PULSE OXIMETRY

Dr. Vijaylakshmi Kamat

Introduction

There is no doubt that pulse oximetry represents the greatest advance in patient monitoring in many years. It has the unique advantage of continuously monitoring the saturation of hemoglobin with oxygen, easily and noninvasively, providing a measure of cardio-respiratory function. By virtue of its ability to quickly detect hypoxaemia, it has become the standard of care during anaesthesia as well as in the recovery room and intensive care unit. Pulse oximetry should be used to monitor any patient who is heavily sedated or is likely to become hypoxic.

The fundamental physical property that allows the pulse oximeter to measure the oxygen saturation of hemoglobin is that blood changes colour as hemoglobin absorbs varying amounts of light depending on its saturation with oxygen. Oxyhemoglobin does not absorb much red light, but as the hemoglobin oxygen saturation drops, more and more red light is absorbed and the blood becomes darker. At the near infrared range of light however, oxyhemoglobin absorbs more light than reduced hemoglobin.

Pulse oximetry is thus based upon two physical principles:

a) The light absorbance of oxygenated hemoglobin is different from that of reduced hemoglobin, at the oximeter's two wavelengths, which include red and near infrared light; and

b) The absorbance of both wavelengths has a pulsatile component, which is due to the fluctuations in the volume of arterial blood between the source and the detector.

Given these two facts, clever engineering techniques have produced an invaluable monitor.

History

The concept of pulse oximetry is not new. In 1935 Carl Matthes built the first device to continuously measure blood oxygen saturation in vivo by transilluminating tissue.

1. Professor & Head
Department of Anaesthesiology
Sri Ramachandra Medical College & Research Institute
Porur, Chennai - 600 116

He used two wavelengths of light, one of which was sensitive to changes in oxygen saturation and the other, which was in the infra-red range, was used to compensate for changes in tissue thickness, hemoglobin content and light intensity. Although useful in following trends in saturation, the device had limitations as it was difficult to calibrate and absolute values could not be obtained.

J.R.Squire in 1940 devised a technique of calibration by compressing tissue to eliminate the blood. This was later incorporated in the first generation of pulse oximeters used in the operating theatres.

In the early 1940s, Glen Millikan coined the term "oximeter" to describe a lightweight earpiece to detect the oxygen saturation of hemoglobin, for use in aviation research to investigate high altitude hypoxic problems. Soon, similar devices were used during anaesthesia to detect episodes of arterial desaturation in patients. An editorial in Anaesthesiology in 1951[1] concluded prophetically "on many occasions this instrument has detected anoxaemia when observations of pulse, blood pressure, and colour of the patient, and peripheral vascular tone have shown no abnormalities". This confirmed Comroe's classic work,[2] which emphasised the unreliability of cyanosis in detecting hypoxaemia. Thus the clinical utility of pulse oximeters was evident to researchers in the field more than half a century ago.

Millikan's ear oximeter was not calibrated, and one had to guess the normal saturation for each subject as well as the thickness of the ear. In order to overcome the problem of calibration, using Squire's concept, Earl Wood added a pneumatic cuff to measure the light increase when the ear was blanched.

In 1964, a surgeon, Robert Shaw, built a self-calibrating ear oximeter, which was marketed by Hewlett Packard in 1970 for use in physiology and cardiac catheterization laboratories.

The year 1972 marked the greatest step forward in monitoring oxygenation, ironically as an incidental finding. Until then, in order to isolate arterial blood for transillumination, oximeters relied on compression and heating the earlobe to remove signals from venous and capillary blood which often caused burns.

Takuo Aayogi at the Nihon Kohden Corp. working on a dye dilution cardiac output monitor using a ear

densitometer, found artifacts due to pulsatile flow. He noted that the washout curves he was measuring, were modified by pulsatile variations. While attempting to eliminate these variations, he discovered that the absorbency ratios of these pulsations at different wavelengths varied with the oxygen saturation. Thus, he could minimize the pulsatile component by balancing the red light signal with an infrared light signal where the dye had no absorption. As this compensation was dependant on oxygen saturation, he incorporated the technique of reducing noise in his signal to measure oxygen saturation. The subsequent development of light emitting diodes (LEDs), photo detectors and microprocessors further refined the technique, and pulse oximeters were widely introduced into clinical practice.

