The continuous monitoring of mixed venous oxygen hemoglobin saturation ($S\text{vO}_2$) by fiberoptic oximetry is useful in the measurement of hemodynamic stability and tissue oxygenation. This technology and the information it provides can be valuable in the high-risk patient requiring anesthesia. The system consists of a fiberoptic pulmonary artery catheter and various components including a main computer/processor. It measures continuous $S\text{vO}_2$ and many hemodynamic parameters such as oxygen delivery and oxygen consumption.

A change in $S\text{vO}_2$ will alert the clinician to a fluctuation in the balance of oxygen delivery and metabolic demand. Factors that affect this physiologic balance include tissue oxygen consumption, arterial oxygen hemoglobin saturation, blood hemoglobin concentration, and cardiac output. By assessing each factor systematically, the clinician is better able to diagnose physiologic changes during anesthesia. Because $S\text{vO}_2$ is sensitive to physiologic conditions, including those affected by anesthesia and surgery, changes in this parameter can detect hemodynamic changes or instability before conventional methods of monitoring.

The use of advanced monitoring devices to aid in decisions, problem solving, and crisis intervention is commonplace in anesthesia practice. However, available methods usually detect changes after deterioration in clinical status. The introduction of the fiberoptic catheter to measure $S\text{vO}_2$ has permitted a sensitive in vivo assessment of cardiopulmonary function. It enables continuous monitoring of oxygen transport and tissue oxygen consumption. If the balance is disrupted, $S\text{vO}_2$ will promptly change. Therefore, this device is very useful in the high-risk surgical patient for perioperative monitoring and assessment. If the patient requires postanesthesia intensive care, $S\text{vO}_2$ measurement will continue to guide therapy.

**Definition and measurement of $S\text{vO}_2$**

$S\text{vO}_2$ represents the percent of hemoglobin which is saturated with oxygen in mixed venous blood. It is termed mixed because it represents the blood in the pulmonary artery after the blood has returned from the superior vena cava, inferior vena cava, coronary sinus, and thebesian veins. This mixture of blood represents an average of saturated and unsaturated blood returning from the body tissues. It therefore reflects oxygen uptake and hemodynamic status with respect to the balance of oxygen transport and tissue demand.

The principal device in use today to measure $S\text{vO}_2$ is the Opticath® fiberoptic pulmonary artery (PA) catheter and Oximetrix® 3 System. The Opticath® is a five-lumen, 7.5 French, 110 cm flow-directed thermodilution catheter that measures pulmonary artery pressure, pulmonary capillary wedge pressure, and central venous pressure. The basic components of the Oximetrix® 3 System con-
sist of the optical module and SO2/CO computer. The optical module contains a triple diode light source and photodetector cell. The SO2/CO computer provides calculation of cardiac output as measured by thermodilution, drug calculations and alarms for SvO2 high/low limit and light signal intensity. The SvO2 and trend graph are continuously displayed on the monitor screen. Calibration is performed preinsertion or in vivo. SO2/CO computer calculation of hemodynamic parameters, such as oxygen delivery and oxygen consumption, provides information on the status of oxygen transport.

The PA catheter contains two fiberoptic transmission fibers. One optical fiber continuously illuminates the blood flow past the distal end of the catheter. A red and infrared light source is supplied by three light-emitting diodes, each giving off a specific wavelength of light. Use of three wavelengths reduces error by compensating for changes in hematocrit, pulsatile flow and scattered light from blood vessel walls. The second optical fiber detects the light as it reflects off the red blood cells and transmits it to the photodetector. The light is then converted to electrical signals in the optical module and sent to the SO2/CO computer. (See Figure 1.)

The relative quantity of oxyhemoglobin and deoxyhemoglobin is determined using reflective spectrophotometric analysis, which interprets the change in absorption characteristics of red blood cells as light is refracted or reflected off their surfaces. The absorption characteristic of red blood cells can be defined simply as the progressive change in color from purple to scarlet as the ratio of oxyhemoglobin to deoxyhemoglobin increases. Therefore, the percentage of oxyhemoglobin and deoxyhemoglobin can be measured and SvO2 calculated by the computer. To consistently provide an accurate SvO2, the computer is designed to compensate for variables such as blood pH, temperature, hematocrit, carboxyhemoglobin and methemoglobin.

