UNDERSTANDING CONTINUOUS MIXED VENOUS OXYGEN SATURATION (SvO₂) MONITORING WITH THE SWAN-GANZ OXIMETRY TD SYSTEM
UNDERSTANDING CONTINUOUS MIXED VENOUS OXYGEN SATURATION (SvO₂) MONITORING WITH THE EDWARDS SWAN-GANZ OXIMETRY TD SYSTEM

2nd Edition
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Although adequate tissue oxygenation is required to sustain life, it is often difficult to assess in critically ill patients. With the development of techniques for continuous monitoring of mixed venous oxygen saturation (SvO₂), the clinician has an additional parameter to use in evaluating tissue oxygenation. While intermittent cardiac output determinations and arterial blood gases provide information on oxygen supply, continuous SvO₂ monitoring reflects moment-to-moment dynamics between oxygen supply and demand. It reflects elements of both the pulmonary and circulatory systems, since SvO₂ measures the oxygen remaining in the blood after passing through the capillary bed. This measurement can now be done continuously using the Edwards Swan-Ganz Oximetry TD System.

The Pulmonary System

The cardiopulmonary system primarily regulates the transport of gases to and from the tissues, with the pulmonary system responsible for gas exchange (respiration). Within this system, gases move by simple diffusion from an area of high partial pressure and concentration to an area of low partial pressure and concentration.

In the lungs, alveolar PO₂ is 100 mm Hg as compared to a typical PO₂ of 40 mm Hg in the venous blood returning to the pulmonary capillaries. This creates a pressure gradient which favors the movement of oxygen from the alveolar space into the capillary blood. In contrast, PCO₂ is approximately 46 mm Hg in venous blood and 40 mm Hg in the alveolar space. This pressure gradient moves carbon dioxide from the blood entering the capillary network into the lungs for exhalation. Although the pressure gradient for carbon dioxide is lower than that for oxygen, its net diffusion rate is about twenty times greater because of its higher solubility.

In the periphery, the gas exchange process is reversed with regard to the direction of oxygen and carbon dioxide movement. The arterial blood, rich in oxygen as it enters the peripheral capillaries, has a PO₂ of about 100 mm Hg, whereas the adjacent tissues have a PO₂ as low as 30 mm Hg. Consequently, the oxygen moves from the blood into the tissues where it diffuses into the cellular mitochondria for ATP (adenosine triphosphate) formation. As the tissues consume oxygen to produce energy, the capillary PO₂ is reduced, stimulating hemoglobin to release its oxygen.

Continued oxygen consumption keeps PO₂ tissue levels low, maintaining the concentration gradient needed to drive oxygen extraction in the peripheral tissues. On the other hand, the carbon dioxide concentration is generally 50 mm Hg in the peripheral tissues and 40 mm Hg in the arterial blood entering the capillary network. This pressure gradient and the solubility of carbon dioxide cause the gas to diffuse into the capillaries.

This gas exchange process is extremely efficient. Under normal circumstances in the lungs, the arterial concentrations of both oxygen and carbon dioxide equilibrate to values almost equal to alveolar gas concentrations. In the periphery, both the amount of oxygen released into the tissues and the amount of carbon dioxide released from the tissues are regulated specifically by tissue activity. This establishes an efficient physical-chemical system by which gas exchange is controlled by the body tissue needs. (See Figure 1.)

The Cardiovascular System

The cardiovascular system supports the process of gas exchange through several mechanisms. It provides the blood medium in which the gases are carried, and the driving force to move the blood, which is reflected by cardiac output. Cardiac output (normally 4-8 liters per minute) is the product of stroke volume and heart rate. Stroke volume is influenced by preload, afterload and contractility. Preload refers to Starling’s Law which states that the more a myocardial fiber is stretched during diastole, the more it will shorten during systole, and it will shorten with greater force.
Preload is measured indirectly by the Swan-Ganz pulmonary artery catheter. It is measured either as pulmonary artery diastolic pressure (PADP) or pulmonary artery wedge pressure (PAWP) for the left ventricle and as right atrial pressure (RAP) for the right ventricle.

Afterload refers to the impedance to ejection of blood from the ventricle. In the clinical setting, the most sensitive measure of afterload is total peripheral resistance, a value derived by dividing mean arterial pressure (MAP) by cardiac output. Pulmonary vascular resistance can be similarly derived from mean pulmonary artery pressure and cardiac output, and it reflects right ventricular afterload.

Contractility is an inherent property of the myocardium which allows it to increase the extent and force of shortening independent of the Starling mechanism. There is no single measure which defines contractility; however, a variety of derived parameters can be considered.

The cardiovascular system also supports the gas exchange process by regulating blood flow to the different tissues. Normally there are several control systems that determine the blood flow rates for the various tissues, yet blood flow is not always matched to the tissues’ metabolic rate. For example, several organs, like the kidneys, are allocated high blood flow, but they require very little oxygen for their metabolic functions. Other organs, like the brain, possess both high flow rates and high extraction needs as well. When oxygen supply becomes limited, the cardiovascular system reroutes blood to restrict flow to tissues with low oxygen extraction. The cardiovascular system utilizes these high flow, low extraction areas as an oxygen reserve.

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**OXYGEN SUPPLY**

**Oxygen Loading**

Oxygen uptake occurs in the lungs and is the first step in the process of oxygen supply. Oxygen is carried in the blood in two forms:

- **Dissolved in plasma,** PO₂ (2%)
- **Combined with hemoglobin,** SO₂ (98%)

The partial pressure of oxygen (PO₂) is measured in mm Hg and reflects the tension or pressure that is exerted by oxygen when it is dissolved in plasma. A high PO₂ is required to dissolve even small amounts of oxygen in plasma. For a normal PO₂ of 100 mm Hg, there is only 0.31 ml of dissolved oxygen per 100 ml of blood. If this were the body’s only source of oxygen, then a resting cardiac output of nearly 120 liters per minute would be required to support life.