Modern pulse oximetry was born with the realization that pulsatile changes in light transmission through living tissues are due to alteration of the arterial blood volume in the tissue. Measurement of the pulsatile component would eliminate the variable absorption of light by bone, tissue, skin, pigment, etc from analysis. The most important premise of pulse oximetry therefore, is that the only pulsatile absorbance between the light source and the photo detector is that of arterial blood.

Two wavelengths of light are used; 660 nanometers (red) and 940 nanometres (near infrared). At 660nm, reduced hemoglobin absorbs about ten times as much light as oxyhemoglobin. At the infrared wavelength, (940nm), the absorption coefficient of oxyhemoglobin is greater than that of reduced hemoglobin. The pulse oximeter directly senses the absorption of red and infra red light, and the ratio of pulsatile to nonpulsatile light at the red and infrared wavelengths are translated through complex signal processing to a function of the arterial oxygen saturation.

A microprocessor integrates the data, and through an elaborate calibration algorithm based on human volunteer data, the oxygen saturation can be estimated.

Physics

Pulse oximetry depends on spectral analysis for measurement of oxygen saturation; i.e. the detection and quantification of components in solution by their unique light absorption characteristics.

The pulse oximeter combines the two technologies of spectrophotometry (which measures hemoglobin oxygen saturation) and optical plethysmography (which measures pulsatile changes in arterial blood volume at the sensor site).

Detection of oxygen saturation of hemoglobin by spectrophotometry is based on Beer-Lambert law, which relates the concentration of a solute to the intensity of light transmitted through a solution. In order to estimate the concentration of a light absorbing substance in a clear solution from the intensity of light transmitted through the solution, one needs to know the intensity and wavelength of incident light, the transmission path length, and absorbance of the substance at a specific wavelength (the extinction coefficient). This is based on the formula:

$$I_{trans} = I_{inc} - A$$
$$A = DCE$$

Itrans = intensity of transmitted light

Iin = intensity of incident light

A = absorption

D = distance light is transmitted through the liquid (path length)

C = concentration of solute (hemoglobin)

E = extinction coefficient of the solute (a constant for a given solute at a specified wavelength)

Thus, in a container of known dimensions (called a cuvette), the concentration of a known solute in clear solution can be calculated from the measurement of the intensity of transmitted and incident light of known wavelengths. If there is one solute, the absorption A is a product of the path length, the concentration and the extinction coefficient. Each solute has a specific extinction coefficient for absorption of light at a specific wavelength.

If more than one solute is present, A is the sum of similar expressions for each solute. The extinction coefficients of each solute vary with the wavelengths of light, as seen with the different forms of hemoglobin.

The absorbance of different wavelengths is dependant on the different solute concentrations (reduced and oxygenated hemoglobin), and is detected by transmitting light of specific wavelengths across the solution and measuring the intensity on the other side.

Using the principle of Beer's law, the concentration of a given solute in a solvent is determined by the amount of light that is absorbed by the solute at a specific wavelength. To measure oxygen saturation, the relative concentrations of reduced and oxygenated hemoglobins must be known, and the two different wavelengths of light used must be such that each will preferentially absorb one of them. This is true of hemoglobin, which has a peak absorption of reduced hemoglobin at 660nm (red

light), and oxygenated hemoglobin at 940nm (near infrared light).

The saturation of a suspension of pure hemoglobin in a cuvette can be determined by measuring the ratio of light absorbed at the red wavelength (660nm) to that absorbed at the infrared wavelength (940nm); and this ratio A660/A940 will correlate with oxygen saturation.

For Beer's law to be accurate, the solvent and the cuvette must be transparent, and there must be no extraneous solute, which can absorb light. Co-oximeters in the laboratory measure the intensity of light transmitted through a cuvette filled with hemoglobin from lysed RBCs to determine concentrations of each of the different forms of hemoglobin. This is difficult to fulfill in vivo and correction factors need to be built into pulse oximeters to overcome absorbance by tissues other than hemoglobin.