The physiology of SvO2

The primary function of the cardiopulmonary system is to deliver adequate oxygen to meet the metabolic demands of the cells. Oxygen delivery (DO2) is defined as the amount of oxygen provided

Figure 1
Fiberoptic measurement of mixed venous oxygen saturation

The fiberoptic catheter lies in the pulmonary artery. Except for the fiberoptics, it functions in the same manner as standard thermodilution pulmonary artery catheters. The catheter's two optical fibers transmit and receive light processed by the optical module to which it is attached. The optical module is attached to the SO2/CO computer for measurement of SvO2.
to the tissues. Normal oxygen delivery at rest is approximately 1,000 ml/min, which is calculated by multiplying the cardiac output (CO) times the arterial oxygen content (CaO₂) times 10.3,5

\[ CO = \text{heart rate} \times \text{stroke volume} \]

\[ \text{CaO}_2 = (\text{SaO}_2 \times \text{hemoglobin} \times 1.34) + (\text{PaO}_2 \times 0.0031) \]

\[ \text{DO}_2 = \text{CO} \times \text{CaO}_2 \times 10 \]

For example:

5 L/min \times 20 ml/dl \times 10 = 1,000 ml/min

Components that affect oxygen delivery include heart rate, stroke volume, hemoglobin level and arterial oxygen saturation. Partial pressure of oxygen contributes little to the equation, so it can be excluded. These parameters are influenced by changes in physiologic homeostasis such as acid-base imbalance, hemorrhage or hypoxia. For example, hemorrhage will reduce the amount of available hemoglobin, and metabolic acidosis can produce negative inotropic effects. Cardiac output is the principal component and compensatory system in the delivery of oxygen.3,4

Oxygen consumption (VO₂) is defined as the amount of oxygen extracted by the tissues to meet metabolic requirements or demand. Normal VO₂ is approximately 225-275 ml/min. This is calculated by multiplying the cardiac output times the arterial-venous oxygen content difference (C(a–v)O₂) times 10. The C(a–v)O₂ is the difference in oxygen content between the mixed venous (CvO₂) and arterial (CaO₂) samples of blood and represents oxygen utilization in the tissues. A normal consumption of 250 ml/min leaves the system 750 ml/min of venous oxygen reserve returning to the heart.3,5

\[ \text{CvO}_2 = (\text{SaO}_2 \times \text{hemoglobin} \times 1.34) \]

\[ \text{C(a–v)O}_2 = \text{CaO}_2 - \text{CvO}_2 \]

\[ \text{VO}_2 = \text{CO} \times \text{C(a–v)O}_2 \times 10 \]

For example: 5 L/min \times 5 ml/dl \times 10 = 250 ml/min

To figure venous oxygen reserve:

1,000 ml/min \times (\text{DO}_2) - 250 ml/min \times (\text{VO}_2) = 750 ml/min oxygen reserve

To arrive at a mathematical expression explaining SVO₂, the Fick equation that defines cardiac output is commonly used.3,5

\[ \text{CO} = \text{VO}_2 + \text{C(a–v)O}_2 \]

For example: 5,000 ml/min = 250 + (.20 – .15)

By means of rearranging the terms of the equation, the factors representing SVO₂ can be obtained.3,5

\[ \text{SVO}_2 = \text{CO} \times (\text{SaO}_2) - \text{CvO}_2 \times 10 \]

\[ \text{SVO}_2 = \text{CO} \times (\text{CaO}_2 - \text{CvO}_2) \times 10 \]

\[ \text{SVO}_2 = \text{CO} \times 10 \{ (\text{Hb} \times 1.34 \times \text{SaO}_2) - (\text{Hb} \times 1.34 \times \text{SvO}_2) \} \]

\[ \text{SVO}_2 = \text{CO} \times \text{Hb} \times 13.4 \times (\text{SaO}_2 - \text{SvO}_2) \]

\[ \text{SVO}_2 \approx \text{SaO}_2 - (\text{VO}_2 \div (\text{CO} \times \text{Hb} \times 13.4)) \]

\[ \text{SVO}_2 \approx \text{SaO}_2 - (\text{VO}_2 - \text{DO}_2) \]

