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Pulse oximetry – understanding the device and the sources of error

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INTRODUCTION

The first pulse oximeter was invented in 1974 by Takuo Aoyagi. In the 50 years since, it has become an indispensable monitor in anaesthesia practice and represents one of the most significant contributions to the safety of modern anaesthesia.

Monitoring patient oxygenation is an essential part of anaesthesia. Historically, this took the form of visual inspection of the patient's skin colour. However, "Is my patient pink or blue?" is a very blunt instrument for assessing oxygenation.

The ability to quantify oxygenation was improved by the development of the blood gas analyser in 1957 allowing clinicians to formally measure arterial oxygen saturation (SaO_2). SaO_2 is the fraction of total haemoglobin that is oxygenated in an arterial blood sample. Despite providing a much more precise measure of oxygenation, the delay between sampling and results limited its utility in continuous assessment of patient oxygenation.

The pulse oximeter is a device that enables non-invasive, continuous measurement of peripheral oxygen saturation, known as SpO_2. Its key advantage is that it provides a beat-to-beat measure of oxygenation, which, in most circumstances, accurately reflects the SaO_2 value.

Pulse oximeters provide additional useful data for the anaesthetist, displaying heart rate, a plethysmography trace and a perfusion index. However, this article focuses on explaining how the pulse oximeter determines oxygen saturation and explores common sources of error.

THE DEVICE

Physical components

The basic principle of pulse oximetry is that each cardiac cycle causes a transient increase in arterial blood volume within the tissue. If light is passed through this tissue bed, light absorption will increase as the volume of blood within the tissues increases.[1]

In order to measure the change in light absorption, each pulse oximeter contains at least two light emitting diodes (LEDs) and a photodetector. There are two broad physical arrangements of these components: transmittance and reflectance. In transmittance devices such as the familiar finger probe, the LEDs and photodetector are positioned on opposite sides of the device. Light emitted from each LED passes through the finger, and the photodetector measures the transmitted light.[2]

Reflectance-type oximeters are arranged so that the LED and photodetector are adjacent. Contrary to their name, emitted light is not reflected off a surface to the receiver but instead diffuses through the tissue bed.[3] These are usually applied to the forehead in situations where an adequate reading is unable to be obtained peripherally.

The photodetector measures the received light intensity, which creates a plot of light intensity (y-axis) against time (x-axis). Transmitted light intensity is mathematically converted to light absorption.[4]

By analysing light absorption at multiple wavelengths, pulse oximeters estimate the concentration of the different haemoglobin species in arterial blood. By estimating the relative proportions of oxyhaemoglobin (oxyHb) and deoxyhaemoglobin (deoxyHb) present, SpO_2 can be determined.

SpO_2 is the fraction of haemoglobin that is oxygenated as measured by a pulse oximeter. Mathematically, SpO_2 is defined as oxyHb divided by the sum of oxyHb and deoxyHb.

SpO_2 may sometimes differ from SaO_2, which is the fraction of total haemoglobin in its oxygenated form in arterial blood. Mathematically, this is the fraction of oxyHb divided by all haemoglobin species – namely oxyHb, deoxyHb, methaemoglobin, and carboxyhaemoglobin.

Signal processing

Starting with the LED's emission of light, five key processes take place to enable the SpO_2 value to be displayed.

1. Spectroscopy at two wavelengths of light.
2. Isolation of the arterial absorbance.
3. Comparison of the arterial absorbance at the two wavelengths.
4. Correlation of this ratio of absorbances to a SpO_2 value.
5. Data processing and display.

Step 1: Spectroscopy

Spectroscopy uses light of specific wavelengths to determine the concentration of an absorbing substance within its path. It is based on the laws of Beer and Lambert.

- Beer's law states that a beam of visible light passing through a chemical solution experiences absorption proportional to the solute concentration.
- Lambert's law states that the light absorbance of a layer of substance is proportional to the thickness of the layer.

