

OXYGEN SENSORS

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INTRODUCTION

This article reviews recent advances in the monitoring of patient oxygenation. We summarize the transport of oxygen from the atmosphere to the cell, and describe monitors that function at four stages of the O_2 transport process. These four stages include respired gas, arterial blood, tissue, and venous blood. History and recent developments in pulse oximetry will be discussed. Continuous intraarterial blood-gas sensors will be described and contrasted with other oxygen monitors. Finally, tissue oxygen monitoring and mixed-venous oximetry are discussed.

OXYGEN TRANSPORT IN THE HUMAN BODY

At rest, we consume $\sim 10^{23}$ molecules of oxygen per second. Our complex cardiopulmonary system has developed to rapidly transport this large amount of oxygen from the atmosphere to every cell in the body (Fig. 1).

The equation for arterial blood oxygen content (CaO_2) shows that $\sim 99\%$ of the oxygen in arterial blood is in the form of hemoglobin-bound oxygen:

$$CaO_2 = 1.38(SaO_2/100)(Hb) + 0.003 PaO_2 \quad (1)$$

where CaO_2 is in units of milliliters per deciliter of blood (also called vols%); SaO_2 is the arterial hemoglobin saturation in percent; Hb is the total hemoglobin concentration in grams per deciliter; and PaO_2 is the arterial oxygen tension (partial pressure) in millimeters of mercury. Upon inserting typical arterial values [$SaO_2 = 100\%$, $Hb = 15 \text{ g}\cdot\text{dL}^{-1}$, $PaO_2 = 100 \text{ mmHg}$ (13.33 kPa)], we find that normal CaO_2 is $\sim 21 \text{ mL}\cdot\text{dL}^{-1}$. The amount of oxygen delivered to the tissues in the arterial blood is then given by the cardiac output (CO) times the CaO_2 (neglecting the small dissolved oxygen term):

$$DO_2 = 13.8(CO)(Hb)(SaO_2/100) \quad (2)$$

(The factor 1.38 becomes 13.8 because Hb is normally measured in grams per deciliter, while CO is measured in liters per minute. There are 10 dL in 1 L.)

Finally, the oxygen consumption by the tissues (VO_2) is determined by the difference between arterial oxygen delivery and venous oxygen return:

$$VO_2 = 13.8(Hb)(CO)(SaO_2 - SvO_2)/100 \quad (3)$$

This Fick equation can be solved for any of the four oxygen variables involved.

If we substitute normal resting values into the equation, we predict a resting VO_2 of

$$\begin{aligned} VO_2 &= 13.8(15 \text{ g}\cdot\text{dL}^{-1})(5 \text{ L}\cdot\text{min}^{-1})(99 - 75)/100 \\ &= 248 \text{ mL}\cdot\text{min}^{-1} \end{aligned}$$

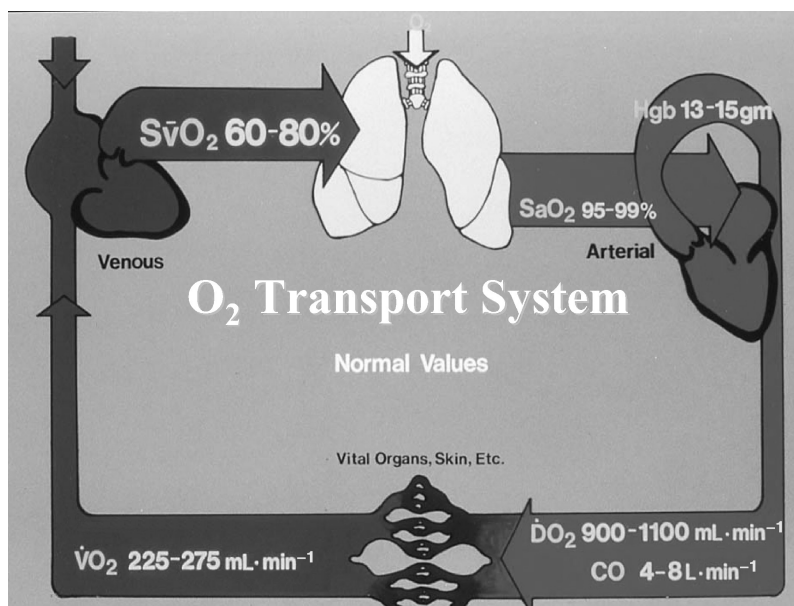


Figure 1. Schematic of the oxygen transport system. Arterial blood leaving the left ventricle (right side of figure) has an oxygen content of 21 mL·dL⁻¹, and the total oxygen delivery ($\dot{D}O_2$) is roughly 1000 mL·min⁻¹. At rest, 0.25 of this oxygen delivery is consumed ($\dot{V}O_2$), leaving a mixed venous saturation of 75%.