It is clear that SVO₂ will be influenced by arterial oxygen saturation and the determinants of oxygen consumption and oxygen delivery. If the delivery system fails to meet oxygen demand, the physiologic compensatory response is an increase in cardiac output or an increase in the use of the venous oxygen reserve.3 Healthy individuals can compensate and increase both their cardiac output and their arterial-venous saturation difference threefold.3,5 Thus, a ninefold increase in oxygen consumption by the tissues can be achieved during the demands of stress or illness. However, if the elements of oxygen delivery are unable to compensate, venous oxygen reserve is used and SVO₂ decreases. As available oxygen diminishes to below a capillary PO₂ of 20 mmHg, the result is anaerobic cellular metabolism, decreased energy production and lactic acidosis. This poor clinical state corresponds to an SVO₂ of approximately 30%.3,5

**Interpretation of SVO₂**

From the Fick equation, one can conclude that mixed venous oxygen saturation is affected by four major factors: 3,5,6,7

1. Tissue oxygen consumption.
2. Arterial hemoglobin oxygen saturation.
3. Hemoglobin concentration.
4. Cardiac output.

It is necessary to assess each factor systematically when determining the reason for a change in SVO₂. Because the cardiopulmonary system is dynamic, the influence of compensatory systems and multiple causation must be considered. Nelson showed that SVO₂ correlates highest with the oxygen utilization coefficient, which is defined as the ratio of oxygen consumption to oxygen delivery.4 Therefore, SVO₂ reflects an overall balance in oxygen delivery and oxygen consumption and does not rely heavily on any single determinant.

The normal range for SVO₂ is approximately 60-80%.4,6,7 Generally, if the SVO₂ drops below 60%, or if there is change of 10%, assessment of the patient is indicated.7 An increasing SVO₂ may indicate improved cardiopulmonary status, decreased oxygen demand by the tissues or catheter migration to
Table I
Interpretation of mixed venous oxygen saturation

<table>
<thead>
<tr>
<th>Saturation percent</th>
<th>Physiologic state</th>
<th>Possible cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>High SV0₂ (80-90%)</td>
<td>Enhanced oxygen delivery due to:</td>
<td>Increased FiO₂</td>
</tr>
<tr>
<td></td>
<td>a. Increased arterial oxygen saturation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>b. Increased cardiac output (heart rate and/or stroke volume)</td>
<td>Positive inotropic agents, thyrotoxicosis, vasodilators, intra-aortic balloon pump support, fluid administration</td>
</tr>
<tr>
<td></td>
<td>c. Increased hemoglobin</td>
<td>Administration of blood products</td>
</tr>
<tr>
<td>Catheter not reflecting true SV0₂</td>
<td></td>
<td>Catheter tip wedging or facing vessel wall, intracardiac shunt, severe mitral regurgitation</td>
</tr>
<tr>
<td>Decreased oxygen demands</td>
<td>Anesthesia, hypothermia, increased systemic vascular resistance (vasoconstriction), sepsis, pharmacologic paralysis or muscle relaxation, cyanide toxicity</td>
<td></td>
</tr>
<tr>
<td>Normal SV0₂ (60-80%)</td>
<td>Oxygen delivery and oxygen demands met by system</td>
<td>Adequate tissue perfusion (considered normal in absence of compensatory mechanisms)</td>
</tr>
<tr>
<td>Low SV0₂ (&lt; 60%)</td>
<td>Increased oxygen demands</td>
<td>Hyperthermia, shivering, seizures, pain, exercise, dyspnea, lightening of anesthesia or any muscular action</td>
</tr>
<tr>
<td>Impaired oxygen delivery due to:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. Decreased hemoglobin</td>
<td>Anemia, hemorrhage</td>
<td></td>
</tr>
<tr>
<td>b. Decreased arterial oxygen saturation</td>
<td>Hypoxia, suction, impaired oxygen diffusion, respiratory failure, airway obstruction</td>
<td></td>
</tr>
<tr>
<td>c. Decreased cardiac output (heart rate and/or stroke volume)</td>
<td>Cardiogenic shock, hypotension, dysrhythmias, acidosis, hypoxemia, negative inotropic agents, sepsis, hypovolemia, vasoconstriction</td>
<td></td>
</tr>
</tbody>
</table>

