

# The Hemodynamic and Physiological Relevance of Continuous Central Venous Oxygenation Monitoring: Moving Beyond Sepsis

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## OVERVIEW

SvO<sub>2</sub>/ScvO<sub>2</sub> (mixed or central venous oxygen saturation) is an important yet frequently misunderstood hemodynamic parameter. Specifically, its use in patients other than those in cardiac surgery and with sepsis has not been widely understood as a useful tool in decision-making with critically ill patients. This paper covers tissue oxygenation and the role of continuous central venous oxygenation monitoring in the assessment of both potential oxygen delivery and consumption in the typical critical care patient.

## MEASURING POTENTIAL OXYGEN DELIVERY

The normal oxygenation cycle begins with deoxygenated blood entering the right side of the heart. As hemoglobin molecules pass out of the right side of the heart and through the lungs/alveoli, four molecules of oxygen become attached to each individual hemoglobin molecule. This oxygenated blood then passes through the left side of the heart and out to the tissue. Upon reaching the capillary beds, an average patient extracts one molecule of oxygen from each hemoglobin molecule, leaving the red blood cells 75% saturated with oxygen as they return back to the right side of the heart.<sup>1</sup>

The amount of oxygen saturation in the arterial system as it passes into the capillary bed is referred to as potential oxygen delivery (DO<sub>2</sub>), as extraction has not yet occurred. In the early days of measuring DO<sub>2</sub>, blood had to be sampled directly from an artery and the amount of oxygen/saturation was measured through instrumentation outside the body.<sup>2</sup> Continuous pulse oximetry, or SpO<sub>2</sub>, was widely used in the operating room beginning in the 1980s.

As the technology was simplified, its use in critical care areas emerged in the early to mid-1990s. Because of the cost of this technology, most hospitals only acquired a handful of these monitors in their ICUs and emergency departments. Medical staff would use the same monitor and go bed to bed at specified times, obtaining a reading of each patient's arterial oxygen saturation.

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This snapshot in time was quickly recognized to be unacceptable because a patient's physiology is constantly changing, sometimes rapidly. Continuous SpO<sub>2</sub> monitoring was adopted as a standard in all ICU patients in the mid to late 1990s as clinicians learned that one value obtained at regular intervals was not sufficient to treat serious and critically ill patients.<sup>3</sup>

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## INCREASING POTENTIAL OXYGEN DELIVERY: FOUR METHODS

Measuring  $DO_2$  is dependent upon hemoglobin/hematocrit, cardiac output, and saturation of the individual hemoglobin molecules. Increasing potential oxygen delivery can be achieved in four ways:

1. The first, and simplest, form is to increase inspired oxygen or  $FiO_2$ . Assuming that a disease process is not inhibiting the transfer of oxygen in the alveolar beds, there should be a rise in partial pressure of oxygen and subsequent saturation of hemoglobin molecules.<sup>1,2,4,5</sup>
2. Mechanical ventilation can be used for patients who require assistance in either the movement of oxygen into the lungs or who require more pressure for oxygen to transfer across the alveolar membranes. BiPAP, endotracheal intubation, and advance ventilator modes such as Airway Pressure Release Ventilation (APRV) and oscillation increase the movement of  $O_2$ , including controlling pressure both during inspiration and at end expiration. Using mechanical ventilation, Positive End Expiratory Pressure (PEEP) can be increased, stretching alveoli to maximize their surface area and resulting in increased oxygen transference into the blood.<sup>2,5</sup>
3. Though recent research has shown that blood transfusions may increase mortality in many patient populations, increasing hemoglobin levels through transfusion of packed red blood cells may result in extra oxygen-carrying capacity. In simple terms, if you increase the amount of hemoglobin molecules crossing the capillary beds, there is more potential for oxygen to be extracted because more oxygen molecules are present. This treatment should be reserved for disease processes shown to require transfusion rather than being based on lab values alone, as blood is a liquid transplant and has many adverse effects.<sup>2,6</sup>
4. The final way to increase potential oxygen delivery is to deliver more blood volume directly to the tissues. Increasing cardiac output (CO) does this by maximizing the amount of blood exiting the left side of the heart that is directly pumped to the capillary beds. CO is composed of stroke volume (the amount of blood pumped from the left ventricle during each cardiac cycle) times heart rate:  $CO = SV \times HR$ .