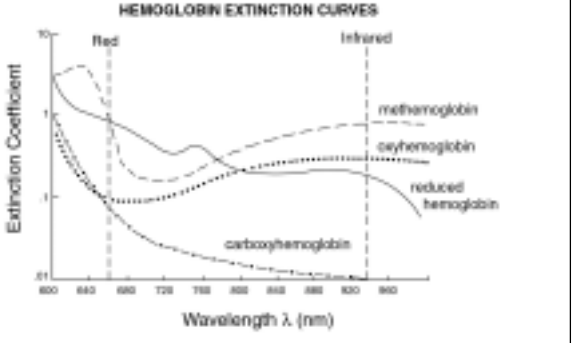

Fig : 1

(Reprinted with permission[3])

The diagram (Fig.1) shows the extinction coefficients of the 4 types of hemoglobin at the red and infrared wavelengths. Methemoglobin absorbs light at both wavelengths to an equal extent; the absorption of red light by carboxyhemoglobin is similar to oxyhemoglobin.

Hemoglobin saturation definitions

Adult blood usually contains four species of hemoglobin: oxyhemoglobin (O_2Hb), reduced hemoglobin (Hb), methemoglobin (MetHb) and carboxyhemoglobin (CO Hb). The last two species are present in the blood in small concentrations, except in pathologic conditions.

Oxygen saturation is defined as the oxygen content expressed as a percentage of the oxygen capacity. By the above definition of oxygen saturation, the two forms of hemoglobin that do not bind oxygen COHb and MetHb) are not included. This is the origin of what is termed "functional hemoglobin saturation".

$$\text{Functional } SaO_2 = \frac{O_2Hb}{O_2Hb + Hb} \times 100\%$$

The extinction coefficients for CoHb and MetHb are not zero in the red and infrared range and their presence will, therefore, contribute to the absorption. Even though the definition of functional hemoglobin saturation involves only two hemoglobin species (O_2Hb and Hb), when MetHb and CoHb are present in appreciable concentrations, the readings would be erroneous.

When oximetry is used to measure the percentage of the oxyhemoglobin fraction accurately, Beer's law must be applied to a solution containing four unknown species: O_2Hb, Hb, COHb, and Met Hb.

Multiwavelength oximeters (co-oximeters in the laboratory) that can measure all four species of hemoglobin, define the ratio of oxyhemoglobin to total hemoglobin as "fractional saturation".

$$\text{Fractional } SaO_2 = \frac{O_2Hb}{O_2Hb+Hb+COHb + MetHb} \times 100\%$$

The fractional hemoglobin saturation is also called the "oxyhemoglobin fraction" or oxyhemoglobin%.

Principle of pulse oximetry

The use of the oximeter's two wavelengths of light is predicated on the following: red and near infrared light readily penetrate tissue, while blue, green, yellow and longer wavelength infrared light are absorbed by tissue and water. Light emitting diodes (LEDs), which reliably emit a specific wavelength of light are widely available at the red and near infrared wavelengths, to use as light sources.

Each pulse oximeter probe contains LEDs, which emit two wavelengths of light, (red and near infrared) through a cutaneous vascular bed. The probe is commonly placed on the digits or earlobe. A photodetector on the other side measures the intensity of transmitted light at each wavelength from which oxygen saturation is derived, based on human volunteer data stored in the memory of the oximeter.

Red and infrared light transmitted through a tissue bed are measured using the finger or ear as a cuvette containing hemoglobin.

Modern pulse oximeters consist of a peripheral probe together with a microprocessor unit displaying a waveform, the oxygen saturation and the pulse rate. The probe is placed on the digit, earlobe or nose. Within the probe are two LEDs, one in the visible red spectrum (660nm) and the other in the infrared spectrum (940nm). The beams of light pass through the tissues to the photo detector. During passage through the tissues some light is absorbed by blood and soft tissues depending on the

concentration of hemoglobin. The amount of light absorption at each frequency depends upon the degree of oxygenation of hemoglobin within the tissues.