The wedged position. A decreasing SV0₂ may be caused by impaired oxygen delivery or diffusion in the lungs, deficient oxygen transport secondary to decreased cardiac output or hemoglobin, or an increased oxygen demand and utilization by the tissues. An SV0₂ of less than 40% reflects a poor clinical state and is usually associated with hypotension, dysrhythmias and cardiovascular or respiratory arrest. Table I describes a guide to the clinical interpretation of SV0₂.

Clinical use and application in anesthesia

SV0₂ monitors the response of the oxygen transport system and tissue demand to stressful events such as induction, intubation, suctioning or change in position. SV0₂ decreases during stressful interventions but should return to baseline within 5 minutes as physiologic correction occurs. If SV0₂ continues to drop or does not return to baseline, the anesthetist is alerted to deterioration in oxygen transport. SV0₂ can assess beneficial or detrimental physiologic responses to therapy such as modification in anesthetic agent or oxygen delivery, or the administration of crystalloids, blood products or vasoactive drugs.

For example, improvement in the SV0₂ can be observed following the administration of muscle relaxants and positive pressure ventilation for respiratory control. As oxygen consumption decreases in the respiratory muscles as a result of a decrease in the work of breathing, SV0₂ will rise. Conversely, a decrease in SV0₂ may be observed following the reversal of respiratory muscle paralysis secondary to an increase in oxygen consumption from the work of breathing. General anesthesia will also decrease oxygen consumption by reducing metabolic oxygen requirements, resulting in an increase in SV0₂.

Another example of changes in SV0₂ is observed in the management of oxygenation with positive end-expiratory pressure (PEEP). Optimal PEEP therapy can be guided by continuous SV0₂. By monitoring the directional trend of SV0₂ following changes in PEEP or mechanical ventilation, the effectiveness of the change can be evaluated. A rising SV0₂ may indicate improved ventilation/per-
Fusion matching resulting in an increase in oxygen delivery. SvO₂ monitoring will also alert the clinician to the hazards of PEEP therapy. If the PEEP compromises the cardiac output, the SvO₂ may drop, despite an expected improvement.¹¹

Vasoactive drugs are often required in high-risk cardiovascular patients. Perioperative hemodynamic fluctuations are common and require careful management. Inotropic, vasopressor or vasodilator drugs may be necessary intraoperatively to maintain adequate tissue perfusion. A decrease in SvO₂ is often the first indication for this kind of therapy.² For example, if cardiac output is impaired from a high afterload or elevated systemic vascular resistance, the SvO₂ can decrease secondary to the restriction of oxygen delivery. By administration of a vasodilator such as sodium nitroprusside, afterload can be reduced and cardiac output improved, leading to a rise in SvO₂. A second example of reduced cardiac output can occur from reduced preload or inadequate cardiac contractility. With administration of fluids or a vasopressor drug such as dopamine, adequate cardiac output can be restored and oxygen delivery improved. Once such drugs are employed, SvO₂ can guide the administration and titration, allowing the anesthetist to quickly observe the physiologic response.