During exercise or stress we can rapidly increase cardiac output to at least 20 L·min⁻¹, and decrease venous saturation to ~ 40%, yielding a $\dot{V}O_2$ of

$$\begin{aligned}\dot{V}O_2 &= 13.8 (15 \text{ g} \cdot \text{dL}^{-1})(20 \text{ L} \cdot \text{min}^{-1})(99 - 40)/100 \\ &= 2029 \text{ mL} \cdot \text{min}^{-1}\end{aligned}$$

The human ability to rapidly adjust cardiac output (CO) and mixed-venous oxygen saturation (SvO_2) can be used to compensate for disease processes that affect other transport variables, such as anemia (hemoglobin) or hypoxemia (SaO_2). For example, consider a severely anemic patient with Hb value of 2.5 g·dL⁻¹, who compensates by increasing cardiac output to 15 L·min⁻¹ and decreasing venous saturation to 50%:

$$\begin{aligned}\dot{V}O_2 &= 13.8 (2.5 \text{ g} \cdot \text{dL}^{-1})(15 \text{ L} \cdot \text{min}^{-1})(99 - 50)/100 \\ &= 254 \text{ mL} \cdot \text{min}^{-1}\end{aligned}$$

This extremely anemic patient can thus maintain a normal oxygen consumption by adaptations in CO and SvO_2 that are milder than those we use during normal exercise.

OXYGEN IN THE ARTERIAL BLOOD: PULSE OXIMETRY

Physiologic Considerations

The normal relationship between SaO_2 and PaO_2 is the familiar oxyhemoglobin dissociation curve, shown in Fig. 2. Three convenient points on this curve to remember are $PaO_2 = 27$ mmHg (3.60 kPa), $SaO_2 = 50\%$; $PaO_2 = 40$ mmHg (5.33 kPa), $SaO_2 = 75\%$; and $PaO_2 = 60$ mmHg (8.00 kPa), $SaO_2 = 90\%$. The curve will be shifted toward the right by acidosis, hypercarbia, or increasing 2,3-DPG. At PaO_2 values greater than 80 mmHg (10.66 kPa), SaO_2 is almost 100% and thus becomes virtually independent of PaO_2 . It is important to remember this fact

during SaO_2 monitoring in the operating room, where elevated inspired oxygen fraction (FIO_2) values will yield PaO_2 values much > 80 mmHg (10.66 kPa) most of the time.

A knowledge of the relationship between SaO_2 and PaO_2 allows us to predict the physiologic limitations of saturation monitoring by pulse oximetry. Specifically, the pulse oximeter will give no indication of downward trends in PaO_2 during anesthesia at elevated FIO_2 until PaO_2 values < 80–90 mmHg (10.66–12.00 kPa) are reached. In an animal study, intentional endobronchial intubations at FIO_2 values > 30% were not detected by the pulse oximeter (1). This results from the fact that the PaO_2 after endobronchial intubation did not decrease below ~ 80 mmHg (10.66 kPa) when FIO_2 was elevated.

Technology

Oximetry, a subset of spectrophotometry, determines the concentrations of various hemoglobin species by measuring the absorbances of light at multiple wavelengths. The absorbance spectra of the four most common hemoglobin species are shown in Fig. 3. The number of oximeter light wavelengths used must be equal to or greater than the number of hemoglobin species present in the sample. A laboratory CO-oximeter, which uses four or more wavelengths, can measure the concentrations of reduced hemoglobin, oxyhemoglobin, methemoglobin, and carboxyhemoglobin. If all four hemoglobin species are present in significant concentrations, then an oximeter must have at least four light wavelengths to determine the concentration of any of the four species.

The conventional pulse oximeter is a two-wavelength oximeter that functions *in vivo*. Conventional pulse oximetry first determines the fluctuating or alternating current (ac) component of the light absorbance signal. At each of its two wavelengths the oximeter divides the ac signal by the