There are several technical problems in accurately estimating oxygen saturation by this method, as scatter, reflection and absorbance of light by other tissue and blood components could confound the values. The system needs to isolate absorbance of arterial blood from venous blood, connective tissue and other extraneous matter. This can be accomplished easily as arterial blood is pulsatile unlike other tissue. Thus the pulse added signal can be distinguished from nonpulsatile signal by filtering the extraneous "noise".

The early oximeters subtracted the tissue absorbance by compressing the tissue during calibration to eliminate all the blood, and using the absorbance of bloodless tissue as the baseline. They also heated the tissue to obtain a signal related to arterial blood with minimum influence of venous and capillary blood.

The problem of isolating arterial signals is handled differently today.

The microprocessor can select out the absorbance of the pulsatile fraction of the blood i.e. that due to arterial blood (AC), from the constant absorbance by nonpulsatile venous or capillary blood and other tissue pigments (DC), thus eliminating the effect of tissue absorbance to measure the oxygen saturation of arterial blood.

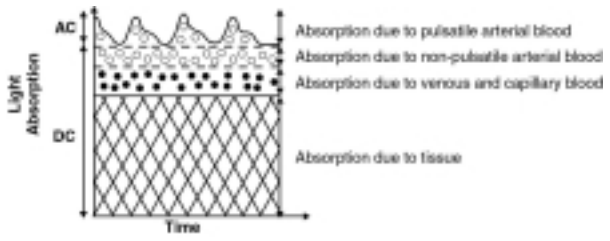

Fig : 2

(Reprinted with permission[3])

Fig.2 shows that the pulsatile (AC) and nonpulsatile (DC) components of light absorption can be separated

The pulsatile expansion of the arteriolar bed produces an increase in path length thereby increasing the absorbance. All pulse oximeters assume that the only pulsatile absorbance between the light source and the photodetector is that of arterial blood. The microprocessor first determines the AC component of absorbance at each wavelength and divides this by the corresponding DC component. From the proportions of light absorbed by each component at the two frequencies it then calculates the ratio (R) of the "pulse-added" absorbance.

$$R = \frac{AC660 / DC660}{AC940 / DC940}$$

Within the oximeter memory, is a series of oxygen saturation values obtained from experiments in which human volunteers were given increasingly hypoxic mixtures to breathe, until saturation values of 80% were obtained. R is compared with the stored values and the oxygen saturation is displayed. Since the microprocessor has no memory of values less than 80% (as it is unethical to make volunteers more hypoxic) accuracy cannot be ascertained below a value of 75-80%. Any saturation below this value would be an extrapolated one and hence inaccurate.

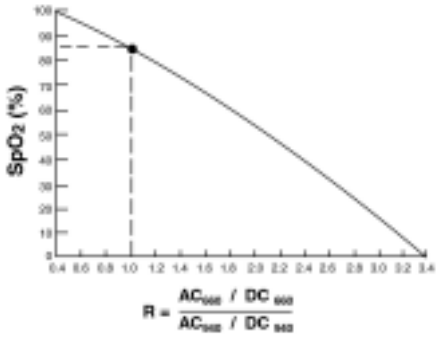

Fig : 3

(Reprinted with permission[3])

The diagram (Fig.3) shows the ratio of red and infrared light absorbed at different saturations. The ratio = 1 at a saturation of 85%. In methemoglobinemia (when equal amounts of red and infrared wavelengths are absorbed), the pulse oximeter's saturation stays at 85%.

Pitfalls and limitations

Despite the reliance placed on the information received from this essential monitor, the underlying principles and limitations of pulse oximetry are poorly understood.

Dyshemoglobinemias

The accuracy of pulse oximetry is excellent when the oxygen saturation is between the range of 70% to 100%, provided the only hemoglobin species present in the blood are reduced hemoglobin and oxygenated hemoglobin. If carboxyhemoglobin or methemoglobin are present in appreciable amounts, the accuracy is suspected.