By observing the trend of SvO₂ following a change in therapy, the effectiveness can be quickly evaluated. Often, the SvO₂ will change within minutes following modification in therapy. Without SvO₂ monitoring, time-consuming conventional tests to determine the effectiveness of therapy are usually necessary. Such tests include complete hemodynamic parameters, thermodilution cardiac output, arterial blood gas analysis or other diagnostic tests. If the SvO₂ improves and remains stable, such tests may be eliminated.⁴,⁶,¹² However, undesirable or unpredicted changes in SvO₂ can make diagnostic tests necessary to determine the reason for such changes.³ In a previous example, the titration of PEEP was guided using SvO₂. If an acceptable improvement is noted in SvO₂ following a change in PEEP, the clinician may elect not to obtain an arterial blood gas analysis. Another example includes reducing the number of routine thermodilution cardiac outputs if the SvO₂ remains stable, a measure that cuts cost and saves time.

SvO₂ is a sensitive parameter of physiologic changes in homeostasis. Therefore, SvO₂ monitoring can be used as an in vivo cardiopulmonary monitor and early warning device for impending hemodynamic changes. During general anesthesia and pharmacologic muscle paralysis, oxygenation and oxygen consumption will be maintained at a fairly stable level. Therefore, SvO₂ will closely trend the cardiac output and cardiopulmonary status.¹³ Studies have shown that changes in SvO₂ have been observed up to 20 minutes before a cardiopulmonary crisis occurs.¹⁴ This is supported by Stevens who has observed major variations in SvO₂ before changes occur in parameters measured by conventional monitors.¹⁵

While caring for postoperative cardiovascular patients, this author has observed substantial changes in SvO₂ before any clinical events occur. For example, a downward trend of SvO₂ can occur up to 10 minutes before there are signs or symptoms of impending cardiac tamponade. Other observations include a falling SvO₂ 5 minutes before an unexpected loss of blood pressure or cardiopulmonary arrest. In general, by maintaining a monitor alarm limit of 60 to 80, the anesthetist will be quickly alerted to clinical problems and intervene before patient compromise.

Decreases in SvO₂ have been shown to be predictive in nature. An SvO₂ of less than 60% for 15 minutes or longer with no other changes in common hemodynamic parameters has been shown to be an indicator of an unstable intraoperative and postoperative course.¹⁵ This instability refers to many circumstances such as the number of days in the postanesthesia or intensive care unit, vasopressor drug support and hypotensive episodes. This may allow the surgical team to construct a postoperative plan that includes close observation and appropriate monitoring, i.e., sending the patient to the intensive care unit or extending the normal recovery room time.

**Conclusion**

Continuous measurement of SvO₂ can provide the anesthetist with a direct link to the hemodynamic balance of the patient. Variation in SvO₂ can occur before clinical status changes or declines. Thus, rapid and early intervention can be provided and therapeutic intervention guided.

**REFERENCES**


Test Yourself Answers
(Questions appeared on page 520.)

1. The types of magnets used in MRI units are permanent, resistive and superconductive.
2. It is important to know the type of magnet being used because a superconductive magnet does not lose its magnetic field immediately when turned off. Therefore, safety precautions relative to the magnetic field must continue to be observed for a period of time should the magnet have to be turned off.
3. Some important safety concerns relative to the MRI environment are:
   (1) The magnetic field interferes with monitors.
   (2) Objects may have unobservable magnetic components and can become projectile unexpectedly.
   (3) Access to the patient is limited and the patient may experience claustrophobia in the unit.
   (4) Pacemaker patients are excluded from MRI because the magnetic field may cause damage to their pacers or lead migration may occur.
   (5) There is potential for heat problems associated with implants, and some implants can potentially be dislocated.
4. Some of the obstacles to practice in the MRI environment have been addressed with simple technology that is currently in use in the operating room. Ferromagnetic components in equipment have been replaced with nonferromagnetic components. The MRI unit has detachable components that will aid with access problems, and manufacturers are exploring ways to adapt monitoring devices.
5. The oscillometric blood pressure cuff can be used for monitoring blood pressure after extending the tubing length and making sure all connections are nonferromagnetic. A nonmagnetic precordial stethoscope with extended double layer tubing can provide auscultation access for breath and heart sounds. Although some interference does occur, ECG monitoring can be accomplished with shielded leads. A nonferromagnetic anesthesia machine has been introduced and should be available for distribution soon. Laryngoscopes and other small items have been produced from nonmagnetic materials such as plastic.

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