Many methods exist to increase CO depending upon the patient's hemodynamic status, overall condition, and disease process. The appropriate method/intervention should be chosen based on current physiological needs.<sup>5,7</sup>

The intermittent measurement of a patient's central venous oxygen saturation does not give a clinician enough information to make good decisions because the saturation levels may vary from minute to minute.<sup>3-5,7</sup>

## MEASURING VENOUS OXYGENATION

As blood returns to the right side of the heart, venous oxygenation can be measured through saturation of the hemoglobin molecules. This was first done, just as with arterial oxygenation, by obtaining a blood sample from the central circulation and measuring the oxygen level, the partial pressure, and/or hemoglobin saturation.

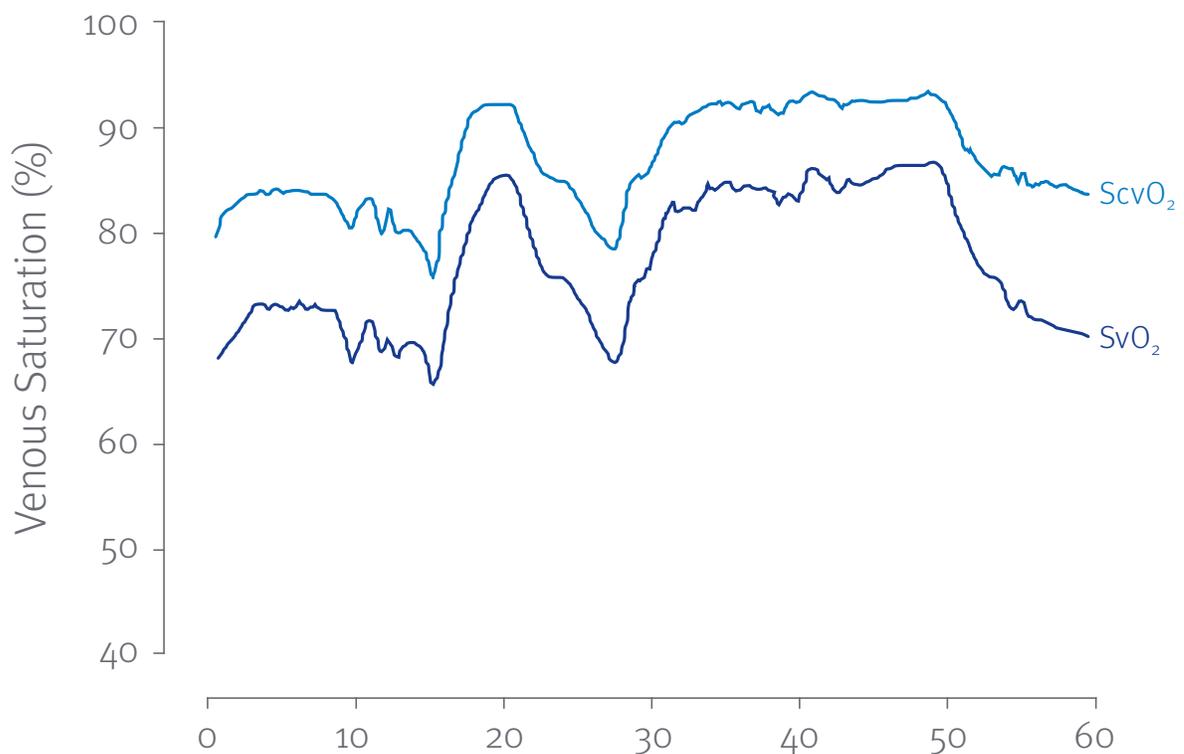
Many clinicians still use this outdated practice of measuring central venous oxygen saturation, giving them only a snapshot in time. As we learned with arterial saturation ( $SpO_2$ ), this method of monitoring does not give a clinician enough information to make good decisions because a patient's condition may vary from minute to minute.<sup>3-5,7</sup>

Continuous SvO<sub>2</sub>/ScvO<sub>2</sub> is measured through technology similar to SpO<sub>2</sub>. Various wavelengths of light are emitted from the tip of a catheter that is located in the pulmonary artery using a PA catheter (SvO<sub>2</sub>), or the superior vena cava using a central line (ScvO<sub>2</sub>). As these wavelengths of light are reflected back to the receiver on the tip of the catheter, venous oxygen saturation is measured and displayed continuously in real time.

Because the normal oxygen cycle delivers one molecule of oxygen in the capillary bed, the average healthy person should have a venous oxygenation saturation of around 70 to 80%. ScvO<sub>2</sub> measures oxygen saturation returning from the upper body.<sup>1,3,7</sup> The value runs approximately 5 to 10% higher than SvO<sub>2</sub>. SvO<sub>2</sub> measures saturation of blood returning from both the lower and upper body mixed with coronary return.