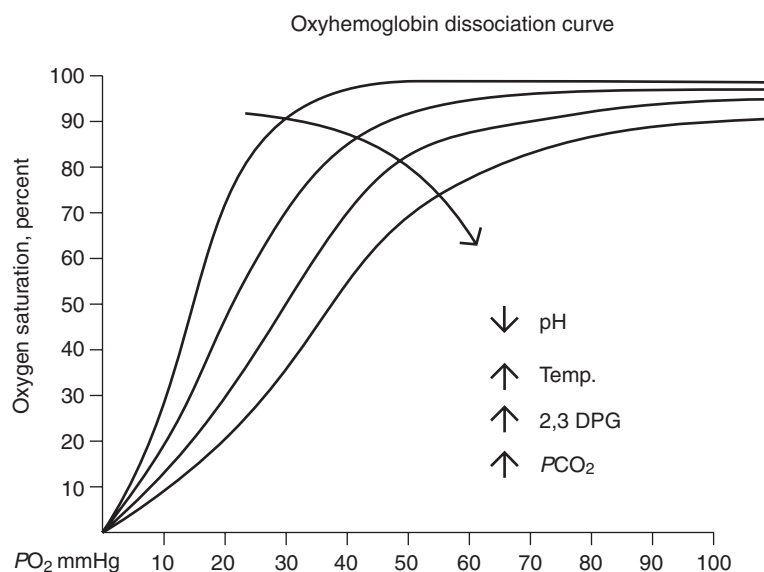


Figure 2. The oxyhemoglobin dissociation curve: a plot of hemoglobin oxygen saturation as a function of oxygen tension (PO_2). The curve is shifted to the right with decreasing pH or increasing temperature, PCO_2 , or 2,3-DPG.

corresponding fixed or direct current (dc) absorbance component, to obtain a pulse-added absorbance. It then calculates the ratio of the two pulse-added absorbances (one for each wavelength), and this ratio R is related to arterial hemoglobin saturation by a built-in calibration algorithm. The resulting pulse oximeter saturation estimate is called SpO_2 . The calibration curve of the pulse oximeter is empirical; that is, it is based on human volunteer experimental data.

Sources of Error

Dyshemoglobins. As previously noted, the pulse oximeter uses two wavelengths and can distinguish only two hemoglobin species: reduced hemoglobin and

oxyhemoglobin. If either carboxyhemoglobin (COHb) or methemoglobin (MetHb) is present, the pulse oximeter effectively has fewer equations than unknowns, and it cannot find any of the hemoglobin concentrations. It is thus unclear *a priori* how the pulse oximeter will behave in the presence of these dyshemoglobins.

Two animal experiments have characterized pulse oximeter behavior during methemoglobinemia and carboxyhemoglobinemia. In one of these, dogs were exposed to carbon monoxide (220 ppm) over a 3–4 h period (2). At a COHb level of 70% (meaning that 70% of the animal's hemoglobin was in the COHb form), the SpO_2 values were $\sim 90\%$, whereas the actual SaO_2 was 30% (Fig. 4). The pulse oximeter thus “sees”

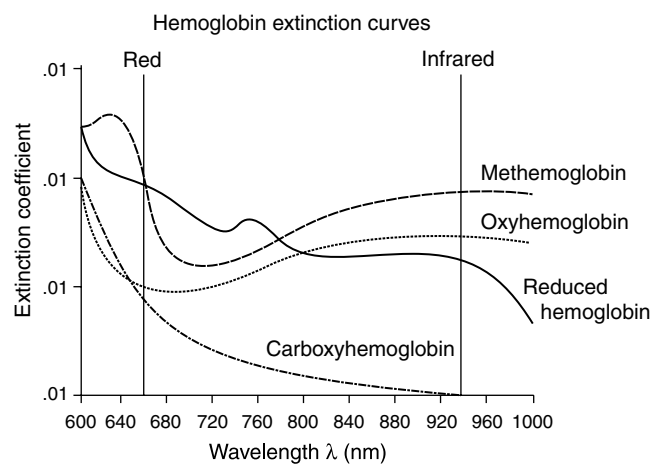


Figure 3. Extinction coefficient (or light absorbance) versus wavelength of light for four different hemoglobins: reduced Hb, O_2Hb , COHb, and MetHb. The two wavelengths used by most pulse oximeters (660 nm, 930 nm) are indicated by vertical lines.

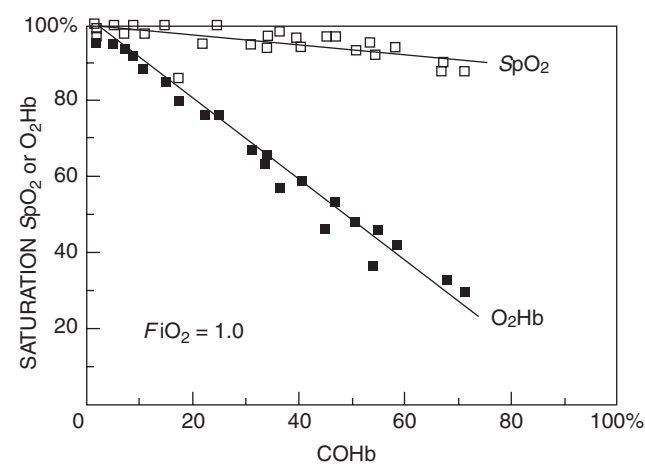


Figure 4. The effect of carbon monoxide on pulse oximetry. Plots of Hb saturation measured by $\blacksquare$ laboratory CO-oximeter, and $\square$ conventional pulse oximeter, as functions of COHb level. As COHb increases, CO-oximeter shows linear decline in saturation, while pulse oximeter remains $> 90\%$ saturation (2).

carboxyhemoglobin as if it were composed mostly of oxyhemoglobin.