COHb and MetHb also absorb light at the pulse oximeter's two wavelengths, and this leads to error in estimating the percentages of reduced and oxyhemoglobins.

Met Hb occurs when the normal ferrous (Fe^{+2})state of the iron moiety in hemoglobin is oxidized to the ferric state (Fe^{+3}).

MetHb absorbs equal amounts of red and near infrared light. The ratio of pulsatile and nonpulsatile absorbances in the two wavelengths is equal to 1 at a hemoglobin oxygen saturation of 85%.

A high concentration of MetHb causes the saturation to approximate 85%. When the patient is hypoxic (saturation 40-50%), the MetHb artifactually increases the pulse oximeter reading to 85%. Conversely, if the oxygen saturation is 100%, the MetHb spuriously decreases the pulse oximeter reading to 85%.[4]

This explains why the pulse oximeter reading stays at 85% in the presence of significant methemoglobinemia, regardless of the "true" oxygen saturation, while a multiwavelength co-oximeter will show decreasing oxygen saturations at increasing MetHb levels (Fig 4).

Whenever significant methemoglobinemia is suspected, it is imperative to check an arterial blood sample in a co-oximeter to measure the amount of oxyhemoglobin and methemoglobin.

The treatment of methemoglobinemia with methylene blue further confuses the picture, as the dye lowers the pulse oximeter reading as well. Once the MetHb [illegible] the methylene blue is diluted or [illegible]e saturation measured by pulse [illegible]mal.

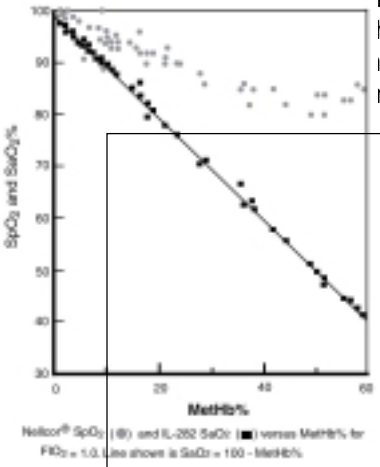

Fig : 4

(Reprinted with permission[5])

Fig 4. While the co-oximeter shows the true oxygen saturation, the pulse oximeter's reading stays at 85%.

Carboxyhemoglobin levels in nonsmokers are less than 2%, while they may be as high as 10-20% in heavy smokers. COHb absorbs very little light at 940nm, while at 660nm its extinction coefficient is very similar to oxyhemoglobin. Thus the presence of significant COHb will resemble the curve of oxyhemoglobin in the red range, with no effect on the infrared, and "look like" oxyhemoglobin, causing the pulse oximeter to over read. For every 1% of circulating carboxyhemoglobin, the pulse oximeter over reads by 1%. Fifty percent of cigarette smokers have a carboxyhemoglobin concentration of 6%.[6]

When the presence of either of these dyshemoglobins is suspected, pulse oximetry should be supplemented by in vitro multiwavelength co-oximetry.

Other common sources of error include extraneous energy sources, especially bright visible or infrared light which may flood or overload the semiconductor detector, and display a value of 85%. The problem of bright fluorescent ambient light causing spurious readings can be reduced by covering the sensor with felt pads.

Pulsatile veins cause under reading as the oximeter cannot differentiate between pulsatile arteries and veins. Tricuspid regurgitation and neonates with hyperdynamic circulation may have inaccurate readings.

Poor function with poor perfusion

In additions to artifacts and misreading, there is a small but definite incidence of failure with pulse oximetry.

