Monitoring of Arterial Oxygenation by Pulse Oximetry
Principles of Operation

Pulse oximeters measure pulse rate and estimate the oxygen saturation of hemoglobin (SPO_2) on a noninvasive, continuous basis.[9] The oxygen saturation (SaO_2) of hemoglobin (as a percentage) is related to the oxygen tension (as a partial pressure, mmHg) by the oxyhemoglobin dissociation curve. On the steep part of the curve, a predictable correlation exists between SaO_2 and partial pressure of oxygen (PaO_2). In this range, the SaO_2 is a good reflection of the extent of hypoxemia and the changing status of arterial oxygenation. For PaO_2 greater than 75 mmHg, the SaO_2 reaches a plateau and no longer reflects changes in PaO_2. Coexisting medical conditions, such as hypercapnia, acidosis, and hyperthermia, cause the oxyhemoglobin dissociation curve to shift to the right and decrease the affinity of hemoglobin for oxygen. This change favors the unloading of oxygen from hemoglobin to peripheral tissues, as shown in Figure 26-1.

26.1 Central Venous Access

Pulse oximetry is based on the following premises:

1. The color of blood is a function of oxygen saturation.
2. The change in color results from the optical properties of hemoglobin and its interaction with oxygen.
3. The ratio of oxyhemoglobin (HbO_2) and hemoglobin (Hb) can be determined by absorption spectrophotometry.

Oxygen saturation is determined by spectrophotometry, which is based on the Beer-Lambert law. At a constant light intensity and hemoglobin concentration, the intensity of light transmitted through a tissue is a logarithmic function of the oxygen saturation of Hb. Two wavelengths of light are required to distinguish HbO_2 from Hb. Light-emitting diodes in the pulse sensor emit red (660 nm) and near infrared (940 nm) light. The percentage of HbO_2 is determined by measuring the ratio of infrared and red light sensed by a photodetector. Pulse oximeters perform a plethysmographic analysis to differentiate the pulsatile “arterial” signal from the nonpulsatile signal resulting from “venous” absorption and other tissues, such as skin, muscle, and bone. The absence of a pulsatile waveform during extreme hypothermia or hypoperfusion can limit the ability of a pulse oximeter to calculate the SPO_2.

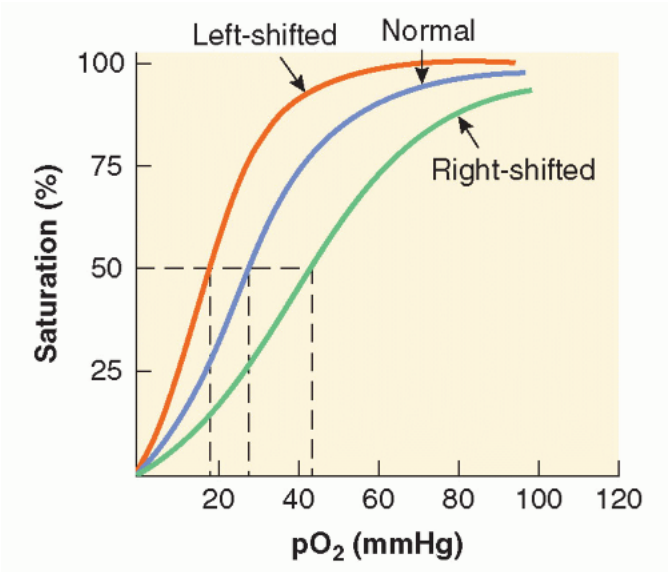

Figure 26-1 The oxyhemoglobin dissociation curve. The relationship between arterial saturation of hemoglobin and oxygen tension is represented by the sigmoid-shaped oxyhemoglobin dissociation curve. When the curve is left-shifted, the hemoglobin molecule binds oxygen more tightly. (Adapted from: Brown M, Vender JS. Noninvasive oxygen monitoring. *Crit Care Clin.* 1988;4:493-509.)

The SPO_2 measured by pulse oximetry is not the same as the arterial saturation (SaO_2) measured by a laboratory co-oximeter. Pulse oximetry measures the "functional" saturation, which is defined by the following equation:

$$SPO_2 = \frac{HbO_2}{HbO_2 + Hb} \times 100\%$$

Laboratory co-oximeters use multiple wavelengths to distinguish other types of Hb, such as carboxyhemoglobin (COHb) and methemoglobin (MetHb) by their characteristic absorption. Co-oximeters measure the "fractional" saturation, which is defined by the following equation:

$$SaO_2 = \frac{HbO_2}{HbO_2 + Hb + COHb + MetHb} \times 100\%$$

In clinical circumstances in which other Hb moieties are present, the SPO_2 measurement may not correlate with the actual SaO_2 reported by the blood gas laboratory. For example, MetHb absorbs red and infrared wavelengths of light in a 1:1 ratio corresponding to a SPO_2 of approximately 85%. Therefore, increases in MetHb produce an underestimation when SPO_2 > 70% and an overestimation when SPO_2 <70%. Similarly, COHb also produces artificially high and misleading results; one study showed that at 70% COHb, the SPO_2 still measured 90%. In most patients, MetHb and COHb are present in low concentrations so that the functional saturation approximates the fractional value.[10]