Continuous ScvO<sub>2</sub> monitoring can be easily achieved through the use of the same triple-lumen central venous catheter currently accepted as the standard of care in critically ill patients.<sup>8</sup>

Research has proven that these values correlate exactly, thus allowing ScvO<sub>2</sub> to be utilized in a less invasive manner as a replacement, or surrogate, for the more invasive SvO<sub>2</sub>. Most critically ill patients require the use of a central venous catheter. Continuous ScvO<sub>2</sub> monitoring can be easily achieved through the use of the same invasive practice (triple-lumen central venous catheter) currently accepted as the standard of care in critically ill patients.<sup>8</sup> This means that no other devices need to be placed.<sup>4,9</sup> The following graphic shows venous saturation by minutes for ScvO<sub>2</sub> and SvO<sub>2</sub>.



**Venous Saturation** Although ScvO<sub>2</sub> values run approximately 5 to 10% higher than SvO<sub>2</sub>, research has proven that these values correlate exactly, thus allowing ScvO<sub>2</sub> to be utilized in a less invasive manner as a replacement, or surrogate, for the more invasive SvO<sub>2</sub>.

## COMPARING FIBER OPTIC TECHNOLOGIES FOR GATHERING ScvO<sub>2</sub> AND SvO<sub>2</sub> DATA

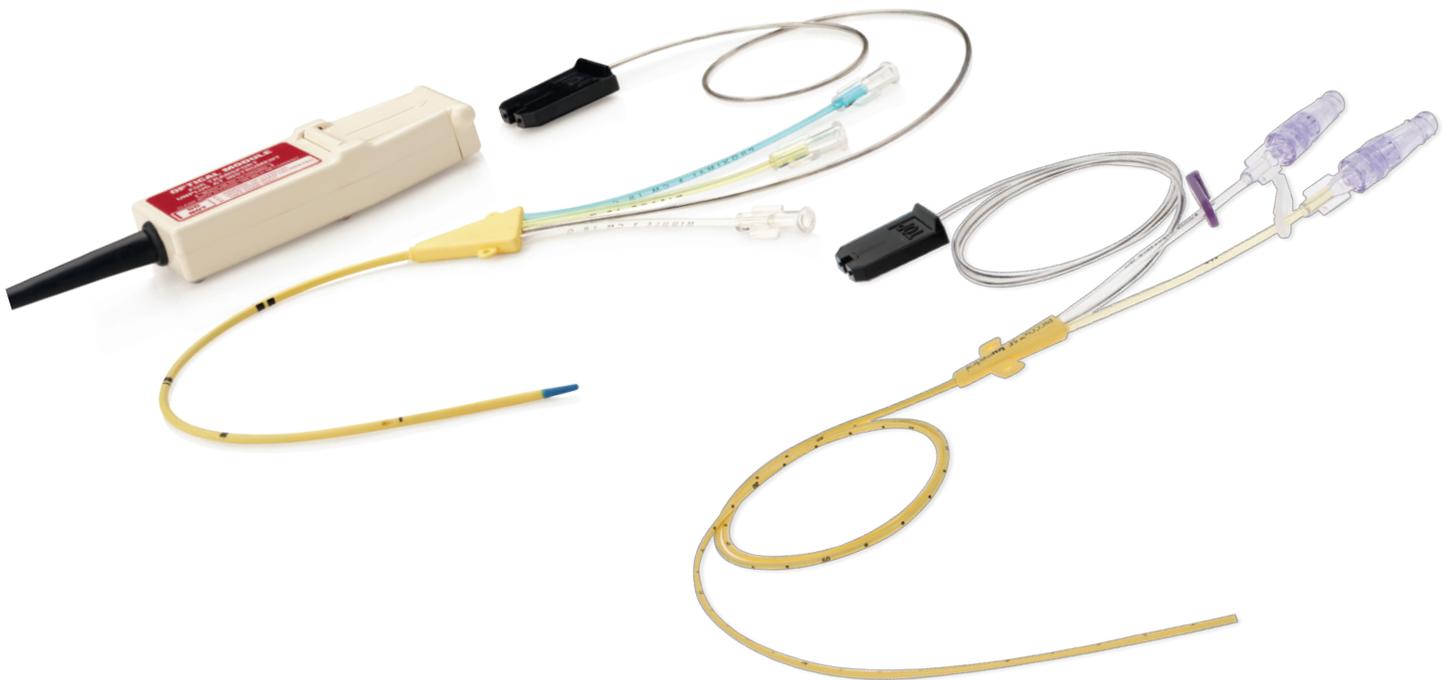
Several fiber optic technologies are available in the medical industry to gather SvO<sub>2</sub>/ScvO<sub>2</sub> data. The first uses only two wavelengths of light, resulting in limited capability because it requires the hemoglobin level to be constant.