**Oxygen saturation** (SO₂) is a measurement of the amount of oxygen bound to hemoglobin (Hb). Hemoglobin consists of four iron-porphyrin groups (heme) attached to a protein (globin). Each heme molecule can carry one molecule of oxygen, so each molecule of hemoglobin can bind four molecules of oxygen. This binding process occurs rapidly and reversibly to facilitate the loading of oxygen in the lungs and the release of oxygen to the tissues.

\[
\text{Hb} + \text{O}_2 \leftrightarrow \text{HbO}_2
\]

Oxygen saturation is the ratio of the amount of oxygenated hemoglobin to the total hemoglobin in 100 ml of blood. It is frequently expressed as a percentage.

\[
\text{SO}_2 = \frac{\text{HbO}_2}{\text{Hb} + \text{HbO}_2} \times 100
\]

The oxygen saturation of arterial blood is normally 95-98%, whereas the saturation of venous blood is typically 60-80%. The saturation in venous blood is referred to as **mixed venous oxygen saturation** (SmO₂). It reflects the average saturation of venous blood as it returns to the right side of the heart from the various tissues. Generally, the tissues of a resting individual use approximately 25% of the available oxygen, leaving an oxygen reserve of 75% for periods of increased activity or physiologic stress.

The oxyhemoglobin dissociation curve (see Figure 2) graphically illustrates the relationship that exists between the partial pressure of oxygen (PO₂) and oxygen saturation.
(SO₂). The sigmoid shape of this curve reflects the optimal conditions that exist to facilitate oxygen loading in the lungs and oxygen release to the tissues. To describe these processes in relation to the curve, it is often divided into two segments: the association segment and the dissociation segment.

The association segment of the curve, or upper portion, is essentially flat and represents oxygen uptake in the lung. (See Figure 3.) In this portion of the curve, changes in PO₂ levels between 60 and 100 mm Hg cause only small changes in oxygen saturation. For example, at a normal arterial PO₂ of 90 mm Hg, the hemoglobin is 97% saturated. Even with a significant decrease in PO₂ to 60 mm Hg, the saturation only falls to 90%. This is advantageous in the lung where fluctuations in alveolar PO₂, and subsequently arterial PO₂, do not affect oxygen loading until PO₂ falls significantly lower than normal.

The lower portion of the curve (below 45 mm Hg) corresponds to the PO₂ levels of venous blood. (See Figure 4.) This steep part of the curve is referred to as the dissociation segment and represents the release of oxygen to the tissues. In this low range of PO₂ values, even small changes in oxygen tension produce large alterations in oxygen saturation. This is advantageous to the tissue because large quantities of oxygen can be extracted from the blood for relatively small decreases in PO₂. For example, at a PO₂ of approximately 40 mm Hg, hemoglobin remains 75% saturated and a large oxygen reserve remains; however, below a PO₂ of 30 mm Hg, the oxygen reserve is rapidly depleted.

Hemoglobin-oxygen affinity refers to the strength of the bond between hemoglobin and oxygen. It is expressed as the PO₂ value where hemoglobin is 50% saturated with oxygen and is referred to as the P50. The P50 is 27 mm Hg under standard conditions (pH 7.40, T 37º C, PCO₂ 40 mm Hg, normal 2,3 DPG).

Several factors affect the affinity of hemoglobin for oxygen. An increase in hydrogen ion concentration (↓pH), PCO₂, temperature, or 2,3 DPG (a byproduct of red blood cell metabolism) will decrease hemoglobin-oxygen affinity, thereby shifting the oxyhemoglobin curve to the right. (See Figure 5.) This shift results in a higher Po₂ value, indicating that a higher PO₂ will be required to saturate 50% of the hemoglobin. Under these conditions, there is a decrease in oxygen saturation for any given PO₂, but hemoglobin will release oxygen more readily to the tissues. However, since the amount of oxygen in the blood is decreased, the total amount of oxygen that can be released to the tissues is limited.
A shift of the oxyhemoglobin curve to the left (see Figure 5) represents an increase in the affinity of hemoglobin for oxygen. It can be caused by either a decrease in hydrogen ion concentration (pH), PCO₂, temperature, or 2,3 DPG. This shift to the left results in a lower P₅₀ value. For any given PO₂, oxygen saturation of the blood will be higher and oxygen loading in the lungs will be improved, but it will be more difficult for the oxyhemoglobin complex to dissociate at the tissue level.

**Blood Oxygen Content**

Blood oxygen content (CO₂) represents the total amount of oxygen in 100 ml of blood and is expressed in vol%, or milliliters of oxygen per deciliter of blood (ml/dl). It can be measured both in arterial (CaO₂) and venous (CvO₂) blood. Since oxygen exists in blood in two forms, the total oxygen content is calculated by adding the amount of oxygen dissolved in plasma to the amount carried by hemoglobin. The amount of oxygen dissolved in plasma is calculated by multiplying the PO₂ by .0031, the solubility coefficient for oxygen in plasma. The amount of oxygen bound to hemoglobin can be determined by multiplying 1.38 times the hemoglobin concentration times oxygen saturation, where the 1.38 represents the ml of oxygen that can be bound by 1 gram of hemoglobin.

\[
\text{CO₂} = \text{O₂ dissolved} + \text{O₂ bound to Hb} \\
= (0.0031 \times \text{PO₂}) (1.38 \times \text{Hb} \times \text{SO₂})
\]

Since 98% of the oxygen in the blood binds to hemoglobin, the formula for oxygen content can be simplified by ignoring the amount dissolved in plasma. Oxygen content, therefore, is highly dependent on hemoglobin concentration and saturation. Oxygen content can be predicted because the relationship between oxygen content and saturation is linear for any given level of hemoglobin. Changes in saturation are directly related to changes in content as long as the hemoglobin remains constant. (See Figure 6.)