These two laws are combined in the Beer-Lambert law, which states that total light absorption is equal to the product of the concentration of the absorbing substance, the distance light travels and the absorptive capacity (extinction coefficient) of the substance. Mathematically, this is written as:

$$A = \varepsilon LC$$

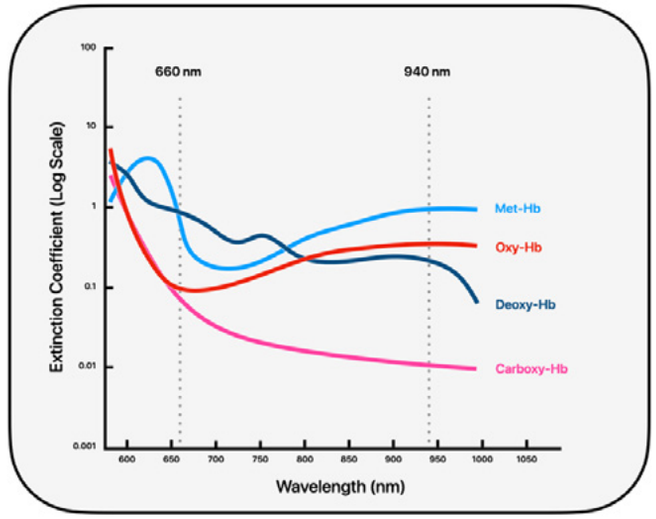
(Equation 1)

Where A is absorption, ε is the extinction coefficient of the substance, L is the path length, and C is the concentration of the substance.[5]

Spectroscopy is the technique used to determine the concentration of haemoglobin species within a blood sample. If the distance travelled by the light beam across the test tube and the substance's extinction coefficient are known, then the concentration can be determined.

Adult blood contains four species of haemoglobin: oxyhaemoglobin (oxyHb), reduced haemoglobin (deoxyHb), methaemoglobin (metHb) and carboxyhaemoglobin (COHb). Each haemoglobin species has a characteristic light absorption profile (Figure 1). When analysing a blood sample, it is necessary to use four different wavelengths of light to determine each species' concentration.

Figure 1. Absorption spectra of different haemoglobin species



The same basic principle is used in a pulse oximeter; however, in standard pulse oximeters, only two wavelengths of light are used. This is because the device only aims to measure the concentration of two substances, oxyHb and deoxyHb. The wavelengths used are 660 nm (red light) and 940 nm (near-infrared light).

The photodetectors used in pulse oximeters are not wavelength-specific. Therefore, the LEDs alternate brief light-emitting periods followed by a period where both are off. This happens 400 times a second. The device identifies which wavelength is being detected based on the timing of the signal received by the photodetector.[6]

In the situation where a mixture of two different substances (oxyHb and deoxyHb) are present within the light path, the Beer-Lambert equation becomes:

$$A = L(\varepsilon_0 C_0 + \varepsilon_d C_d)$$

(Equation 2)

Where ε_o is the extinction coefficient for oxyHb, C_o is the concentration of oxyHb, ε_d is the extinction coefficient for deoxyHb, C_d is the concentration of deoxyHb, and L is the path length.[4]

Unlike a test tube, a finger contains many different light-absorbing substances: bone, muscle, tendons, skin, and both arterial and venous blood. To determine the arterial concentration of oxyHb, the pulse oximeter cannot simply measure the total light absorption. Instead, it must isolate the component of light absorption occurring specifically within the arterial blood.