In a similar animal experiment, increasing methemoglobin concentrations (up to 60%) produced SpO_2 readings that gradually decreased to $\sim 85\%$ (3). As these animals were further desaturated by lowering the FIO_2 during methemoglobinemia, the pulse oximeter SpO_2 reading failed to track either functional or fractional saturation. On the other hand, the presence of fetal hemoglobin has little effect on pulse oximeter accuracy, which is fortunate in the treatment of premature neonates. There are conflicting anecdotal reports on the influence of sickle hemoglobin, and it is impossible to perform volunteer hypoxia studies on patients with sickle-cell disease.

As of this date, no commercially available pulse oximeter can either measure dyshemoglobins or function accurately in the presence of substantial levels of COHb or MetHb. Masimo Inc. has very recently (March, 2005) announced the development of a new Rainbow Technology pulse oximeter that uses multiple light wavelengths to measure COHb and SaO_2 simultaneously. There are as yet no published data regarding the success of this approach, but it is a potentially important new advancement.

Intravenous Dyes. As abnormal hemoglobin species can adversely affect the accuracy of pulse oximetry, so can intravenous dyes injected during surgery or critical care. Two studies found that intravenous methylene blue causes large, rapid decreases in displayed SpO_2 without changes in actual saturation, and that indocyanine green causes smaller false decreases in SpO_2 (4,5). Intravenous fluorescein or indigo carmine appeared to have little effect.

Reductions in Peripheral Pulsation; Ambient Light. Several studies have examined the effects of low perfusion upon SpO_2 (6,7). In a clinical study of critically ill patients during a wide range of hemodynamic conditions, extremes in systemic vascular resistance were associated with loss of pulse oximeter signal or decreased accuracy. During reduced pulse amplitude, pulse oximeters may become more sensitive to external light sources, such as fluorescent room lights (8). Most modern pulse oximeters effectively measure and correct for ambient light intensity.

Motion Artifact. Patient motion, which causes a large fluctuating absorbance signal, is a very challenging artifact for pulse oximetry. Motion artifact rarely causes great difficulty in the operating room, but in the recovery room and intensive care unit it can make the pulse oximeter virtually useless. Design engineers have tried several approaches to this problem, beginning with increasing the signal averaging time. Most current pulse oximeters allow the user to select one of several time averaging modes. However, improving motion performance by simply increasing averaging time is potentially dangerous—it can cause the instrument to miss signifi-

cant, but short-lived hypoxemic events, which are very common in neonates.

Masimo, Inc. has developed a completely different approach to the analysis of the oximeter light absorbance signals, using adaptive digital filtering. This has led to improved accuracy and reliability during motion artifact, both in laboratory studies (9,10) and in the neonatal intensive care unit (11). The new technology has spurred other manufacturers (e.g., Nellcor, Philips, Datex-Ohmeda) to improve their signal analysis methods, so that today's generation of pulse oximeters has much improved performance during motion.

Venous Pulsations. Conventional pulse oximeter design is predicated on the assumption that the pulsatile component of tissue light absorbance is entirely caused by arterial blood. However, the light absorbance of venous blood can also have a pulsatile component, and this may affect SpO_2 values under some conditions (12). Conventional pulse oximeters may read falsely low values or may fail to give any reading in circumstances leading to venous congestion. This can occur, for example, when using an earlobe sensor on a patient who is undergoing mechanical ventilation, or who is in the Trendelenberg position.

Penumbra Effect. When a pulse oximeter sensor is not properly positioned on the finger or earlobe, the light traveling from the source to the detector may pass through the tissues at only grazing incidence. This penumbra effect reduces the signal/noise ratio, and may result in SpO_2 values in the low 1990s in a normoxemic subject. More importantly, a volunteer study has shown that in hypoxemic subjects, the penumbra effect can cause SpO_2 to either overestimate or underestimate actual SaO_2 values, depending on the instrument used (13). A pulse oximeter with a malpositioned sensor may therefore indicate that a patient is only mildly hypoxemic when in fact he or she is profoundly so.