![Shift in the oxyhemoglobin dissociation curve resulting from changes in the oxygen affinity of hemoglobin](image)

**Figure 5**

**Relationship of oxygen saturation to oxygen content**

Given a normal value of 15 gms of hemoglobin per 100 ml of blood, the normal blood oxygen content can be calculated. Assuming an arterial saturation of 97%, normal CaO₂ is 20.1 vol%. On the venous side where the oxygen saturation is 75%, CvO₂ is normally 15.5 vol%. This represents an arterial-venous oxygen content difference, or oxygen extraction, of 5 vol%. Based on these values, the tissues generally use 25% of the oxygen delivered to them, returning 75% to the lungs.

\[
\begin{align*}
\text{CaO₂} &= 1.38 \times \text{Hb} \times \text{SaO₂} \\
\text{CvO₂} &= 1.38 \times \text{Hb} \times \text{SvO₂} \\
\text{CaO₂} &= 1.38 \times 15 \times 97\% \\
\text{CvO₂} &= 1.38 \times 15 \times 75\% \\
\text{CaO₂} &= 20.1 \text{ vol}% (\text{ml/dl}) \\
\text{CvO₂} &= 15.5 \text{ vol}% (\text{ml/dl})
\end{align*}
\]
Oxygen Transport

Oxygen transport/delivery (DO₂) is the amount of oxygen delivered to the tissues and is measured in ml of oxygen per minute. Oxygen transport is dependent upon two key factors. The first is the ability of the lungs to oxygenate blood as it passes through the pulmonary capillary network. This ability is reflected by the oxygen content of arterial blood, which is a function of hemoglobin and saturation. Oxygen transport also depends on the heart to maintain adequate blood flow to the tissues. This is reflected by cardiac output. The formula for oxygen transport is:

\[ \text{O₂ Transport} = \text{Cardiac Output} \times \text{Oxygen Content} \times 10 \]

*10 converts oxygen content to ml per minute

With a normal cardiac output of 5 liters per minute and a CaO₂ of 20.1 vol%, normal oxygen transport is 1005 ml of oxygen per minute. This value represents total oxygen delivery. Substituting CvO₂, this same equation can be used to determine venous oxygen transport, the amount of unused oxygen returning to the heart. Normal venous oxygen transport is 775 ml of oxygen per minute.

Arterial O₂ Transport = \( CO \times \text{CaO}_2 \times 10 \)
\[ = 5 \times 20.1 \times 10 \]
\[ = 1005 \text{ ml O}_2/\text{min} \]

Venous O₂ Transport = \( CO \times \text{CvO}_2 \times 10 \)
\[ = 5 \times 15.5 \times 10 \]
\[ = 775 \text{ ml O}_2/\text{min} \]

OXYGEN DEMAND

Oxygen Demand

Oxygen demand is the amount of oxygen necessary to satisfy the metabolic requirements of all body tissues. It is determined by metabolic rate, which is frequently modified by temperature, metabolic conditions and muscular work. Total oxygen delivery must equal total oxygen demand to maintain organism homeostasis. (See Figure 7)

\[ \text{Arterial O}_2 \text{ Transport} = \text{CO} \times \text{CaO}_2 \times 10 \]
\[ = 5 \times 20.1 \times 10 \]
\[ = 1005 \text{ ml O}_2/\text{min} \]

\[ \text{Venous O}_2 \text{ Transport} = \text{CO} \times \text{CvO}_2 \times 10 \]
\[ = 5 \times 15.5 \times 10 \]
\[ = 775 \text{ ml O}_2/\text{min} \]

Oxygen Consumption

Oxygen consumption (VO₂) is the amount of oxygen actually used by the tissues. In healthy individuals, the oxygen transport system ensures that oxygen consumption and demand are equal. (See Figure 8.) During disease states, however, oxygen demand may exceed consumption, causing tissue oxygen deprivation.

\[ \text{Oxygen Consumption} = \text{Arterial Oxygen Transport} - \text{Venous Oxygen Transport} \]
\[ = (\text{CO} \times \text{CaO}_2 \times 10) - (\text{CO} \times \text{CvO}_2 \times 10) \]
\[ = \text{CO} \times (\text{CaO}_2 - \text{CvO}_2) \times 10 \]
\[ = \text{CO} \times (\text{Hb} \times \text{SaO}_2 \times 13.8) - \text{CO} \times (\text{Hb} \times \text{SvO}_2 \times 13.8) \]
\[ = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]
The formula for oxygen consumption is actually a simple restatement of the Fick equation. This equation identifies all of the pertinent variables of oxygen supply and demand.

\[ \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

Oxygen Cardiac Hemo- K Arterial Venous Consumption Output globin Factor O2 Saturation

Although the actual oxygen demand and the value calculated for oxygen consumption may differ, oxygen consumption is the best index of tissue oxygen requirements available to the clinician. Normal oxygen consumption is 230 ml of oxygen per minute (1005 ml O2/min – 775 ml O2/min = 230 ml O2/min).

**ANALOGY FOR MIXED VENOUS OXYGEN SATURATION**

Oxygen is carried in the blood primarily bound to the hemoglobin molecule. This uptake occurs in the lungs and normally results in complete saturation of hemoglobin with oxygen. In this analogy, the blood is compared to a train (see Figure 9a) consisting of boxcars (hemoglobin molecules). These boxcars have been filled (saturated) with a valuable product (oxygen) at the loading station (the lungs).

![Figure 9a](Loading)

\[ \text{Oxygen Content} = (\text{Hb} \times 1.38 \times \text{Sao}_2) + (0.0021 \times \text{PO}_2) + \text{O}_2 \text{ Dissolved} \]

Just as an engine is required to move the train, the pumping action of the heart, reflected by cardiac output, is necessary to transport the blood. (See Figure 9b.)

![Figure 9b](Transport)

\[ \text{O}_2 \text{ DELIVERY (TRANSPORT)} = \frac{\text{Cardiac Output} \times \text{Arterial O}_2 \text{ Content} \times 10}{(\text{CO} \times \text{Hb} \times \text{SaO}_2 \times 1.38 \times 10)} \]
As the train progresses along the track, it stops at multiple depots. A portion of the product carried by the train is unloaded at each of these depots. The number of empty boxcars represents the amount of product that has been unloaded. Similarly, as blood flows from the left ventricle through the capillaries, oxygen is needed to meet the metabolic needs of the tissues. The difference between the amount of oxygen carried to the tissues (arterial oxygen delivery) and the amount of oxygen returned to the heart (venous oxygen delivery) indicates the total amount of oxygen consumed by the tissues. (See Figure 9c.)

