) does not increase to cover the increased \(\dot{V}_\text{O}_2\). Adequate measurement of tissue oxygenation by venous oximetry can occur only if the tissue is still capable of extracting oxygen. During arteriovenous shunting on the microcirculatory level or in cell death, the values of S\(\text{vO}_2\) and S\(\text{cVO}_2\) may not decrease or be elevated.

### Serum Levels of Lactate

When the critical amount of DO\(_2\) is decreased because the amount of oxygen extracted can no longer be increased, tissue hypoxia occurs and an increase in the serum level of lactate may ensue. Increases in the serum level of lactate and in lactate clearance are indicators of the severity of tissue hypoxia and the adequacy of resuscitation from shock. The relationship between lactate and pH exists only at higher lactate levels and therefore should be termed lactate-associated acidosis. Yet, lactate increases and base deficits also have several limitations: overresuscitation can occur because hyperlactatemia may persist for hours after anaerobic metabolism is resolved. The values of lactate and base are not continuous and could provide erroneous clinical assurance when the patient’s actual condition is deteriorating, as in shock, thus allowing less severe cases of hypoperfusion to be overlooked.

In a retrospective observational study, prehospital serum levels of lactate were assessed as a predictor of hypoperfusion is the most common event preceding the onset of MODS during shock resuscitation.
Lactate is a natural byproduct of glycolysis. The normal level of lactate present in the blood in unstressed patients is 0.5 to 1 mmol/L. Hyperlactatemia (without metabolic acidosis) is defined as a persistent, mild to moderate (2-4 mmol/L) increase in the blood level of lactate. Adequate tissue perfusion, intact buffering systems, and adequate tissue oxygenation can occur with hyperlactatemia or lactic acidosis (type B). Type B1 lactic acidosis is associated with systemic diseases such as diabetes mellitus, renal and liver diseases, and malignant neoplasms; type B2 is caused by alcohol (ethanol), epinephrine, thiamine deficiency, biquanides, zidovudine, salicylates, and cocaine; and type B3 is due to inborn errors of metabolism. Types B1, B2, and B3 are among the causes of lactic acidosis when tissue oxygenation is adequate. In the case of type B lactic acidosis, occult tissue hypoperfusion accompanies the primary cause.

Stewart Approach With Strong Ion Gap

Peter Stewart’s approach to acid-base status of body fluids puts water dissociation at the center of the controversial acid-base issue. Mammals are approximately 60% water and follow the acid-base physiological behavior of water, such as the model of water dissociation H₂O ⇌ H⁺ + OH⁻. According to the law of mass action, even a small dissociation change, such as that caused by temperature, can have a marked effect on pH. Stewart designates pH, bicarbonate (HCO₃⁻), carbonate, a single anionic form (A⁻), and hydroxide (OH⁻) as dependent variables (passive). The 3 independent variables that are the heart of many subcellular and molecular processes: membrane acid-base transporters, the mechanism of mitochondrial oxidative phosphorylation, and the functioning of ionophores considered to facilitate proton or HCO₃⁻ transfer along concentration gradients. Calculations are complex; thus, new tools are available for clinicians to enter patients’ acid-base and chemistry data online and receive online support for complex acid-base disorders.

In a study by Gezer et al, the results of 409 arterial blood gas analyses of 90 patients were collected retrospectively and evaluated by using the same blood gas analyzer. HCO₃⁻ level and the anion gap were calculated by using the Stewart method via an online tool. In outcomes in trauma patients. A rotorcraft transported 1168 patients to a level I trauma center during an 18-month period. Each patient’s prehospital serum level of lactate was determined. The primary outcome was in-hospital mortality. Secondary outcomes were emergent surgery and MODS. Covariates included age, sex, prehospital vital signs, and mental status, for which a multivariable logistic regression model was created. Among the patients, 7.4% required surgery, 5.7% experienced MODS, and 5.6% died in the hospital. The median prehospital serum level of lactate was 2.4 mmol/L (to convert millimoles per liter to milligrams per deciliter, divide by 0.111). Lactate was associated with mortality (odds ratio, 1.23; P < .001), surgery (odds ratio, 1.13; P < .001), and MODS (odds ratio, 1.14; P < .001). When a threshold lactate value of more than 2 mmol/L was added to the predictive model of shock, respiratory distress, or altered sensorium, sensitivity improved from 88% to 97% for death, 64% to 86% for surgery, and 94% to 99% for MODS. The investigators concluded that the prehospital serum level of lactate is a predictor of mortality, surgery, and MODS in trauma patients.