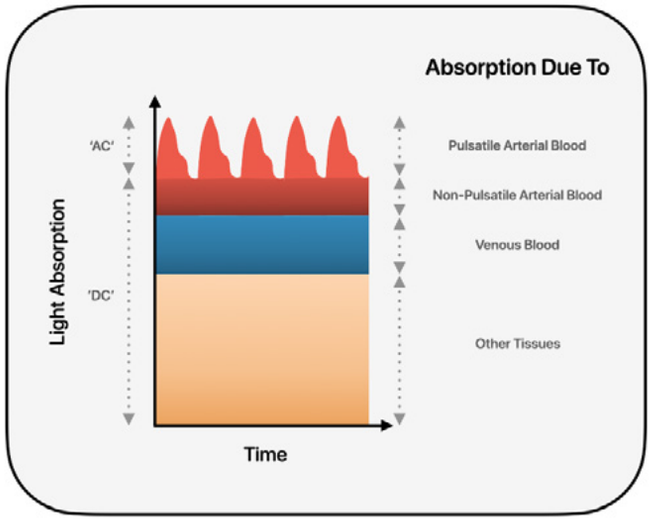
Step 2: Isolation of the arterial absorbance

The device exploits the pulsatile nature of arterial blood to exclude absorption occurring in tissues and venous blood. If we assume that the change in the volume of the fingertip during the cardiac cycle results mainly from arterial expansion, by comparing the absorption during systole to that during diastole, we can isolate the portion of absorption that occurs only within the arteries.

Figure 2 is a visual representation of this concept; the y-axis is absorption, and the x-axis is time. By convention, the pulsatile fraction of the absorption is referred to as "AC" absorption. This term is somewhat misleading as it does not refer to an alternating electrical current. In fact, it is not a current at all! A more

accurate description is “pulsatile absorption”, though “AC” remains the commonly used term in most texts. The static component of absorption is conventionally referred to as “DC”, which again is not “direct current”. This figure is not to scale as the AC component is often only 1-5% of the total absorption under normal physiological circumstances.[2]

Figure 2. Fractional absorption of light by different tissue components



Total absorption at any given time is the sum of the AC and DC absorption, which can be separated mathematically.[4]

Relating the AC absorption value back to the Beer-Lambert law, pulsatile expansion of the arteries increases the light path length (L), thereby increasing absorbance (Lambert’s law). This means that if we know the AC absorbance, we are a step closer to calculating the oxyHb concentration. Framing the previous equation in terms of pulsatile absorption only, we get the following:

$$A_{AC} = \Delta L(\varepsilon_0 C_0 + \varepsilon_d C_d)$$

(Equation 3)

There are still several unknowns in this equation. Path length is not measured by the device, so ΔL is unknown, and C_o and C_d have yet to be determined. This is where comparing the two wavelengths becomes important.[4]

Step 3: Comparison of the arterial absorbance at the two wavelengths

To solve the problem of the unknown path length, an *absorbance ratio* is calculated. This relies on the assumption that because both red and infrared light are travelling through the same tissue, the distance travelled (path length) is the same for both wavelengths. By dividing the absorbance of red light by the absorbance of infrared light, the change in length value (ΔL) can be removed from the equation. This ratio of absorbances is known as R.

The two remaining unknowns are the oxyHb and deoxyHb concentrations. But rather than the concentration of oxyHb, we want to determine the fraction of oxyHb. Conveniently, this is related to the fraction of deoxyHb! If we know that 80% of the Hb present is oxyHb, then the fraction of deoxyHb must be 20%, so we only need to determine one value, and the other will follow logically.

Rather than determining individual concentrations of oxy and deoxyHb, we could instead determine the total Hb concentration and the fraction in the oxyHb form. If we rewrite the R ratio with this in mind, we can remove Hb concentration (as this does not change regardless of whether red or infrared light is being used) and just express R in terms of the fraction of oxyHb (F_o). For the detailed mathematics of this, refer to Appendix 1.

The resulting equation looks like this:

$$R = \frac{(\varepsilon_0 F_0 + \varepsilon_d (1 - F_0))_{660}}{(\varepsilon_0 F_0 + \varepsilon_d (1 - F_0))_{940}}$$

(Equation 4)

This equation represents R as derived from the Beer-Lambert Law.