Those boxcars filled with oxygen as the train returns to the loading station represent the amount of product that was not used, and therefore, not unloaded at the depots. In the same respect, mixed venous oxygen saturation reflects the amount of oxygen returning to the pulmonary capillaries, since it was not needed by the tissues to support metabolic function. (See Figure 9d.)
DYNAMICS OF OXYGEN SUPPLY/DEMAND

A number of factors can alter the balance between oxygen supply (delivery) and demand (consumption). A decrease in supply (↓O2, ↓Hb, ↓SaO2) or an increase in demand (↑VO2) are both threats to this delicate balance.

Although oxygen is a requirement for normal cellular function, the body possesses no stores of this valuable substance. In fact, it is estimated that a 70 kg man has only about 1500 ml of oxygen in his body at any given moment. In the absence of an adequate oxygen supply, metabolic activity changes from the normal aerobic pathway to the less efficient anaerobic pathway. This mechanism produces lactic acid as an end product and yields a metabolic acidosis, which can be identified by an increase in the blood lactate level. Consequently, lactic acidosis is presumptive evidence of tissue hypoxia and is associated with a poor prognosis.

Compensatory Mechanisms

When the balance between oxygen supply and demand is threatened, the body mobilizes its compensatory mechanisms to ensure adequate oxygen availability. The two most important ones are an increase in cardiac output and an increase in oxygen extraction.

An increase in cardiac output is the primary mechanism that the body uses to compensate for either a decrease in oxygen supply or an increase in oxygen demand. Evidence indicates that this increase in cardiac output is achieved mainly by an increase in the rate and inotropic state of the heart. Healthy individuals can increase cardiac output threefold.

An increase in oxygen extraction is the second fundamental compensatory mechanism. When an increased need for oxygen exists, the body can extract more than the usual 25% of oxygen from arterial blood as it passes through the capillary network. Enhanced oxygen extraction is reflected by a larger arterial-venous saturation difference and a lower oxygen content in venous blood (↓SvO2). When the oxygen balance is disrupted, the removal of oxygen is facilitated by the effects of pH on the affinity of hemoglobin for oxygen; when there is insufficient oxygen, lactic acid is formed causing a reduction in cellular pH. Under these acidic conditions, the oxyhemoglobin curve shifts to the right because the affinity of hemoglobin for oxygen decreases, facilitating oxygen unloading at the tissues.

Typically, normal oxygen extraction is approximately 22-25%. Under conditions of extreme demand, a normal person can extract nearly 75% of the oxygen delivered to the tissues. Critically ill patients are at threat when their compensatory oxygen extraction reaches close to 50%, especially for a prolonged period of time. Oxygen extraction indices evaluate if the compensatory increase in cardiac output was sufficient to meet the increased tissue demands.

Figure 10 shows an extreme example of the potential compensatory responses. The cardiac output increased threefold from 5 to 15 liters per minute. Oxygen extraction can be assessed by evaluating SaO2 – SvO2 (arterial-venous oxygen saturation difference) which is denoted as changing from 22 to 66% as the SvO2 decreases to 31%. Even though oxygen consumption increased to nine times the normal range, the increase in oxygen extraction leads the clinician to conclude that the increase in cardiac output was insufficient to meet the tissue demands.

\[ \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times \left( \text{SaO}_2 - \text{SvO}_2 \right) \]

![Figure 10](image)

Effective compensatory mechanisms are essential for maintaining homeostasis. If the responses are inadequate, oxygen balance is compromised and may be assessed by measuring oxygen extraction. In a compromised state, the threat of developing tissue hypoxia and subsequent oxygen debt becomes greater.

![Figure 11](image)

To help the body compensate further, the cardiovascular system selectively redistributes blood flow to areas of greatest oxygen need. For example, when a tissue's oxygen
needs are not being met, vasoactive metabolites are formed to dilate vessels and enhance blood flow. Meanwhile, in areas of low extraction (i.e., skin, renal, splanchnic beds), sympathetic vasoconstrictive fibers increase vascular resistance. Hence, blood is diverted away from regions of high flow/low extraction and is redistributed to sites where oxygen need is greatest. Although this mechanism is extremely beneficial, it is not elicited until local metabolite accumulation has occurred, and it is not a response that can be measured clinically. Therefore, unlike alterations in cardiac output and oxygen extraction, it possesses little utility as a warning of an imbalance in oxygen supply and demand.

**Specific Threats to Oxygen Balance**

An increase in oxygen consumption (VO₂) is a threat to the balance of oxygen supply and demand. Typically the body consumes the oxygen it needs provided there is adequate oxygen delivery. Once the demand increases, the body’s compensatory mechanisms are activated to maintain sufficient delivery. Shivering, seizures and fever are a few of the clinical situations that cause an increase in oxygen consumption.

\[ \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

If VO₂ increases in a healthy individual, cardiac output will increase to maintain the balance between oxygen delivery and demand/consumption.

\[ \Rightarrow \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

If additional oxygen is required, then extraction of available oxygen will increase, causing a fall in S-O₂.

\[ \Rightarrow \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

With these two mechanisms, 2049 ml of oxygen per minute is the maximum amount of oxygen that can be supplied.

\[ \text{VO}_2 = 15 \times 15 \times 13.8 \times (0.97 - 0.31) = 2049 \text{ ml O}_2/\text{min} \]

In the critically ill patient with inadequate cardiac reserve, the response to an increase in oxygen consumption is restricted to the patient’s ability to extract more of the available oxygen, thus decreasing SvO₂.

\[ \Rightarrow \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

A decrease in hemoglobin represents another threat to oxygen balance. Under normal circumstances, changes in hemoglobin concentration occur gradually. However, frank hemorrhage or concealed intradominal bleeding can cause a rapid decrease in hemoglobin.

\[ \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

In the healthy individual with mild anemia, cardiac output generally increases to augment the delivery of oxygen.

\[ \Rightarrow \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

Increasing oxygen extraction is a second compensatory mechanism that enhances the availability of oxygen.

\[ \Rightarrow \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

Even in patients with profound anemia, the heart can compensate to varying degrees. In this example the patient has an abnormally low hemoglobin concentration of 1.7 gm%. However, an increase in cardiac output and a decrease in oxygen saturation assure that normal oxygen needs are met and lactic acidosis is avoided.