OXYGEN IN THE ARTERIAL BLOOD: CONTINUOUS INTRAARTERIAL PO_2 MEASUREMENT

There have been a number of efforts to monitor intraarterial oxygen tension directly and continuously, using miniaturized sensors passed through arterial cannulas. The first practical approach to this problem employed the Clark electrode, the same oxygen electrode used in the conventional laboratory blood-gas analyzer. Although miniaturized Clark electrodes have been used in several clinical studies, the technique never achieved popularity because of problems with calibration drift and thrombogenicity (14). More recently, the principle of fluorescence quenching was used to develop fiberoptic "optodes" that can continuously monitor pH, PaCO_2 , and PaO_2 through a 20 gauge radial artery cannula (Fig. 5).

Fluorescence quenching is a result of the ability of oxygen (or other substances to be measured) to absorb energy from the excited states of a fluorescent dye, thus preventing this energy from being radiated as light.

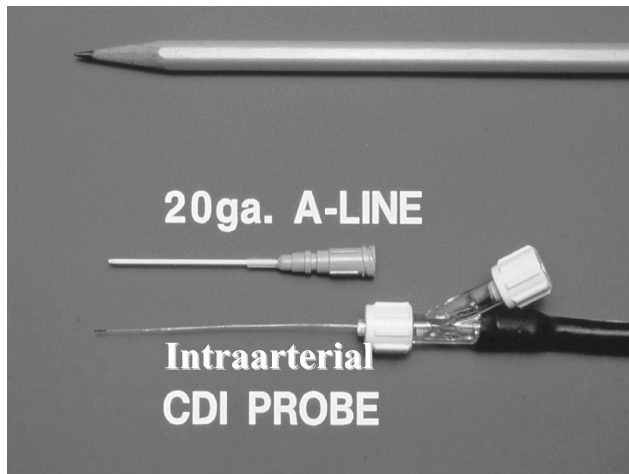


Figure 5. An intraarterial fiber-optic optode sensor. The optode is 0.6 mm in diameter and fits easily through a 20 gauge radial artery cannula, also shown.

Lubbers and Opitz (15) developed the first fluorescence quenching optode that simultaneously measured oxygen and carbon dioxide tensions in gases or liquids. In the 1980s, optodes were successfully miniaturized for intraarterial use, and several studies were reported in both animal and humans (16,17).

Clinical Studies

Several clinical studies suggested the usefulness of intraarterial optodes in the operating room (18). The scatter (random error) of optode oxygen tension values is lowest at low oxygen tensions, a characteristic of these sensors. The accuracy of the optode appeared to be within the clinically acceptable range when 18-gauge arterial cannulas were used. The optode can display complete blood-gas data continuously at the patient's bedside, with a time response measured in seconds. Nevertheless, the high costs of the disposable sensors (~\$300 each) and their inconsistent reliability have caused the intraarterial optodes to disappear from the clinical market. These devices have other potential applications in tissues and organs, which may be realized in the future. One manufacturer today is marketing an optode sensor for assessment of the viability of tissue grafts.

OXYGEN IN TISSUE: TRANSCUTANEOUS OXYGEN

Physiologic Considerations

The transcutaneous oxygen ($P_{tc}O_2$) sensor is a Clark electrode that measures oxygen diffusing through the surface of the skin from dermal capillaries (Fig. 6). The sensor must be heated to at least 43 °C (in adults) to facilitate diffusion through the stratum corneum. Surface heating also produces local hyperemia of the dermal capillaries, which tends to "arterialize" the blood and cause a rightward shift in the oxyhemoglobin dissociation curve. The effects above tend to increase $P_{tc}O_2$, and these are counterbalanced by other effects that decrease it, namely

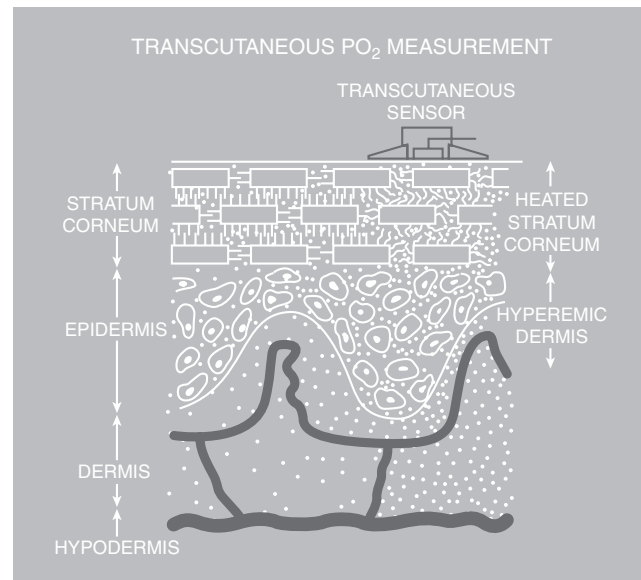


Figure 6. Schematic of transcutaneous PO_2 sensor on skin surface. Heat from sensor "melts" the diffusion barrier of the stratum corneum layer, and "arterializes" the blood in the dermal capillaries beneath.

diffusion gradients and metabolic oxygen consumption by the skin. In neonates, these competing effects nearly cancel and $P_{tc}O_2$ is approximately equal to PaO_2 . In adults, the stratum corneum is thicker and hence the $P_{tc}O_2$ is usually lower than PaO_2 . The transcutaneous index, $P_{tc}O_2/PaO_2$, has average values of 1.0 in neonates, 0.9 in pediatric patients, 0.8 in adults, and 0.6–0.7 in the elderly.