The device itself measures R from the received light intensity signals, which are converted mathematically to absorption values. R, as measured by the device, is expressed as:

$$R = \frac{AC_{660} \,/\, DC_{660}}{AC_{940} \,/\, DC_{940}}$$

(Equation 5)

Where AC_{660} is the pulsatile component of the red wavelength absorption, DC_{660} is the static component of the red absorption. AC_{940} is the pulsatile component of the infrared absorption, and DC_{940} is the static component of the infrared absorption.

Why has the DC signal been used in this measurement when we are interested in comparing only the pulsatile parts of the signal?

Because the red and infrared LEDs are different light sources, their emitted intensities are different. Both red and infrared received DC and AC signal amplitudes will be affected by the intensity of their corresponding LED emitter. Let’s say the received red signal has a DC amplitude of 10 and an AC amplitude of 1, while the received infrared DC amplitude is 40 and the AC amplitude is 2. Does this mean that the infrared light was less absorbed en route to the detector, or simply that the infrared LED was “brighter” to begin with?

In order to isolate any changes in pulse amplitude from changes in light intensity, the AC signal must be scaled by the DC signal at that wavelength. Dividing the AC signal by the DC signal gives a scaled AC signal, which is no longer a function of light intensity.[7] This is why the measured value of R is a scaled ratio (sometimes referred to as a “ratio of ratios”).

So, we have now reached a point where we have a value for R measured by the device. We can also convert that value to a fraction of oxyHb by substituting the measured R and the known extinction coefficients into Equation 4. Doesn’t that mean we have figured out SpO_2?

The answer is yes! This method of calculating saturation was used in the very first pulse oximeters from 1974-1981. They worked well for patients who were not hypoxic, but unfortunately, they were not very accurate at saturations below 90%.[8]

The reason for this inaccuracy has become clear more recently. Red and infrared light are strongly scattered by human tissue, so light does not take the most direct path between the LED and the photodetector. The detected light is actually a composite of light that has travelled many different paths of varying lengths.[3] It has been found that the scattering constants and light path length differ significantly between the red and infrared wavelengths. Consequently, the relationship between the oxyHb fraction and R cannot be derived directly from the Beer-Lambert law.[1] In essence, ΔL_{660} is not equal to ΔL_{940}, and therefore, these cannot be removed from the calculation of R.

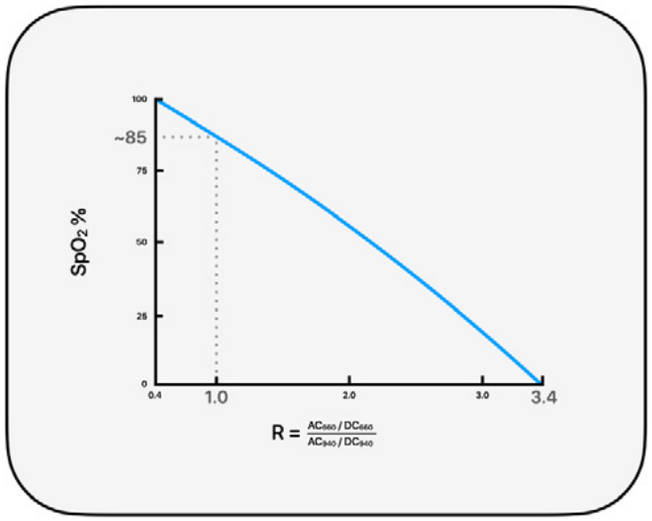
Fortunately, a way of linking the measured value of R to a value of SpO_2, which does not rely on knowing the difference in red vs infrared light path length, was found.

Step 4: Correlation of R to an SpO_2 value

In 1981, a device was developed that used a calibration curve to link measured R values to SpO_2 rather than the Beer-Lambert law, as described above.[8] This process experimentally determines the relationship between R and SaO_2. These pulse oximeters were found to have greater accuracy than those that used the Beer-Lambert law, and this has continued to be the process used in modern pulse oximeters.

To produce this calibration curve, subjects inhale hypoxic gas mixtures, and SpO_2 readings are compared to SaO_2 readings obtained simultaneously from a blood gas analyser. For ethical reasons, data is only obtained down to SpO_2 70%, and the remaining values are extrapolated (Figure 3).