\[ \text{VO}_2 = 15 \times 1.7 \times 13.8 \times (0.97 - 0.31) = 232 \text{ ml O}_2/\text{min} \]

If the patient cannot increase cardiac output sufficiently, S-O₂ decreases as capillary blood is maximally desaturated.

\[ \Rightarrow \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

A decrease in SaO₂ also constitutes a threat. However, in most instances, even in patients with severe pulmonary disease, the SaO₂ will remain above 90% and pose little danger to the dynamics of oxygen balance.

\[ \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

If the SaO₂ decreases to an unacceptable level, the body responds first by increasing cardiac output.

\[ \Rightarrow \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

If this activity is inadequate, then oxygen extraction increases.

\[ \Rightarrow \text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]
The hypoxemic patient in the example below has an $\text{SaO}_2$ of 38%. Regardless, he can still compensate by increasing cardiac output and oxygen extraction. For this reason, uncomplicated hypoxemic patients usually do not develop lactic acidosis.

$$\text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2)$$

$$= 15 \times 15 \times 13.8 \times (0.38 - 0.31)$$

$$= 217 \text{ ml O}_2/\text{min}$$

In the event of inadequate or nonexistent cardiac reserve, the only available mechanism is to increase oxygen extraction.

$$\text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2)$$

A fall in cardiac output is perhaps the most serious threat to the patient because an inadequate cardiac output eliminates one of the key compensatory mechanisms. Therefore, proportionately, a patient cannot tolerate as great a fall in cardiac output as he can a decrease in $\text{SaO}_2$ or hemoglobin before lactic acidosis ensues. This is why perfusion failure is the most common etiology of lactic acidosis in clinical practice.

$$\text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2)$$

As cardiac function deteriorates and cardiac output decreases, the body’s last resort is to extract more available oxygen, which causes a decrease in $\text{SvO}_2$.

$$\text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2)$$

The example below shows a patient in shock. By extracting more oxygen from the capillary blood, the body can compensate for a cardiac output of 1.7 liters per minute, provided there are no further threats to oxygen balance.

$$\text{VO}_2 = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2)$$

$$= 1.7 \times 15 \times 13.8 \times (0.97 - 0.31)$$

$$= 232 \text{ ml O}_2/\text{min}$$

A normal cardiac output of 5 liters per minute and a normal oxygen extraction of 5 vol% will provide 250 ml of oxygen per minute. (See Figure 11.) If cardiac output doubles and there is no alteration in oxygen consumption, then oxygen extraction will be reduced by half. If there is no alteration in oxygen consumption, but cardiac output is reduced to half its normal value, then oxygen extraction will have to double to deliver adequate oxygen to the tissues.

**MIXED VENOUS OXYGEN SATURATION**

$\text{SvO}_2$ represents the end result of both oxygen delivery and consumption at the tissue level.

$$\text{SvO}_2 = \frac{\text{Oxygen Delivered} - \text{Oxygen Consumed}}{(\text{SaO}_2, \text{Hb, CO}) (\text{VO}_2)}$$

When a threat to normal oxygen supply/demand occurs, the body attempts to compensate, and its success is immediately reflected by $\text{SvO}_2$. If the $\text{SvO}_2$ value is normal, there is sufficient oxygen supply available to the tissues. However, if the $\text{SvO}_2$ value is low, then either the oxygen supply is insufficient or the oxygen demand is elevated. Regardless of the cause, a decrease in $\text{SvO}_2$ indicates that the body has called upon its last line of defense to preserve oxygen balance and therapeutic interventions may be appropriate.

**Measurement**

The pulmonary artery is the site where $\text{SvO}_2$ values should be measured. It is important to sample only at this site to allow for adequate mixing of blood from the superior and inferior vena cavae and coronary sinus. $\text{SvO}_2$ can be monitored continuously in the pulmonary artery with the Swan-Ganz Oximetry TD System; the system measures thermodilution cardiac output intermittently, or continuously with the CCOmbu catheter technology. (See Figure 12.)

![Swan-Ganz Oximetry TD Catheter](image-url)
Abnormal SvO₂ Values

The normal range for SvO₂ is 60 – 80%. If the SvO₂ value is normal, then the clinician may assume that there is adequate tissue perfusion. If SvO₂ falls below 60%, a decrease in oxygen delivery and/or an increase in oxygen consumption should be suspected. When SvO₂ falls below 40%, the body’s ability to compensate is limited, and oxygen is relatively unavailable for use by the tissues. Table 2 illustrates various clinical circumstances that can create an imbalance between oxygen supply and demand.

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The technology for measuring SvO₂ is based on reflection spectrophotometry (See Figure 13.) This involves transmitting light of selected wavelengths down one fiberoptic filament in the catheter body to the blood flowing past the catheter tip. The reflected light is then transmitted back through the second fiberoptic filament to a photodetector located in the optical module. Since hemoglobin and oxyhemoglobin absorb light differently at the selected wavelengths, the reflected light can be analyzed to determine the percent SvO₂.

Two wavelengths of light were selected based on the different absorption characteristics of hemoglobin and oxyhemoglobin. One wavelength is sensitive to changes in oxygen saturation and the other is not. These two wavelengths are both subject to the same interferences, so when a ratio of the readings is used to calculate SvO₂, the effects of these interferences are cancelled.

The most significant interference in SvO₂ measurements is usually caused by changes in hematocrit. To correct for this, the Swan-Ganz Oximetry TD System has several calibration curves generated at different hematocrits. During calibration, the patient’s hematocrit value is entered, and the system selects the corresponding calibration curve. (See Figure 14.)

Measuring SvO₂ at two wavelengths and using calibration curves generated for specific hematocrit levels contribute to the system’s accuracy over a broad range of saturations and hematocrits. In critically ill patients with saturations below 50%, this accuracy is important since clinical interventions may be necessary to either avoid, or reverse, life-threatening situations.
If SvO₂ levels are elevated above 80%, an increase in oxygen supply and/or a decrease in demand should be suspected. An increase in oxygen delivery can be caused by an increased FiO₂. A decrease in oxygen consumption can be seen in hypothermic states or in patients who are anesthetized and mechanically ventilated or pharmacologically paralyzed.