Lactic acidosis is characterized by persistent blood lactate levels (usually >5 mmol/L) in association with metabolic acidosis. Major metabolic dysregulation, tissue hypoperfusion (ischemia), the effects of certain drugs or toxins, transient metabolic demands such as those that occur after seizures, congenital abnormalities in carbohydrate metabolism, and respiratory failure are associated with lactic acidosis. A continued debate about the mechanism of lactic acidosis in septic shock suggests that the use of lactate levels as an index of tissue perfusion has its limitations.
addition, HCO$_3^-$ levels, the anion gap, and the strong ion difference were calculated by using the Stewart method along with incorporating the parameters of age; serum levels of lactate, glucose, and sodium; and pH. The mean HCO$_3^-$ level was 22.4 (SD, 7.2) mEq/L and the anion gap was 20.1 (SD, 4.1) mEq/L when the classic Henderson method was used for the calculations. When the Stewart method was used, the mean HCO$_3^-$ level was 22.6 (SD, 7.4) mEq/L, and the anion gap was 19.9 (SD, 4.5) mEq/L. The values for all parameters used in blood gas analysis differed significantly (P < .001) according to the method used for the calculations. However, a strong correlation was found between the classic method and the Stewart method for calculating the HCO$_3^-$ level and the anion gap. Both methods could be used accurately in acid-base disorders. The Stewart method may be a more effective method for evaluating complex metabolic acidosis.

**Tissue Oxygen Monitoring Technology**

This technology is based on near-infrared spectroscopy (NIRS), an optical method of illuminating chemical compounds that absorb, reflect, and scatter light directed at the muscle tissue bed. Oxyhemoglobin, deoxyhemoglobin, and total hemoglobin concentrations in the muscle tissue bed are measured.

The near-infrared spectrum includes wavelengths of 700 to 1000 nm, and wave lengths of approximately 650 to 900 nm are used in the clinical application of NIRS technology. Visible light can penetrate tissue no deeper than 1 cm, whereas NIRS can penetrate up to 8 cm. The noninvasive sensor is placed on the thenar eminence (Figure 1). Wavelength analysis of 3 forms of hemoglobin (oxyhemoglobin, deoxyhemoglobin, and total hemoglobin) are the basis of the skeletal muscle tissue oxygen saturation of hemoglobin (StO$_2$). The sensor illuminates and light scatters into the tissue of the thenar eminence (Figure 2). The light is absorbed by hemoglobin in
accordance with the oxidation state of hemoglobin. Highly oxygenated hemoglobin absorbs less light than does deoxygenated hemoglobin. The sensor then measures the amount of light returning to the spectrometer, which is calculated as Sto2. Unlike pulse oximetry, NIRS and Sto2 can be used to measure perfusion in a pulseless or hypothermic patient. Thus, NIRS is widely used in trauma and emergency departments and for patients in whom therapeutic hypothermia is being implemented.

Detecting subtle changes in perfusion, prompt investigation of the cause, and subsequent treatment may lead to less risk for MODS. The value of Sto2 for prediction of mortality in high-risk patients with trauma to the torso who have hemorrhagic shock was studied at 7 level I trauma centers. The sample population consisted of 383 patients who arrived within 6 hours of injury, had evidence of shock (systolic blood pressure <90 mm Hg or base deficit >6 mEq/L), and had broken bones or trauma to the torso. MODS developed in 50 of the 383 patients. All clinicians were blinded to the data obtained upon arrival and at 24 hours, along with information on other predictors of hypoperfusion. For discrimination of MODS patients, minimum Sto2 was similar to maximum base deficit. Sensitivity for both measures (Sto2 cutoff, 75%; base deficit cutoff, 6 mEq/L) was 78%. Specificity was 34% to 39%. The positive predictive value was 18% to 20%, and the negative predictive value was 88% to 91%. In the first hour, the sensitivity of minimum Sto2 (cutoff, 75%) was significantly greater ($P = .02$) than that of maximum base deficit (cutoff, 6 mEq/L) and significantly greater ($P = .05$) than that of minimum systolic blood pressure (cutoff, 90 mm Hg) for predicting death. The negative predictive value of minimum Sto2 was also significantly greater than that of maximum base deficit ($P = .01$) and minimum systolic blood pressure ($P = .04$). For prediction of mortality, minimum Sto2 had a greater specificity than did minimum systolic blood pressure ($P = .03$). The results indicated that NIRS-derived Sto2 could be used to detect patients within the first hour of arrival to the emergency department who would later experience MODS or die. Of note, Sto2 had the same discriminatory power as base deficit and systolic blood pressure for prediction of both MODS and mortality.