Proper Use and Interpretation

The assessment of arterial oxygenation is an integral part of anesthesia practice. Early detection and prompt intervention may limit serious sequelae of hypoxemia. The clinical signs associated with hypoxemia (e.g., tachycardia, altered mental status, cyanosis) are often masked or difficult to appreciate during anesthesia.
The appropriate use of pulse oximetry necessitates an appreciation of both physiologic and technical limitations. Despite the numerous clinical benefits of pulse oximetry, other factors affect its accuracy and reliability. Factors that may be present during anesthesia care and that affect the accuracy and reliability of pulse oximetry include dyshemoglobins, dyes (methylene blue, indocyanine green, and indigo carmine), nail polish, ambient light, light-emitting diode variability, motion artifact, and background noise. Electrocautery can interfere with pulse oximetry if the radiofrequency emissions are sensed by the photodetector. Surgical stereotactic positioning systems that make use of infrared position sensors may interfere with the infrared signals used by the pulse oximeter. Reports of burns or pressure necrosis exist but are infrequent. Inspecting the digits during monitoring can reduce these complications.
Recent developments in pulse oximetry technology reportedly may permit more accurate measurements of SPO_2 during patient movement, low-perfusion conditions, and in the presence of dyshemoglobins. Some of these instruments use complex signal processing of the two wavelengths of light to improve the signal-to-noise ratio and reject artifact. Studies in volunteers suggest that the performance of pulse oximeters incorporating this technology is superior to conventional oximetry during motion of the hand, hypoperfusion, and hypothermia.[11,12] Other pulse oximetry devices incorporate eight wavelengths of light to more accurately measure COHb and MetHb.[10]

Indications

Pulse oximetry has been used in all patient age groups to detect and prevent hypoxemia. The clinical benefits of pulse oximetry are enhanced by its simplicity. Modern pulse oximeters are noninvasive, continuous, and autocalibrating. They have quick response times and their battery backup provides monitoring during transport. The clinical accuracy is typically reported to be within ±2% to 3% at 70% to 100% saturation and ± 3% at 50% to 70%

saturation. Published data from numerous investigations support accuracy and precision reported by instrument manufacturers. Quantitative assessment of arterial oxygen saturation is mandated by the ASA monitoring standards,[1] and the convenience and safety of pulse oximetry has supplanted earlier techniques such as heated transcutaneous pO_2 electrodes.[13]

Pulse oximetry has wide applicability in many hospital and nonhospital settings. However, there are no definitive data demonstrating a reduction in morbidity or mortality associated with the advent of pulse oximetry. An older large randomized trial did not detect a significant difference in postoperative complications when routine pulse oximetry was used.[14] However, a reduction of anesthesia mortality, as well as fewer malpractice claims from respiratory events, coincident with the introduction of pulse oximeters suggests that the routine use of these devices may have been a contributing factor.

Contraindications

There are no clinical contraindications to monitoring arterial oxygen saturation with pulse oximetry.

Common Problems and Limitations

Arterial oxygen monitors do not ensure adequacy of oxygen delivery to, or utilization by, peripheral tissues and should not be considered a replacement for arterial blood gas measurements or mixed central venous oxygen saturation when more definitive information regarding oxygen supply and utilization is required.

Pulse oximetry is a poor indicator of adequate ventilation; patients who have been breathing supplemental oxygen may be apneic for several minutes before desaturation is detected by the pulse oximeter. Once the PaO_2 has fallen sufficiently to cause a detectable decrease in SPO_2, further desaturation may occur precipitously once the steep part of the oxyhemoglobin dissociation curve is reached.

Placing and obtaining reliable data from blood pressure cuffs and electrocardiogram leads may be challenging in an awake and vigorous child prior to inhalation induction. Therefore, at a minimum, efforts should be made to place a pulse oximetry device on the child or infant prior to induction of anesthesia. Pulse oximetry has also been shown to be a more sensitive monitor than capnography for unrecognized main-stem/endobronchial intubation in pediatric anesthesia.[15] Respiratory events leading to inadequate ventilation and oxygenation represent the majority of perianesthetic morbidity in the pediatric anesthesia population. In conjunction with vigilant clinical assessment of the child's airway and oxygenation, the pulse oximeter usually provides the most important indicator of patient well-being during pediatric anesthesia.[16] Stress caused by hypoxemia and respiratory acidosis in infants and young children triggers a vagal response and subsequent systemic hypoperfusion. Infants cannot adequately increase their cardiac stroke volume (SV) in compensation and so, according to the cardiac output (CO) equation:

Cardiac Output = Heart Rate × Stroke Volume

Therefore, in infants, CO must be maintained with an increase over their baseline HR. A decline in the pitch or rapidity of pulse oximetry tones may be the first signs of impending cardiovascular collapse.