The most serious challenges with the interpretation of $P_{tc}O_2$ values are their dependence upon cardiac output and skin perfusion. Several studies have shown that the transcutaneous index falls when the cardiac index decreases below its normal range (19). Animal shock studies have shown that $P_{tc}O_2$ decreases when either PaO_2 or cardiac index decreases, and that it closely follows trends in oxygen delivery (i.e., the product of CO and CaO_2). In other words, $P_{tc}O_2$ monitors oxygen delivery to the tissues rather than oxygen content of arterial blood.

Technical Problems

There are several practical problems associated with the use of $P_{tc}O_2$ sensors. The transcutaneous electrode must be gas calibrated before each application to the skin, and then the sensor requires a 10–15 min warm-up period. In children, the warm-up period is usually shorter. The sensor membrane and electrolyte must be replaced at least once a week. The heated $P_{tc}O_2$ electrode can cause small skin burns, particularly at temperatures of 44 °C or greater. Lower probe temperatures (43 or 43.5 °C) should be used on premature infants and neonates, and the sensor location should be changed every 2–3 h. In adults with a sensor temperature of 44 °C, we have used the same location for 6–8 h with no incidence of burns.

Summary

Transcutaneous oxygen sensors provide continuous, non-invasive monitoring of oxygen delivery to tissues. By contrast, pulse oximetry provides continuous monitoring of arterial hemoglobin saturation. The dependence of $P_{tc}O_2$ on blood flow as well as PaO_2 sometimes makes it difficult to interpret changing values. If $P_{tc}O_2$ is normal or high, we know that the tissues are well oxygenated. When $P_{tc}O_2$ is low, we must determine whether this is the result of low PaO_2 or a decrease in skin perfusion.

OXYGEN IN VENOUS BLOOD: PULMONARY ARTERY OXIMETRY

Physiology of Mixed-Venous Saturation

Oxygen saturation in venous blood is related to venous oxygen content CvO_2 by an equation similar to equation 1:

$$CvO_2 = 1.38(Hb)(SvO_2)/100 + 0.003(PvO_2) \quad (4)$$

The normal CvO_2 value [with $SvO_2 = 75\%$, $PvO_2 = 40$ mmHg (5.33 kPa)] is $15.6 \text{ mL} \cdot \text{dL}^{-1}$. If we solve equation 3 (the Fick equation) for the venous saturation (SvO_2), we obtain:

$$SvO_2 = SaO_2 - VO_2 / [(13.8)(Hb)(CO)] \quad (5)$$

Equation 8 shows how SvO_2 depends on the four oxygen transport variables: SaO_2 , VO_2 , Hb, and CO.

When VO_2 falls behind oxygen demand, lactic acidosis will result, eventually leading to death if the problem is

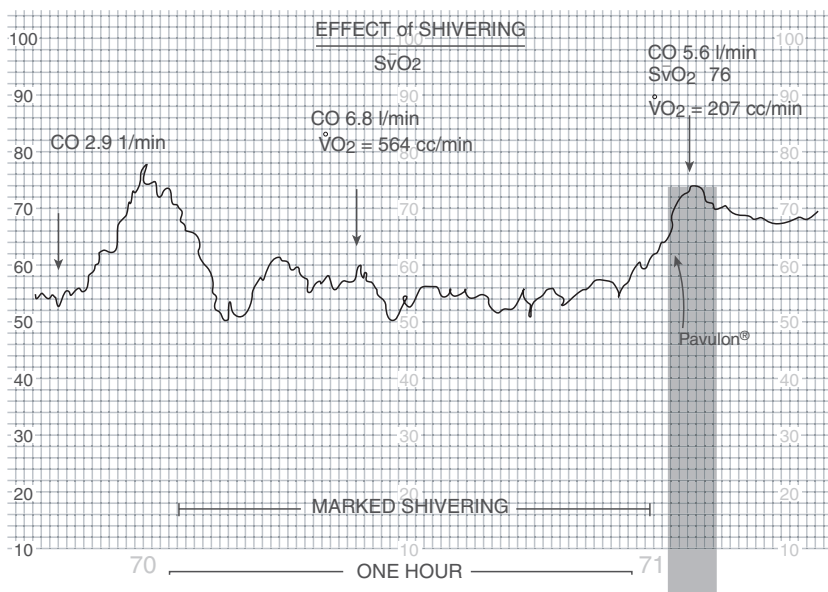
not corrected. When this begins to occur in disease (e.g., anemia), the patient's body will try to maintain normal VO_2 using the same two compensatory mechanisms described above during exercise: increasing CO and/or decreasing SvO_2 . In the case of anemia, we saw that such compensation can maintain normal VO_2 values even at hemoglobin levels $< 3 \text{ g} \cdot \text{dL}^{-1}$. Thus, a decrease in SvO_2 indicates that a patient is using oxygen reserves to compensate for a supply-demand imbalance. Decreasing oxygen supply may result from low CO (shock), low hemoglobin (anemia), abnormal hemoglobin (carboxyhemoglobinemia), or low PaO_2 (hypoxemia). On the other hand, increasing oxygen demand can result from fever, malignant hyperthermia, thyrotoxicosis, or shivering.