A wedged pulmonary artery catheter can also cause high SvO₂ values. When the balloon on a pulmonary artery catheter is inflated, the blood distal to the balloon stagnates. This blood absorbs oxygen from surrounding alveoli, and its saturation approaches that of arterial blood. If this artificial increase in SvO₂ occurs, however, the oximeter will flash an alert.

Occasionally, normal or increased SvO₂ values are observed in a patient, who, by all other criteria, demonstrates compromised tissue oxygenation. Three etiologic mechanisms have been postulated for this observation: arterial admixture, abnormalities in distribution of blood flow, and histotoxic hypoxia.

Arterial admixture occurs when mixed venous blood becomes contaminated by arterial blood. When this happens, total oxygen delivery (QT or cardiac output x CaO₂ or arterial oxygen content) is divided peripherally into two fractions. These fractions include oxygen delivery to the tissues (Qc x CaO₂) and oxygen delivery to the arterial-venous shunt (Qs x CaO₂). (See Figure 15.)

Since this division reduces oxygen delivery to the tissues, oxygen consumption is limited, and the tissues’ demand for oxygen is not met. As a result, tissue hypoxia develops. The blood emanating from the shunt, however, has the same oxygen saturation as arterial blood, since it bypasses the tissues. The oxygen content of the mixed venous blood reflects the mixture of the oxygen-rich, shunted blood and the blood leaving the tissue. This causes an increased SvO₂ in spite of tissue oxygen deprivation. This phenomenon is believed to be one of the mechanisms occurring in sepsis.

Abnormalities in the distribution of blood flow can also account for increases in SvO₂ in the presence of inadequate oxygen transport. Under these circumstances, blood flow is normally restricted in tissues with low extraction to provide additional flow to tissues with high demand for oxygen. If this redistribution of blood does not occur, then hypoxia will develop in those tissues receiving insufficient oxygen, while the tissues with low extraction still receive abundant blood flow. The blood returning from low extraction tissues has a high oxygen content which increases SvO₂ even though the other tissues are hypoxic.

A decrease in oxygen consumption can also be caused by tissues whose oxidative enzymes are blocked or only partially functional. As a result, the oxygen content of venous blood resembles that of arterial blood, and SvO₂ is elevated. The tissues, however, are oxygen-starved. This mechanism is referred to as histotoxic hypoxia and is believed to be exhibited in cyanide poisoning with nitroprusside therapy.

When utilizing continuous SvO₂ monitoring, the clinician should note when measurements move outside the normal range. In addition, any deviation greater than 10% from baseline (even within normal limits) should be considered significant if it persists for longer than 3-5 minutes. In the event of a change in SvO₂, the clinician should determine the specific cause by examining the variables that affect the balance of oxygen supply and demand and take appropriate action.

**Catheter Tip Migration**

As with any pulmonary artery catheter, the oximetry catheter can potentially migrate into the pulmonary bed. The oxygen saturation value will be affected by the vessel wall’s absorption properties and may be inaccurate under these conditions. Also, damage from migration may occur by prolonged occlusions or by overdistension of the vessel upon reinflation of the balloon. Although migration is typically monitored by catheter tip pressures and X-ray film, the oximetry catheter’s fiberoptics can also be used to detect the periphery of the pulmonary bed. The light that is used to
measure SvO₂ may also come into contact with the vessel wall, particularly when the catheter has advanced too far in the vasculature. Edwards oximeter systems will detect this situation and display a pulsatility alert. Disabling the pulsatility alert is not a solution to the migration problem and should not be encouraged.

Migration is often seen in cardiac surgery cases, particularly when the heart is being manipulated. It is also common in active and/or agitated patients and in some ventilated patients. The solution to catheter migration is to reposition the catheter. Deflate the balloon and pull back the catheter 1 to 3 cm. Rewedge by inflating the balloon, checking balloon inflation volume, until a wedge pressure tracing is obtained. If a wedge is obtained at a balloon volume substantially less than 1.25 ml, pull the catheter back to a position at which full or near full inflation volume produces a wedge pressure tracing. Repositioning the catheter should: 1) correct the pulsatility alert, 2) improve the accuracy of the SvO₂ measurement, 3) reduce the risks of pulmonary artery rupture and/or pulmonary infarction, and 4) improve the accuracy of cardiac output determinations and PAWP.

To obtain the most reliable information, it is good practice to periodically verify catheter tip position whenever hemodynamic monitoring is indicated. There are many ways to identify a migration problem. The key techniques are as follows:

– **Monitor catheter tip pressure**
  If a wedge pressure tracing is observed when the balloon is deflated, the catheter has migrated. The sudden appearance of a respiratory artifact in the pressure waveform may also indicate migration.

– **Monitor balloon inflation volume necessary to obtain PAWP**
  If PAWP is obtained at a volume less than 1.25 ml, poor positioning of the catheter tip should be suspected. The Swan-Ganz balloon typically “pops open” at a volume greater than 0.7 ml. PAWP is obtained with less volume; the tip is probably in a small vessel or bifurcation, which can affect the accuracy of the oxygen saturation measurement.

– **Observe SvO₂ value before, during, and after PAWP measurement**
  Upon balloon inflation, SvO₂ value should increase rapidly and dramatically to reflect the reflux of arterial blood to the catheter tip. Upon deflation, the SvO₂ value should return to its initial value before wedging. If either of these conditions does not occur, catheter repositioning should be considered, even if an acceptable PAWP was obtained.

– **Observe the pulsatility and intensity alerts**
  The presence of these alerts, particularly in conjunction with any of the preceding conditions, strongly suggests that catheter position is less than optimal and may be affecting the validity of the oxygen saturation measurement. For details regarding these alerts, refer to the oximeter Operations Manual.
CLINICAL APPLICATIONS

SvO₂ is a sensitive indicator of the patient’s status and generally precedes other indications of cardiopulmonary instability. Since continuous SvO₂ monitoring alerts the clinician to a change in the patient’s condition sooner than traditional methods, diagnostic and therapeutic decisions can be made earlier in the patient’s clinical course.