Another study of the correlation between initial Sto2 and the development of MODS was done in an urban, academic, level I trauma center/emergency department and met standardized criteria for activation of a trauma team. Measurement of Sto2 began immediately on arrival and was continued up to 24 hours for patients admitted to the trauma intensive care unit. The results of other resuscitation laboratory measurements and clinical evaluation tools were collected. Descriptive statistical analyses were performed; numeric means, multivariate regression, and rank sum comparison were used. The clinicians had no knowledge of the Sto2 values. A total of 78 patients were enrolled in the study during a 14-month period. The mean injury severity score was 18.5 (SD, 12.9), and 76.9% of the patients had experienced blunt trauma. MODS developed within the first 24 hours in 26 of the 78 patients (33%), who had a mean initial Sto2 of 53.3% (SD, 10.3). Among the non-MODS patients, the mean Sto2 was 61.1% (SD, 10.0%). The difference between the 2 groups was significant ($P = .002$). The mean injury severity score of 29.9 (SD, 11.5) in MODS patients was significantly higher ($P < .001$) than the score of 12.1 (SD, 9.1) in non-MODS patients. The shock index also differed significantly ($P = .001$) between the 2 groups: 0.92 (SD, 0.28) for MODS patients and 0.73 (SD, 0.19) for non-MODS patients. Serum levels of lactate did not differ significantly between the 2 groups. In this study, Sto2 values during initial trauma resuscitation correlated with the later development of MODS.

During active hemorrhage, the Sto2 value should decrease (predictive of imminent hemorrhagic shock). Although external bleeding is easily recognized, internal bleeding is not as obvious. Upon arrival at a trauma center, an initial Sto2 value (measured concurrently with or before vital signs) less than 65% is predictive of hemorrhagic shock, indicating a requirement for early administration of blood. Sto2 can be used as an early predictor of hemorrhage and may be superior to clinical assessments of blood pressure, heart rate, arterial oxygen saturation, urine output, and hemoglobin concentration that may not appear until class III/IV shock has occurred. NIRS technology also has been used by the armed forces during conflicts to detect early states of hypovolemic shock.

Macdonald and Brown used the MeSH terms near-infrared spectroscopy, sepsis, shock, tissue oxygen saturation, and microcirculation to search for relevant
litterature on NIRS. They reviewed 286 articles (of which 38 were considered relevant), including 6 observational clinical studies. The published information was examined for use of NIRS at the thenar eminence of the hand. Three studies indicated that StO$_2$ was significantly lower in patients with severe sepsis (76%) than in healthy control patients (87%) and that the recovery slope for StO$_2$ was significantly lower after vascular occlusion in sepsis patients. In 6 studies, the mean baseline StO$_2$ reading was lower among patients with sepsis than among controls and lower in patients with septic shock. All 6 studies were observational and subject to reporting bias.

Neto et al. did a systematic review and meta-analysis of publications between 1966 and 2013 on the static and dynamic variables derived by using NIRS in patients with sepsis. The association between StO$_2$, reperfusion slope, occlusion slope, maximum StO$_2$ value minus basal StO$_2$ value, and prognosis in patients with severe sepsis or septic shock was evaluated. The search yielded 20 articles with 962 participants: 717 with severe sepsis or septic shock and 245 healthy control patients. Patients with sepsis had significantly lower values than did healthy controls for the following variables: mean StO$_2$, 78.27% (SD, 4.91%) vs 82.02% (SD, 3.57%), $P = .01$; reperfusion slope, 2.75% (SD, 0.63%) vs 5.19% (SD, 2.86%) per second, $P = .003$; and maximum StO$_2$ value minus basal StO$_2$ value, 7.86% (SD, 0.11%) vs 12.53% (SD, 2.65%), $P = .01$.