This forces the user to calibrate the catheter at least daily, including entering frequent hemoglobin values. The other limiting factor with this method is that the value measured can be inaccurate if there is a small change in hemoglobin, which is frequent in critical care patients.<sup>10-12</sup>

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It is available in a standard triple-lumen central venous catheter or in a new configuration utilizing the same three-wavelength fiber optic bundle delivered through a peripherally inserted cardiac catheter (known as a PICC line). This less invasive option may allow for more widespread adoption of ScvO<sub>2</sub> monitoring outside the OR and ICU.



ICU Medical's TriOx™ central venous oximetry catheters, available in CVC and PICC form, are currently the only technologies providing three-wavelength oximetry for greater accuracy in assessing real-time tissue oxygenation status.

## MAKING DECISIONS IN TREATMENT AND PLAN OF CARE

Continuous measurement of both ScvO<sub>2</sub> and SpO<sub>2</sub> allows us to calculate oxygen consumption by subtracting the two values. In a normal patient, there is 25% extraction of oxygen based on the normal values of each of these monitoring techniques. Through interpretation of these values, ScvO<sub>2</sub> can indicate a change in either oxygen delivery or consumption. As stated earlier, oxygen delivery is composed of CO, hemoglobin, and SpO<sub>2</sub>.

Changes in oxygen consumption can be from metabolic rate, oxygen extraction rate, or many other physiologic processes that will be discussed shortly.<sup>1-5, 7-9</sup> As with all hemodynamic monitoring, a value must be interpreted through analyzing all the parameters together, including the patient's condition, to make much earlier and better decisions in treatment and plan of care.

When oxygen demand increases in a healthy person, the body attempts to maximize delivery by increasing cardiac output and respiratory rate. ScvO<sub>2</sub> values should remain in the normal range as the person compensates for this increased demand. If the body is unable to compensate because of disease processes or other physiologic problems, tissues extract more than one oxygen molecule, resulting in lower venous oxygenation saturation as evidenced by a decrease in ScvO<sub>2</sub>.<sup>2, 4, 7, 13</sup>

Clinicians must keep in mind that patients compensate differently. A person who is athletic and exercises regularly will have a more increased stroke volume over heart rate. A sedentary individual will increase his or her heart rate, as he or she is unable to increase stroke volume because of lack of cardiac muscle and training. Interpreting compensation must take into account the individual patient and other hemodynamic values.<sup>2, 4, 7, 13</sup>

The first sign that a patient is beginning to decompensate will be a decrease in ScvO<sub>2</sub>, prior to other hemodynamic or lab values changing. This early detection makes continuous ScvO<sub>2</sub> an invaluable tool in the monitoring and treatment of the critically ill patient. ScvO<sub>2</sub> monitoring is simply about meeting the oxygen demand by determining the cause of changes in either delivery or extraction.<sup>4, 7, 13</sup> Causes of decreased ScvO<sub>2</sub> are listed in the following table:

Causes of Decreased ScvO <sub>2</sub>	Examples
Increased O <sub>2</sub> consumption	Hyperthermia Shivering Infection Seizures Pain Trauma Head injury
Decreased CO/CI	Negative inotropic agents Decreased preload/increased afterload Hypovolemia Sepsis MODS
Decreased O <sub>2</sub> supply	Respiratory failure Ventilatory changes Suctioning Airway obstruction Increased WOB Decreased alveolar/arterial oxygenation
Nursing Interventions	Position changes CPT EKG Visitors Bathing (25%)

## SUMMARY

Treating low ScvO<sub>2</sub> is done by first determining the cause. The clinician must analyze the patient's overall condition, ScvO<sub>2</sub>, and other hemodynamic values before determining the next therapeutic intervention. After examining the patient's data and referring to the above table, one of the four causes should be apparent. The exact cause is then determined and a treatment course set and implemented.