The most important limitation of pulse oximeters is that they are inaccurate in patients who need them the most. As it is mandatory to have a good pulse waveform (this is essential for the oximeter to calculate the ratio of pulsatile to non-pulsatile absorbance and derive the oxygen saturation), the pulse oximeter fails to give accurate readings whenever the peripheral pulsations are poor. Adequate arterial pulsations are required to distinguish the light absorbed by arterial blood from that absorbed by venous blood and tissue and readings may be unreliable or unavailable if there is loss or diminution of the peripheral pulse (proximal blood pressure cuff inflation, leaning on an extremity, improper positioning, hypotension, hypothermia, cardiopulmonary bypass, low cardiac output, hypovolaemia, peripheral vascular disease or infusion of vasoactive drugs).[7] A Valsalva maneuver, such as is seen in laboring patients, will cause a decrease in pulse amplitude, which adversely affects the oximeter's ability to provide useful data. Cold extremities may impair the functioning of the pulse oximeter especially if the patient has Raynaud's phenomenon. Under these conditions, some pulse oximeters blank the display or

give a message such as Low Quality Signal or Inadequate Signal.[8] Others freeze the display at the previous reading when they are unable to detect a consistent pulse wave. The presence of a functioning pulse oximeter should not be construed as evidence of adequate tissue oxygenation or oxygen delivery to vital organs.

Methods to improve the signal include application of vasodilating cream, digital nerve blocks,[9] administration of intra-arterial vasodilators, or placing a glove filled with warm water in the patient's hand. Warming cool extremities may increase the pulse amplitude, provided the cardiac output is not depressed.

Other factors reported to contribute to higher failure rates include the very young and very elderly patients; ASA III and IV patients; during orthopedic, vascular and cardiac surgery; use of electrocautery; hypertension; prolonged duration of intraoperative procedure; chronic renal failure; low hematocrit; and pigmented skin.[10] The actual failure rate varies with the individual monitor and is increased with ear and nose sensors. Monitors that can analyze the signal and reject artifacts have fewer episodes of failure. Pulse oximeters with signal extraction technology may perform better during low perfusion states.

Pulse oximeters are most unreliable in the newborn, as minor changes in skin temperature, as well as minor adjustments in contact can cause motion artifacts and a poor signal. Most experienced neonatologists and paediatric anaesthesiologists use more than one pulse oximeter to improve accuracy.

Difficulty in detecting high oxygen partial pressures

At high saturations, small changes in saturation are associated with relatively large changes in PaO_2. Thus the pulse oximeter has a limited ability to distinguish high but safe levels of arterial oxygen from excess oxygenation, which may be harmful, as in premature newborns, or patients with severe COPD who need the hypoxic drive to breathe.

Delayed detection of hypoxic events[3]

While the response time of the pulse oximeter is generally fast, there may be a significant delay between a change in alveolar oxygen tension and a change in the oximeter reading. It is possible for arterial oxygen to reach dangerous levels before the pulse oximeter alarm is activated.

Delay in response is related to sensor location. Desaturation is detected earlier when the sensor is placed more centrally. Lag time will be increased with poor perfusion and a decrease in blood flow to the site monitored. Performance of a neural block may cause the lag time to decrease while venous obstruction, peripheral vasoconstriction, hypothermia and motion artifacts delay detection of hypoxaemia. Increasing the time over which the pulse signals are averaged also increases the delay time.

Erratic performance with irregular rhythms

Irregular heart rhythms can cause erratic performance. During aortic balloon pulsation, the augmentation of diastolic pressure exceeds that of systolic pressure. This leads to a double or triple-packed arterial pressure waveform that confuses the pulse oximeter so that it may not provide a reading. Pulse oximetry is notoriously unreliable in the presence of rapid atrial fibrillation.

Nail polish and coverings

Some shades of black, blue and green nail polish may cause significantly lower saturation readings. Synthetic nails may interfere with pulse oximetry readings. One way to overcome this problem is to orient the probe so it transmits light from one side of the finger to the other side. The presence of onychomycosis, a yellowish gray color caused by fungus can cause falsely low SpO_2 readings. Dirt under the nail can also cause difficulty in obtaining reliable readings. Although there is one report of dried blood on a finger causing erroneous low saturation readings, other authors have found that dried blood does not affect the accuracy of the pulse oximeter.

Loss of accuracy at low values

Measurement of SpO_2 is less accurate at low values, and 70% saturation is generally taken as the lowest accurate reading.