The aforementioned are possible clinical causes of a decrease in SvO_2 . There are also conditions that can increase SvO_2 above its normal range of 68–77%. High SvO_2 values can result from decreased tissue uptake of oxygen, peripheral arteriovenous shunting, and inappropriate increases in CO. Clinical conditions that produce elevated SvO_2 values include sepsis, Paget's disease of bone, excessive use of inotropes, cyanide poisoning, and hypothermia. A wedged pulmonary artery catheter will also cause a high SvO_2 reading, but this is a measurement artifact. This can actually be a useful artifact, since it warns the clinician of an inadvertently wedged catheter.

Technical Considerations

Pulmonary artery SvO_2 catheters use the technology of reflectance spectrophotometry; that is, they measure the color of the blood in a manner similar to pulse oximetry. The SvO_2 catheters use fiberoptic bundles to transmit and receive light from the catheter tip. Light-emitting diodes provide monochromatic light sources at two or three wave-

Figure 7. Effect of oxygen consumption (VO_2) upon mixed venous saturation (SvO_2). After weaning from cardiopulmonary bypass, the SvO_2 reaches a normal value of 75%, but then falls to 55%. Measured VO_2 at this time is $564 \text{ mL} \cdot \text{min}^{-1}$, or twice normal resting value. Patient noted to be shivering. Administration of muscle relaxant stops shivering, restores VO_2 to normal ($207 \text{ mL} \cdot \text{min}^{-1}$), and SvO_2 also returns to normal values.



lengths. (Currently, the Edwards system uses two wavelengths, while the Abbott instrument employs three.) A theoretical advantage of a three wavelength system is that its measurements should not depend on the total hemoglobin level (20). Another problem common to all SvO_2 catheters is the so-called wall artifact, whereby reflection from a vessel wall can produce a signal that is interpreted as an SvO_2 of 85–90%. This problem has been reduced by the addition of digital filtering to the processor, which effectively edits out sudden step increases in SvO_2 . However, a persistently high value of SvO_2 may alert the user that the catheter is in the wedged condition, as noted above.

Applications and Limitations

When interpreting continuous SvO_2 versus time tracings in the operating room and intensive care unit, we must always consider equation 5, the Fick equation solved for SvO_2 . When SvO_2 changes, we should ask which term(s) in equation 5 are responsible. In the operating room, the terms most likely to change significantly are cardiac output (CO) and hemoglobin (Hb). During general anesthesia with mechanical ventilation, SaO_2 and VO_2 are usually constant, with the exception that VO_2 will decrease during hypothermia. On the other hand, this is not the case in the intensive care unit. Patients in respiratory failure will have varying degrees of arterial desaturation (low SaO_2). Note that SvO_2 is directly related to SaO_2 ; if SaO_2 decreases by 20% and nothing else changes, then SvO_2 will decrease by 20%. Critical care unit patients may also have frequent changes in VO_2 , which can be increased by agitation, shivering, coughing, fever, pain, seizures, defecation, or eating, to name just a few (Fig. 7).

Continuous SvO_2 is a valuable adjunct in the treatment of ventilator-dependent patients. As positive end-expiratory pressure (PEEP) is slowly increased to improve oxygenation, SaO_2 will usually increase, but eventually the cardiac output will begin to decrease as venous return is compromised. At this point, oxygen delivery to tissue may begin to decrease (and SvO_2 begins to decrease) even though SaO_2 is still increasing. SvO_2 is a reflection of oxygen delivery in this situation, and can thus provide a means to optimize positive end-expiratory pressure without the need of serial blood gases and CO measurements.