Generally, continuous SvO₂ monitoring can be useful as:

- Surveillance and early warning system.
- Guide for adjusting and assessing therapy and routine nursing care.
- Means for interpreting other variables.

**Surveillance and Early Warning**

Case No. 1: Shivering

The patient in Figure 16 maintained a normal SvO₂ during an extensive and prolonged operative event, yet in the recovery room SvO₂ decreased suddenly to 50%. The logical assumption was that cardiac output had decreased. However, thermodilution cardiac output indicated that cardiac output had actually increased. The patient was examined and found to be shivering. Shivering increased oxygen consumption, which the body attempted to meet by increasing cardiac output. Since this was insufficient, increased amounts of oxygen were extracted from the blood, causing a decrease in SvO₂. A paralyzing agent was administered and oxygen consumption returned to normal. This eliminated the need for increased oxygen consumption, therefore, cardiac output and SvO₂ returned to normal limits.

Case No. 2: Septic Shock

The patient in Figure 17 was admitted in profound cardiogenic shock with an SvO₂ of only 35%. Various aggressive therapies were initiated without any improvement. Several hours later, SvO₂ increased for no identifiable reason. The cause was found when the patient’s temperature spiked to 104°F. The patient was septic. In this case, the change in SvO₂ toward the normal range did not herald the patient’s survival. Peripheral shunting involved in septic shock caused large amounts of oxygenated blood to be returned to the heart. Traditionally a high cardiac output and low peripheral vascular resistance have been considered indications of sepsis. Perhaps now an unexplained rise in SvO₂ may be considered diagnostic for sepsis as well.

**Guide for Adjusting and Assessing Therapy and Routine Nursing Care**

Case No. 3: Routine Nursing Care

In the example shown in Figure 18, a small and then larger drop in SvO₂ accompanied turning and suctioning the patient, respectively. The SvO₂ remained depressed and preceded cardiac arrest by 15 – 20 minutes.
SvO₂ monitoring on a continuous basis may signal the clinician when to modify care. In this case, perhaps suctioning should have been delayed until SvO₂ returned to normal, and cardiac arrest might have been avoided.

Case No. 4: Suctioning

Various therapeutic maneuvers may interfere with the concentration of inspired oxygen. The case illustrated in Figure 19 represents the effects of tracheobronchial suctioning. The fall in SvO₂ is profound and prolonged. The clinician, after observing such a change in SvO₂, might have elected to preoxygenate this patient prior to repeated bouts of suctioning.

In the case represented in Figure 20, an IV infusion of dobutamine was disrupted. SvO₂ fell immediately and remained low until the infusion was reestablished. This patient was dependent on this drug to maintain cardiac output.

In the case illustrated in Figure 21, nitroprusside was used to reduce afterload. As nitroprusside was titrated, cardiac function improved. If only intermittent SvO₂ sampling was done, this improvement in cardiac function could have been missed.

Continuous SvO₂ monitoring is also useful to track changes in respiratory therapy. The case shown in Figure 22 shows the usefulness of SvO₂ in weaning a patient from the respirator. The patient was receiving an FIO₂ of 0.7 and maintained an SvO₂ value in the normal range. A reduction in FIO₂ did not cause an appreciable drop in SvO₂. Despite the FIO₂ reduction, oxygen delivery remained adequate. Blood gases were not drawn.
Continuous \( \text{SvO}_2 \) Monitoring is a convenient and accurate means to adjust PEEP rapidly to optimal levels without repeated blood gases and cardiac output determinations. At higher levels of PEEP, usually above 10 cm of water, there may be interference with cardiac output that outweighs the benefit of improved pulmonary gas exchange. In this example (see Figure 23), PEEP was titrated using \( \text{SvO}_2 \) values to assess what was happening at the tissue level.

The use of intra-aortic balloon counterpulsation may be required for circulatory assistance. The pump augments diastolic aortic pressure, reduces left ventricular afterload, facilitates coronary perfusion, and reduces myocardial oxygen consumption and myocardial ischemia. Continuous \( \text{SvO}_2 \) monitoring can be helpful in assessing the efficacy of therapy as well as in the weaning procedure. As the patient in Figure 24 was decreased from 1:1 to 1:2 counterpulsation, \( \text{SvO}_2 \) fell substantially. With reinstitution with 1:1 counterpulsation, \( \text{SvO}_2 \) improved. Continuous \( \text{SvO}_2 \) monitoring identified the hemodynamic changes immediately in this patient.

A normal cardiac output is frequently mistaken to represent a normal flow of oxygen to the vital organs. However, cardiac output is only one variable in the equation for oxygen delivery. It does not provide information about the availability of oxygen relative to the demand at the tissue level. A cardiac output of 6 liters per minute coupled with an abnormally low \( \text{SvO}_2 \) should prompt the clinician to investigate further and/or institute therapy. (See Figure 27.)

### Means for Interpreting Other Variables

In certain situations, a normal blood pressure or cardiac output may be inadequate. Conversely an abnormal blood pressure or cardiac output may, in fact, be adequate. \( \text{SvO}_2 \) monitoring can assist the clinician in making this decision.

A fall in systolic blood pressure may indicate decreased perfusion. On the other hand, it may represent a decrease in systemic vascular resistance and improved perfusion. The therapeutic implications are quite different in these two instances. If the blood pressure falls by 20 mm Hg with no change in \( \text{SvO}_2 \) or with an \( \text{SvO}_2 \) in the normal range, it is likely that no therapy is indicated. If, however, that same drop in blood pressure is accompanied by a fall in \( \text{SvO}_2 \) or by an \( \text{SvO}_2 \) outside the normal range, therapy may need to be instituted quickly. (See Figures 25 and 26.)
What Do You Do If:
• CO = 6.0 L/minute
• SvO₂ = 45%

On the other hand, a cardiac output of 2.1 liters per minute may often be interpreted as inadequate. However, when coupled with a normal SvO₂ (as with a hypothermic patient), this cardiac output may be considered adequate. Many clinicians may no longer find it necessary to treat “low” cardiac output states in the face of a normal SvO₂. (See Figure 28.)