Electrical interference

Electrical interference from an electrosurgical unit can cause the oximeter to give an incorrect pulse count (usually by counting extra beats) or to falsely register decrease in oxygen saturation. This problem may be increased in patients with weak pulse signals. The effect is transient and limited to the duration of the cauterization. Manufacturers have made significant progress in reducing their instruments' sensitivity to electrical interference and some monitors display a notice when significant interference is present. Steps to minimize electrical interference include locating the electrosurgery grounding plate as close to and the oximeter sensor as far from, the surgical field as possible; routing the cable from the sensor to the oximeter away from the electrosurgery apparatus;

keeping the pulse oximeter sensor and console as far as possible from the surgical site and the electrosurgery grounding plate and table; raising the high pulse rate alarm; and operating the unit in a rapid response mode. The electrosurgical apparatus and pulse oximeter should not be plugged in to the same power source.

Motion artifacts[11]

Motion of the sensor relative to the skin can cause an artifact that the pulse oximeter is unable to differentiate from normal arterial pulsations. Motion may produce a prolongation in the detection time for hypoxaemia without giving a warning. Motion is usually not a problem during general anaesthesia, but if the patient is shivering, moving about (as during inhalation induction of small children) or being transported it can be significant. Evoked potential monitors and nerve stimulators can produce motion artifacts if the pulse oximeter sensor is on the same extremity.

The ability of an oximeter to deal with motion artifacts depends on the correlation with the onset of the motion and the start of monitoring. If the motion precedes the onset of monitoring, there is a greater decrement in performance. Motion artifacts can usually be recognized by false or erratic pulse rate displays or distorted plethysmographic waveforms. Increased pulse amplitude is an indicator of movement but not necessarily of artifactual SpO_2 readings. Artifacts caused by motion can be decreased by careful sensor positioning on a different extremity from that being stimulated. Ear, cheek and nose probes may be more useful than finger probes in restless patients, and flexible probes that are taped in place, or probes lined with soft material are less susceptible to motion artifacts than clip-on probes. Neonates and children with their tiny digits and poor contact with probes are most susceptible to motion artifact.

Pulse oximeters vary in their ability to identify readings associated with movement. Lengthening the averaging mode will increase the likelihood that enough true pulses will be detected to reject motion artifacts. Some manufacturers use the R-wave of the patient's ECG to synchronize the optical measurement. Oximeters with signal extraction technology which use mathematical manipulation of the oximeter's light signals to measure and subtract the noise components associated with motion will have fewer artifacts.

Pressure on the Sensor

Pressure on the sensor may result in inaccurate SpO_2 readings without affecting pulse rate determination.

Hyperemia

If a limb is hyperemic, the flow of capillary and venous blood becomes pulsatile. In this situation the absorption of light from these sources will be included in the saturation computations with resulting decrease in accuracy of the oxygen saturation measured by pulse oximetry. A pulse oximeter placed near the site of blood transfusion may show transient decreases in oxygen saturation with rapid infusion of the blood.

Failure to detect absence of circulation

A pulse oximeter signal and a normal reading do not necessarily imply adequacy of tissue perfusion. Some pulse oximeters show a pulse despite inadequate tissue perfusion or even when no pulse is present, as ambient light may produce a false signal.

Discrepancies in readings from different monitors

A discrepancy in readings between difference brands of oximeters on the same patient at the same time is not uncommon.[12] One reason for this is differences in methods of calibration and the variation in the time it takes various monitors to detect desaturation.

Failure to detect hypoventilation

Hypoventilation and hypercarbia may occur without a decrease in hemoglobin oxygen saturation, especially if the patient is receiving supplemental oxygen. Pulse oximetry should not be relied upon to assess the adequacy of ventilation or to detect disconnections or oesophageal intubations. A capnograph is necessary to detect these complications. It is important to realize that while a capnograph detects oesophageal intubation or a disconnection, pulse oximetry detects only the effects of such a mishap, namely hypoxia, and valuable time may be lost before taking corrective measures.

Conclusion

Despite problems and limitations, pulse oximetry remains the standard of care in all clinical situations and its use for all patients under anaesthesia must be mandated. As with all monitors one must be familiar with its performance characteristics, advantages and limitations.