In summary, continuous SvO_2 monitoring is a valuable technology for the operating room and the critical care unit. It reflects the overall health and functional state of the oxygen transport system. To realize the most benefit from this monitor, it is essential to thoroughly understand the physiology of SvO_2 and how it relates to the other oxygen transport variables.

CONCLUSIONS

Monitoring of oxygen in the respired gases and arterial blood is the standard of care during all anesthetics today.

None of us would consider administering general anesthesia without both an FiO_2 monitor and a pulse oximeter. New advances in pulse oximetry will make these instruments more reliable in moving or poorly perfused patients, but they will still be subject to the fundamental limitations of saturation monitoring. Further developments will include pulse oximeters that can function in the presence of COHb and MetHb. In the near future, noninvasive monitors of oxygenation in specific organs and tissues (heart, brain) will become available. Finally, mixed venous oxygen saturation indicates how much is "left over" at the end of the oxygen transport process, which gives an indication of the status of the transport system and the degree to which reserves are being used.

BIBLIOGRAPHY

1. Barker SJ, Tremper KK, Hyatt J, Heitzmann H. Comparison of three oxygen monitors in detecting endobronchial intubation. *J Clin Monitoring* 1988;4:240–243.
2. Barker SJ, Tremper KK. The effect of carbon monoxide inhalation on pulse oximetry and transcutaneous PO_2 . *Anesthesiology* 1987;66:677–679.
3. Barker SJ, Tremper KK, Hyatt J. Effects of methemoglobinemia on pulse oximetry and mixed venous oximetry. *Anesthesiology* 1989;70:112–117.
4. Sidi A, et al. Effect of fluorescein, indocyanine green, and methylene blue on the measurement of oxygen saturation by pulse oximetry (abstract). *Anesthesiology* 1986;65(3A):A132.
5. Scheller MS, Unger RJ, Kelner MJ. Effects of intravenously administered dyes on pulse oximetry readings. *Anesthesiology* 1986;65:550–552.
6. Lawson D, et al. Blood flow limits and pulse oximeter signal detection. *Anesthesiology* 1987;67:599–603.
7. Narang VPS. Utility of the pulse oximeter during cardiopulmonary resuscitation. *Anesthesiology* 1986;65:239–240.
8. Eisele JH, Downs D. Ambient light affects pulse oximeters. *Anesthesiology* 1987;67:864–865.
9. Barker SJ, Shah NK. The effects of motion on the performance of pulse oximeters in volunteers. *Anesthesiology* 1997;86:101–108.
10. Barker SJ. Motion Resistant pulse oximetry. A comparison of new and old models. *Anesth Analg* 2002;95:967–972.
11. Bohnhorst B, Peter C, Poets CF. Pulse oximeters' reliability in detecting hypoxia and bradycardia: Comparison between a conventional and two new generation oximeters. *Crit Care Med* 2000;28:1565–1568.
12. Kim JM, et al. Pulse oximetry and circulatory kinetics associated with pulse volume amplitude measured by photoelectric plethysmography. *Anesth Analg* 1986;65:133–139.
13. Barker SJ, et al. The effect of sensor malpositioning on pulse oximeter accuracy during hypoxemia. *Anesthesiology* 1993;79:248–254.
14. Rithalia SVS, Bennett PJ, Tinker J. The performance characteristics of an intraarterial oxygen electrode. *Intensive Care Med* 1981;7:305–307.
15. Lubbers DW, Opitz N. Die pCO_2/pO_2 -optode: eine neue pCO_2 bzw. pO_2 -Messsonde zur Messung des pCO_2 oder pO_2

- von Gasen and Flussigkeiten. Z Naturforsch 1975;30:532–533.
16. Barker SJ, et al. Continuous fiberoptic arterial oxygen tension measurements in dogs. J Clin Monitoring 1987;39:48–52.
 17. Barker SJ, et al. A clinical study of fiberoptic arterial oxygen tension. Crit Care Med 1987;15:403.
 18. Barker SJ, Hyatt J. Continuous measurement of intraarterial pHa, PaCO₂, and PaO₂ in the operating room. Anesth Analg 1991;73:43–48.
 19. Tremper KK, Waxman K, Shoemaker WC. Effects of hypoxia and shock on transcutaneous PO₂ values in dogs. Crit Care Med 1979;7:52.
 20. Gettinger A, DeTraglia MC, Glass DD. *In vivo* comparison of two mixed venous saturation catheters. Anesthesiology 1987;66:373–375.
- See also BLOOD GAS MEASUREMENTS; SAFETY PROGRAM, HOSPITAL.

OXYGEN TOXICITY. See HYPERBARIC OXYGENATION.