Figure 3. Calibration curve for a pulse oximeter



Explaining this in terms of light absorption amplitude, each different model of pulse oximeter will have a slightly different relationship between the red light path length and the infrared light path length, depending on the physical positioning of the LEDs and photodetector.[9] This means that the calibration process must be performed for every new type of device that is manufactured.[10]

The calibration process allows the R value measured by the device to be linked to the correct calibration curve. It finds the equation linking R and SpO_2, which is expressed as:[9]

$$SpO_2 = K_1 + K_2R$$

(Equation 6)

A brief inspection of this curve demonstrates that low R values are associated with high values of SpO_2 and vice versa. Often, this curve is simplified to $SpO_2 = 110\text{-}25R$. Substitution of R = 1 into this equation results in the "when SpO_2 = 85% then R = 1", which is mentioned in many equipment texts. In reality, the exact equation will vary slightly from device to device.

Step 5: Data processing and display

The SpO_2 reading on a pulse oximeter is usually an average of readings over a period ranging from 2 to 20 seconds, depending on the device.

Signal averaging commonly follows a moving technique in which the pulsations analysed move sequentially. Signal averaging was developed to combat motion artefacts and decrease false alarms.

Increasing signal averaging time will reduce false alarms significantly, but this comes at the cost of longer start-up time (time from the application of probe until the reading is displayed), delayed detection of desaturation events (increased lag time) and missed short desaturations.[11]

Lag time depends on both signal averaging time and the location of the sensor, simply because it takes longer for the blood to reach the distant measurement site. Centrally located sensors in areas of high cardiac output will respond more rapidly than peripherally placed sensors, particularly when peripheral perfusion is poor.[2]

Application of principles to a clinical event

What makes a pulse oximeter useful is its ability to rapidly detect a desaturation event. Ideally, in terms of pulse oximeter design, we want changes in the relative concentrations of oxyHb and deoxyHb to result in proportional changes in the measured value of R. Imagine if there was one wavelength of light that was only absorbed by oxyHb and another that was only absorbed by deoxyHb. Any change in relative haemoglobin fractions would result in an immediate and equal change in R. Unfortunately, this is not possible within the current range of wavelengths produced by commercial LEDs.

So, of all the wavelengths of light that could have been used, why were 660 nm and 940 nm chosen in the development of this device? Like many breakthroughs in science, the answer is part design and part luck! In 1939, an ear oximeter was developed that used two wavelengths of light. Red light was used as it was known to be absorbed by oxyhaemoglobin, while green light was selected because its transmission appeared to be unaffected by haemoglobin saturation. As it turned out, the production of the "green" light resulted in an unidentified infrared wavelength also being produced.[12]

In the early 1970s Aoyagi was using a later version of this device (employing both red and infrared wavelengths) to research cardiac output measurement. He was troubled by pulsatile flow in the ear interfering with his calculations. He found that by adjusting the ratio of red to infrared light used, he could cancel out the pulsatile "noise". Serendipitously, he then observed that when he held his breath, the pulsations returned. He hypothesised that this was because the change in ratio of oxygenated to deoxygenated haemoglobin had changed the degree of absorption in one wavelength of light relative to the other. This principle could then be used in reverse to estimate the degree of saturation.[13]

When commercial production of pulse oximeters began in the early 1980s LEDs were beginning to be mass produced, but there was a limited selection of wavelengths available. It is likely that 660 nm and 940 nm were the most readily available wavelengths in the red and infrared range at that time.