In early goal-directed therapy, a component of the Surviving Sepsis Campaign, ScvO$_2$ monitoring is an important step. ScvO$_2$ monitoring is invasive and expensive. A study of 13 patients was performed to determine if a correlation exists between ScvO$_2$ and StO$_2$ as indicated by the Pearson correlation coefficient. A high correlation was detected: $r = 0.91$, StO$_2$ = 1.00, and ScvO$_2$ = 2.85. Bland-Altman analysis was also performed: 100% of monitored points fell within the range of the mean plus or minus 1.96 SD. The standard deviation of the difference between the StO$_2$ and the ScvO$_2$ was 7.2% to 13.2%. The results indicate that StO$_2$ can be used for directing clinical treatment, although further research is needed to improve precision.

Mozina and Podbregar studied use of NIRS for evaluating skeletal muscle StO$_2$ in patients with left-sided heart failure (cardiogenic shock) with or without severe sepsis, and then compared StO$_2$ with SvO$_2$. Patients with left-sided heart failure ($n = 24$) had lower StO$_2$ than did healthy volunteers: means, 58% (SD, 13%) and 84% (SD, 4%), respectively; $P < .001$. Correlation was good between StO$_2$ and SvO$_2$ and between SvO$_2$ and the plasma level of lactate. StO$_2$ and SvO$_2$ tracked well together over time, although StO$_2$ led to an overestimate of SvO$_2$, with a bias of -2.3% and a precision of 4.6%. Overestimation may be due to use of NIRS, which does not discriminate between compartments and provides a global assessment of oxygenation in all vascular compartments (arterial, venous, and capillary). The results confirmed that skeletal muscle StO$_2$ values in patients with cardiogenic shock could be used for fast noninvasive estimation of SvO$_2$ and that the trend in the values of StO$_2$ can be substituted for the trend of values of SvO$_2$.

Data suggest that in patients in the early phase of septic shock, low StO$_2$ is predictive of low SvO$_2$ and higher mortality rates. However, in patients with severe sepsis and severe heart failure, the discrepancy between ScvO$_2$ and SvO$_2$ presents a clinically important difference between both variables; mean ScvO$_2$-SvO$_2$ was 72% (SD, 8), and the ScvO$_2$-SvO$_2$ difference was 9.4% (SD, 7.5). The skeletal muscle StO$_2$ deoxygenation rate is inversely proportional to the difference between ScvO$_2$ and SvO$_2$. Dobutamine did not influence this relationship. If ScvO$_2$ is used as a treatment goal, NIRS may be a useful noninvasive diagnostic method to detect patients who have normal ScvO$_2$ but also have a potentially abnormal low StO$_2$. Research on muscle physiology showed the capability of noninvasive hybrid NIRS–diffuse correlation spectroscopy (DCS) for continuous monitoring of hemodynamic and metabolic parameters, including absolute blood flow, blood oxygenation, and VO$_2$ rate in local muscle groups during exercise. Healthy volunteers (n = 9) performed a handgrip exercise to increase blood flow and VO$_2$ in forearm flexor muscles while a hybrid optical probe on the skin was used to directly monitor concentrations of oxyhemoglobin, deoxyhemoglobin, and total hemoglobin; StO$_2$; relative blood flow, and relative VO$_2$ rate. The signals for relative blood flow and relative VO$_2$ rate were calibrated by using absolute baseline blood flow and VO$_2$ obtained through venous and arterial occlusions. Skeletal muscle function depends...
on oxidative metabolism and blood-flow response. Mean blood flow before exercise was 1.76 mL/100 mL per minute (SE, 0.42). The mean plateau value toward the end of exercise was 8.93 mL/100 mL per minute (SE, 2.81). Mean V̇O₂ before exercise was 0.11 (SE, 0.03) mL O₂/100 g per minute and the mean plateau value toward the end of exercise was 0.57 (SE, 0.13) mL O₂/100 g per minute. These results were consistent with previous findings in a systematic review of the literature. Because it can be used to measure blood flow, blood oxygenation, and V̇O₂, the hybrid NIRS-DCS technology is relevant to the study of muscle physiology and pathology.  

**Relevance of StO₂ to Care Providers**

Clinical manifestations of inadequate ḊO₂ might include tachycardia, tachypnea, prolonged capillary refill time, an increase in temperature, reduced level of consciousness, decreased urine output, diaphoresis, or cool extremities. Familiarity with the application of StO₂ monitoring could lead to early detection of patients at risk for shock. StO₂ correlated well with changes in ḊO₂, base deficit, and lactate levels in early clinical testing during active shock resuscitation. StO₂ monitoring can also provide earlier detection of poor or altered perfusion in elderly patients. Traditionally, the elderly show nonspecific signs and symptoms. A correct diagnosis could be confounded with multiple comorbid conditions in elderly patients. Clinical settings need technology that is fast, noninvasive, and portable and provides results in real time. The NIRS-DCS device is reusable and relatively inexpensive, characteristics that make the technology accessible and affordable.