Does the use of pulse oximeters help prevent complications?

Certainly, early warning of hypoxic events helps the anaesthesiologist/intensivist take remedial action expeditiously, before irreversible organ damage occurs.

Does the use of pulse oximeters actually help save lives?

A closed claim analysis concluded that the incidence of critical incidents due to airway accidents declined in the 1980s since the introduction of pulse oximetry.[13] This led the ASA Standards for Basic Monitoring during anaesthesia to adopt pulse oximetry as of January1,1990.

A multicentre Danish study involving more than 20,000 patients, however, failed to demonstrate a decrease in mortality with the use of pulse oximetry even though the frequency of ischemic events were lower.[14] Despite this study, pulse oximetry has become an essential monitor, and few would argue against its universal use.

Intelligent use of pulse oximetry can truly help save lives and prevent disasters due to hypoxic events.

To quote an editorial in Anesthesiology "..as the blindfolded anesthetist walks unknowingly toward the cliff of hypoxia - whether due to problems of inspired gas, equipment failure, underventilation, or abnormal pulmonary shunting - the protective hand of the pulse oximetry sentry stops him from falling over the edge".[15]

References

1. *Stephen RC, Slater HM, Johnson AL, Sekelj P.* The oximeter-A technical aid for the anesthesiologist. Anesthesiology 1951;12: 541-555.
2. *Comroe JH, Botelho S* : The unreliability of cyanosis in the recognition of arterial hypoxemia. Am J Med Sci.1947; 214:1-8.
3. *Tremper KK, Barker SJ.* Pulse Oximetry. Anesthesiology 1989;70;98-108.
4. *Eisenkraft JB* : Pulse oximeter desaturation due to methemoglobulin. Anesthesiology 1988; 68:279-285.
5. *Barker SJ, Tremper KK, Hyatt J* : Effects of Methemoglobinemia on Pulse Oximetry and Mixed Venous Oximetry. Anesthesiology 1989; 7:112-117.
6. *Barker SJ, Tremper KK,* : The effects of carbon monoxide inhalation on pulse oximetry and transcutaneous PO_2. Anesthesiology 1987; 66: 677-679.
7. *Severinghaus JW, Spellman MJ.* Pulse oximeter failure thre sholds in hypotension and vasoconstriction. Anesthesiology 1990; 73: 532-537.
8. *Pologe JA* : Pulse oximetry : Technical aspects of machine design Int Anesthesiol Clinics1987; 25:137-153.
9. *Bourke DL, Grayson RF*; Digital nerve blocks can restore pulse oximeter signal detection Anesthesia and Analgesia 1991; 73: 815-817.
10. *Volgyesi GA, Spahr-Schopfer I.* Does skin pigmentation affect the accuracy of pulse oximetry? An in vitro study. Anesthesiology 1991; 75: A406.
11. *Trivedi NS, Ghouri AF,Shah NK, Lai E, Barker SJ* : Effects of motion, ambient light, and hypoperfusion on pulse oximeter function. J Clin Anesth. 1997 (3):179-83.
12. *Wutemberger G, Muller S, Matthys H, Sokolow I* : Accuracy of nine commercially available pulse oximeters in monitoring patients with chronic respiratory insufficiency. Monaldi Arch Chest Diseases 1994; 49: 348.
13. *Tinker JH, Dull DL, Caplan RA, et al.* Role of monitoring devices in prevention of anesthetic mishaps. A closed claims analysis. Anesthesiology 1989; 71; 541-546.
14. *Moller JT, Pederson T, Rasmussen LS, Jensen PF, Pederson BD, Ravlo O, Rasmussen NH, Esperson K, Johannessen NW, Cooper JB, Gravenstein JS, Chrammer-Jorgensen B, Wiberg-Jorgensen F, Djernes M, Heslet L, Johansen SH* : Randomised Evaluation of Pulse Oximetry in 20,802 Patients Anesthesiology 1993: 436-453.
15. *Fairley HB.* Changing Perspectives in Monitoring Oxygenation Anesthesiology (editorial) 1989 70: 2-4.