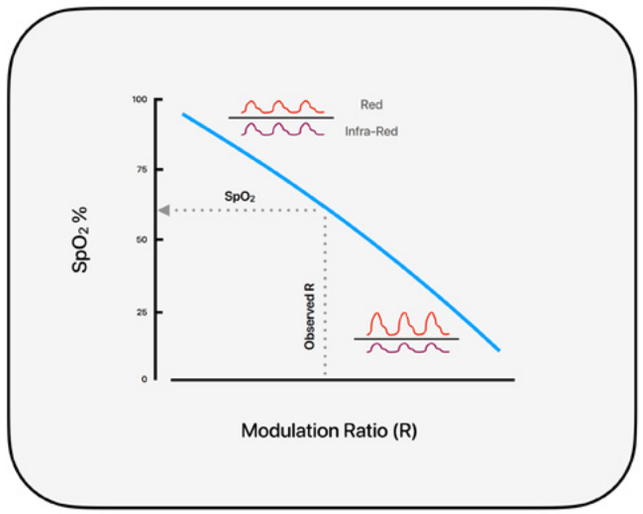
So, how exactly does desaturation cause a change in the relative absorbance of these two wavelengths of light?

Referring back to Figure 1, we can see that at 660 nm (red light), deoxyHb and oxyHb have wide separation in their absorptive capacity (ε), where deoxyHb absorbance is 10 times greater than oxyHb. However, at 940 nm (infrared) their absorptive capacities are similar (oxyHb is slightly greater). Additionally, the two wavelengths also fall on either side of an isobestic point where the absorption spectra cross.

Consider the situation where a desaturation occurs. Any decrease in oxyHb is met with a reciprocal increase in deoxyHb under normal physiological circumstances. With the red LED, the wide separation in extinction coefficients (10-fold), means that an increase in deoxyHb will result in a dramatic increase in overall light absorption (large increase in AC amplitude). However, at the infrared LED, reciprocal changes in oxyHb and deoxyHb will result in a much smaller amplitude change. This is because the increase in absorption by deoxyHb is partially offset by the reduction in absorption by oxyHb.

Figure 4 demonstrates this. The amplitude of the red AC component increases dramatically as saturations decrease while the infrared AC component changes minimally.

Figure 4. Red and infrared AC amplitude at different SpO_2 values



In summary, the value of R will increase whenever there is an increase in red light absorption relative to infrared light, which is reflected as a decrease in SpO_2.[3] The reciprocal nature of the changes in oxyHb and deoxyHb fractions and the wide versus narrow separation of extinction coefficients at these particular wavelengths is essential for R to change in this way.

ERRORS

Clinically, SpO_2 is assumed to reflect SaO_2 and thus provide data about patient oxygenation. However, a number of assumptions must be met for this to hold true.

- The ratio of path lengths for the red to infrared light in the patient is the same as during the empirical calibration.
- There are no other haemoglobin species present besides oxyhaemoglobin and deoxyhaemoglobin.
- The AC signal is entirely attributable to arterial blood.

Errors in the SpO_2 reading displayed may be conveniently classified into three related groups:

1. Errors due to the intrinsic accuracy and calibration of R with experimental data.
2. Errors due to the use of only two wavelengths of light.
3. Errors in isolating the pulsatile component of the signal.

Errors due to the intrinsic accuracy and calibration of R with experimental data

Intrinsic accuracy

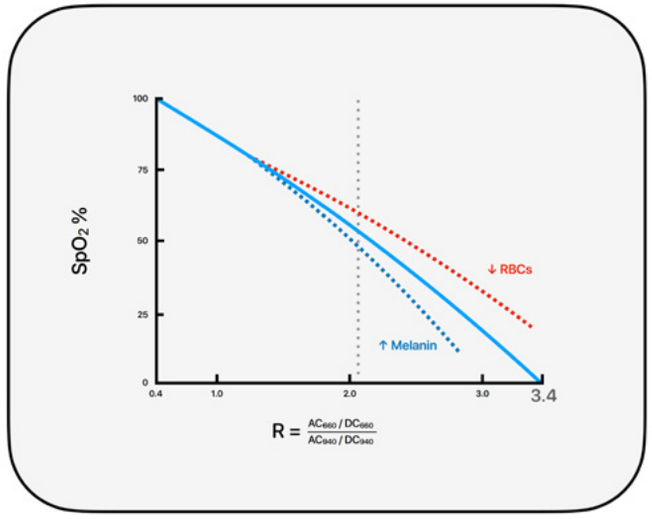
The accuracy of a pulse oximeter is evaluated by the difference between SpO_2 measured by the device and SaO_2 measured by laboratory co-oximetry. Between a SpO_2 of 70% and 100%, the accuracy of most devices is reported to be within 2-3%, and the international standard for manufacture requires ≤4% error in this range.[10] Below an SpO_2 of 70%, accuracy is not stated but will be less since this portion of the calibration curve is by extrapolation only.