In the intensive care unit, results of StO₂ monitoring have been encouraging. For example, during weaning from mechanical ventilation, StO₂ monitoring could be used during a 30-minute spontaneous breathing trial to discriminate between patients in whom weaning would be successful and those in whom weaning would be unsuccessful. Thus, StO₂ can be used as a monitoring system for detecting limited cardiovascular reserve.

A multicenter prospective cohort study was designed to identify patients at risk for MODS after trauma. Trauma patients with MODS require more resource-intensive hospitalization than do trauma patients who do not experience MODS. In intensive care, the leading causes of death are multiorgan failure, cardiovascular failure, and sepsis. Multiorgan failure has a mortality rate of 11% to 18%. The mean intensive care unit length of stay was 23 days for MODS patients and 7 days for non-MODS patients. The median hospital charges in 2007 were $312,104 for a MODS patient and $148,384 for a non-MODS patient. Early detection of shock, prompt investigation of the cause of shock, and treatment of hypoperfusion of organs facilitated by technology could have a major impact on cost-effective patient care, could improve patient outcomes, and ultimately could save lives.

Limitations and benefits of the NIRS-DCS technology should be compared with those of alternative measurement techniques (see Table). A definite advantage of the NIRS-DCS method is that it is completely noninvasive and is easier to use and more comfortable for patients than are other methods of measuring. Use of NIRS-DCS is inexpensive compared with the cost of other techniques such as positron emission tomography, perfusion magnetic resonance imaging, and ³¹P-magnetic resonance spectroscopy. One concern about NIRS technology is the influence of the thickness of adipose tissue, which can sometimes distort hemodynamic measurements. Gurley et al found that up to 3 mm of adipose tissue affected the sensitivity of the NIRS-DCS signal by less than 10%. In addition, no significant correlation was found between the thickness of adipose tissue and V̇O₂ or blood flow. A reasonable assumption is that hemodynamic measurements were not adversely influenced by skin thickness.

**Conclusion**

Currently, no gold standard exists for diagnosis of shock. NIRS is a noninvasive technology that has shown potential in detecting patients who are at risk for organ dysfunction and that could be used to guide resuscitation. Future research efforts, to include randomized control trials, are required to address the usefulness of StO₂ in outcome prediction and as a target end point for initial resuscitation. The StO₂ values could be increasingly more sensitive and specific if coupled with other “traditional” measures of organ perfusion, such as blood lactate levels, in a controlled environment such as an emergency department, a trauma department, or an intensive care unit. On the battlefield or before emergency medical transport to a
treatment facility, StO₂ has proven to be portable, noninvasive, comfortable for the patient, and easy to use. StO₂ monitoring is becoming a valuable addition to the prognostic care of trauma patients and may be the catalysis for a shift in the management of trauma patients. CCN

Financial Disclosures
None reported.

References

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Table  Benefits and limitations of measurements used to indicate or detect shock

<table>
<thead>
<tr>
<th>Feature</th>
<th>Lactate</th>
<th>SVO₂</th>
<th>SCVO₂</th>
<th>StO₂</th>
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<tbody>
<tr>
<td>Direct measurement of the adequacy of oxygen available to the tissue?</td>
<td>No</td>
<td>No</td>
<td>No</td>
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<tr>
<td>Continuous measurement?</td>
<td>No</td>
<td>Yes</td>
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<td>Noninvasive?</td>
<td>No</td>
<td>No</td>
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<td>Predictor of bad outcome for patient?</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Limitations?</td>
<td>Hyperlactatemia may persist for hours after anaerobic metabolism is resolved¹⁰</td>
<td>A flow-weighted mean of venous saturations from perfused vascular beds²</td>
<td>Central venous pressure monitoring cannot be used to measure cardiac output and does not provide reliable information on the status of pulmonary circulation in the presence of left ventricular dysfunction²⁹</td>
<td>Adipose tissue aposits the sensitivity of signal by &lt;10%²²</td>
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</table>
| Abbreviations: SCVO₂, central venous oxygen saturation; SVO₂, mixed venous oxygen saturation; StO₂, skeletal muscle oxygen saturation.

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