What Do You Do If:
• CO = 2.1 L/minute
• SvO₂ = 70%

Incorporating Continuous Cardiac Output and SvO₂ at the Bedside

Advances in technology have led to the development of a means to assess cardiac output on a continuous basis at the bedside. Standard bedside assessment of cardiac output has been performed using the Swan-Ganz pulmonary artery catheter and thermodilution technology. A known amount of solution at a known temperature is injected into the vena cava or right atrium, and the resulting temperature is measured downstream in the pulmonary artery by a thermistor. The computer applies a modified Stewart-Hamilton equation to the washout curve which determines the cardiac output on an intermittent basis.

By adapting intermittent thermodilution principles, continuous cardiac output (CCO) can now be obtained at the bedside. The system consists of a modified Swan-Ganz catheter and advanced cardiac output computer. Rather than using a cooler than blood temperature injectate for the input signal, as is done with the intermittent method, the catheter has a 10 cm thermal filament that emits a pulsed pseudo-randomized on-off energy signal in a 30-60 second sequence. The sophisticated computer algorithm identifies when the pulmonary artery temperature change matches the input signal. Cross-correlation of the input and output signal produces a thermodilution washout curve. The modified Stewart-Hamilton equation is applied to determine the cardiac output value. This process occurs approximately every 30-60 seconds and the values are averaged to produce a continuously displayed parameter. CCO technology eliminates many of the technique sources of error associated with the bolus method.

Assessment parameters for tissue oxygenation require all the components of oxygen delivery as well as the measurement of SvO₂. SvO₂ is a valuable index of the dynamic relationship between the patient’s oxygen balance: DO₂ and VO₂. SvO₂ does not reflect changes in any one component of the determinants of oxygen delivery and consumption, but rather changes in the global relationship between DO₂ and VO₂.

Figure 29 shows a patient example where the combining of the two assessment parameters provided the clinician with additional information in a timely manner. The patient showed a normal SvO₂ trend value. Upon assessing the CCO, it was noted that CO was decreasing significantly. The impact of peripheral perfusion, redistribution of blood flow, and regional oxygen requirements are often not completely noticed by the global parameter of SvO₂. By monitoring
either CCO or SvO₂ alone, the impact of the response to patient interventions may have been lost.

In Figure 30 the CCO was increasing which would lead the clinician to assume that oxygen delivery was improving. However, during patient movement and stress, the SvO₂ was noted to decrease. This reflects that the first compensatory response of increased cardiac output was insufficient to meet demands since the SvO₂ values decreased as a result of increased oxygen extraction.

**SUMMARY**

The clinician is now able to monitor the major component of oxygen delivery, cardiac output, and the index of oxygen balance, SvO₂, on a continuous basis. By incorporating pulse oximetry into a special computer system such as the Edwards Vigilance monitor, on-line assessment of oxygen extraction can also be obtained. Combining all of these important cardiorespiratory variables provides the means to assess individual oxygen variables and serves as an excellent surveillance and warning system. It is also a time-saving and cost-effective tool to institute and adjust therapies and a means to interpret other variables.

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TERMS AND VALUES

**Arterial-Venous Oxygen Content Difference (extraction)** – The difference between arterial oxygen content and mixed venous oxygen content.

Normal Value: 4-6 vol%

\[ A-\text{VO2 diff} = \text{CaO}_2 - \text{CvO}_2 \]

\[ = 1.38 \times \text{Hb} \times (\text{SaO}_2 - \text{SvO}_2) \]

**Mixed Venous Oxygen Saturation (SvO₂)** – The percentage of hemoglobin saturated with oxygen in the venous blood as measured in the pulmonary artery. This represents the oxygen "reserve" — that amount of oxygen that can be utilized in periods of increased demand.

Normal Value: 60-80%

**Oxygen Consumption (VO₂)** – The amount of oxygen used by the tissues per minute. It is determined by subtracting the amount of oxygen returning to the heart from the amount of oxygen delivered to the tissues.

Normal Value: 230 ml O₂/min

\[ \text{VO}_2 = \text{CO} \times \text{A-VO2 diff} \]

\[ = \text{CO} \times \text{Hb} \times 13.8 \times (\text{SaO}_2 - \text{SvO}_2) \]

**Oxygen Content (CO₂)** – The amount of oxygen carried by 100 ml of blood.

**Arterial Oxygen Content (CaO₂)** – The total amount of oxygen in 100 ml of arterial blood equal to the sum of the amount combined with hemoglobin and the amount dissolved in plasma.

Normal Value: 20.1 vol% (ml/dl)

\[ \text{CaO}_2 = (1.38 \times \text{Hb} \times \text{SaO}_2) + (0.0031 \times \text{PaO}_2) \]

**Venous Oxygen Content (CvO₂)** – The total amount of oxygen in 100 ml of venous blood equal to the sum of the amount combined with hemoglobin and the amount dissolved in plasma.

Normal Value: 15.5 vol% (ml/dl)

\[ \text{CvO}_2 = (1.38 \times \text{Hb} \times \text{SvO}_2) + (0.0031 \times \text{PvO}_2) \]

**O₂ Saturation** – The amount of hemoglobin bound with oxygen. It is determined by dividing the amount of oxygen carried by hemoglobin over the amount of oxygen that could be carried by hemoglobin.

**Oxygen Transport (DO₂)** – The quantity of oxygen carried by the blood per minute. It is determined by multiplying cardiac output and oxygen content of blood.

**Arterial Oxygen Transport (Delivery)** – The volume of oxygen delivered each minute to the tissues. It is the product of the cardiac output and the arterial oxygen content.

Normal Value: 1005 ml O₂/min

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

**Venous Oxygen Transport** – The volume of oxygen returned to the heart from the tissues each minute. It is the product of the cardiac output and the venous oxygen content.

Normal Value: 775 ml O₂/min

\[ \text{Venous Oxygen Transport} = \text{CO} \times \text{CvO}_2 \]

**Partial Pressure of Oxygen (PO₂)** – The pressure exerted by oxygen when oxygen is in a mixture of gases. This pressure is proportional to the percentage of oxygen in the mixture.

Normal Values: \( \text{PaO}_2 = 80-100 \text{ mm Hg} \)

\[ \text{PvO}_2 = 35-45 \text{ mm Hg} \]
BIBLIOGRAPHY


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