The four basic treatment regimens include the following:

1. Decreasing metabolic demand (need for extraction)
2. Increasing oxygen delivery/saturation of hemoglobin
3. Increasing circulating hemoglobin
4. Increasing cardiac output

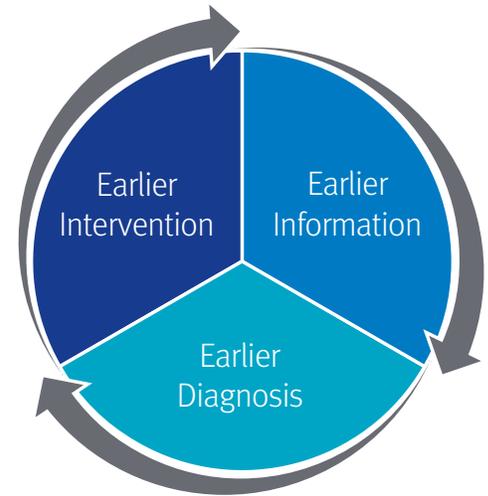
Note that cardiac output and blood pressure are not synonymous. Utilizing ScvO<sub>2</sub> may show that a lower CO or MAP is acceptable to meet the current metabolic demands of the patient, resulting in lower use of pressors, oxygen/mechanical ventilation, blood products, and/or inotropes. ScvO<sub>2</sub> can also be used for real-time weaning of pressors, inotropes, and ventilator support.<sup>1, 2, 4, 5, 7, 9, 13</sup>

The following table evidences increased ScvO<sub>2</sub>:

Causes of Increased SvO <sub>2</sub>	Examples
Decrease in O <sub>2</sub> Demand	Hypothermia NMB Anesthesia Relaxation/sedation Sepsis Pressure support ventilation A-V shunting
Increased CO/CI	Positive inotropic agents Afterload reduction Fluid administration IABP
Increased O <sub>2</sub> Supply	Increased FiO <sub>2</sub> Increased SaO <sub>2</sub> Increased PaO <sub>2</sub>

## RESPONDING TO TREATMENT

Increased ScvO<sub>2</sub> may indicate either a positive response to current treatments or that oxygenation extraction has decreased because of blockages such as micro-clotting in sepsis or AV shunting. Increased ScvO<sub>2</sub> may be used (after cause is determined) to alter treatment such as weaning the ventilator or decreasing inotropes and pressors, remembering



**Continuous ScvO<sub>2</sub>** Use continuous ScvO<sub>2</sub> to obtain information about the patient earlier than other parameters, so earlier diagnosis may lead to earlier intervention.

that typical hemodynamic goals such as a MAP greater than 60, CI  $\geq$  2, HGB  $\geq$  7, and PaO<sub>2</sub>  $\geq$  60 may not be required in a particular patient because of his or her decreased metabolic demand.<sup>1, 2, 4, 5, 7, 9, 13</sup>

Intervention should be done when ScvO<sub>2</sub> moves out of the normal 70 to 80% range or when there is a change of plus or minus 10% for more than 5 to 10 minutes. The trend of ScvO<sub>2</sub> should be taken into account, especially with patients that present with atypical hemodynamic values.

The following table represents standard accepted values of ScvO<sub>2</sub>. Please note that the ideal time to intervene is when the patient's ScvO<sub>2</sub> is in the 50 to 70% range. At this point, the patient is in a compensatory mode with little variation in other parameters being monitored. As ScvO<sub>2</sub> drops below 50%, other hemodynamic monitoring values will begin to be apparent. Unfortunately, damage is already being done to the tissues once you see a change in other monitoring values.<sup>2, 4, 5, 7, 9, 13, 14</sup>

Continuous ScvO<sub>2</sub> should be considered in all critically ill patients, especially those who require use of a central venous or peripherally inserted cardiac catheter.

SvO <sub>2</sub> /ScvO <sub>2</sub>	Oxygenation Status
70-80%	Normal extraction DO <sub>2</sub> $\geq$ VO <sub>2</sub>
50-70%	Increasing VO <sub>2</sub> or decreasing DO <sub>2</sub> Compensatory O <sub>2</sub> extraction
30-50%	Exhausting of extraction Beginning of lactic acidosis
25-30%	Severe lactic acidosis
<25%	Cellular death

Patients who may benefit from continuous ScvO<sub>2</sub> monitoring may include myocardial infarction (risk for drop in cardiac output), sepsis (as evidenced by the surviving sepsis campaign), trauma/GI bleed or other bleeding diathesis (which can cause abrupt changes in hemoglobin but is not recognized for hours until another H&H is drawn), shock of any kind, or basically any critical care patient with potential for acute changes in hemoglobin, oxygen consumption/demand, or cardiac output.

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