Degradation of LEDs can occur over time. Interrogation of devices in current use has shown that actual wavelengths emitted by the LEDs may vary significantly from 660 nm and 940 nm.[10] Since the absorption curve for deoxyHb is steeper than oxyHb, an error of 4 nm in the red wavelength will produce an error that increases in magnitude as SpO_2 decreases.[10] Electrical integrity may also be compromised with repetitive use, where damage results in electrical noise being detected as part of the signal. This will cause the R-value to approach 1, resulting in SpO_2 readings approaching 85%.[10]

The empirical calibration process assumes that the red to infrared path length ratio will not change between subjects and will be consistent between the calibration subjects and the patients on whom the device will be used.[1] What this means in practice is that if an individual patient's finger differs in the way that it scatters light compared to the fingers of the calibration subjects, there is the potential for error. This could include differences in finger anatomy (i.e. clubbing[14]), tissue characteristics (i.e. nail thickness[15]) and sensor location (i.e. use of a finger probe on the helix of an ear[3]).

Under normal physiological conditions, the ratio of red to infrared path lengths decreases with desaturation (path length decreases as light absorption increases). In situations where the amount of light scatter differs from the calibration population in the red wavelength more than the infrared wavelength, there is the potential for errors to occur predominantly in conditions of hypoxaemia. In terms of the SpO_2 versus R relationship, the slope of this relationship is altered, resulting in a measured value of R that over or underestimates SpO_2 depending on whether the slope is increased or decreased. Additionally, the magnitude of the error increases with decreasing saturation. Clinically, this has been shown to occur in severe anaemia and darkly pigmented skin, albeit the errors are in opposite directions. Figure 5 demonstrates this.

Figure 5. Change in SpO_2 vs R relationship with change in red light scattering



Skin pigmentation

Historically, studies of the impact of skin pigmentation on pulse oximeter accuracy have been conflicting. Difficulties with these study results included how skin tone is measured and small sample sizes.[16]

In terms of device calibration, manufacturers are only required to have 15% of participants with darkly pigmented skin when calibrating R with SpO_2. If the ratio of red to infrared light path lengths in the calibration patients is not representative of the ratio of path lengths in patients with increased melanin, there is the potential to decrease accuracy.[9]

In the red-light spectrum, individuals with dark skin have an extinction coefficient which is more than 11 times that of fair-skinned individuals. In the infrared spectrum the difference is around 10 times greater.[17] Melanin light absorption decreases almost linearly with wavelength. Does increased melanin fraction potentially change the scattering properties of the two wavelengths?

A recent study investigated the effect of the range of wavelengths emitted by LEDs used in current pulse oximeters. Despite the fact that LEDs are theoretically emitting light of one specific wavelength, they actually emit light in a wavelength band (called bandwidth), which peaks at the specified 660 nm and 940 nm. When these devices were compared to devices that use a narrowband (smaller range) source, a systematic difference in the calculated R values was found.[9] So the R values obtained by conventional oximeters are inaccurate in darkly pigmented patients.

From first principles, if melanin increases the amount of light absorption disproportionately in the red wavelength, this will reduce sensitivity to the small increase in red light absorption that occurs with desaturation. This results in an undetected increase in the slope of the SpO_2 versus R relationship. Hence, for a given value of R, SpO_2 will be overestimated, with the difference between reported and actual values increasing as SpO_2 decreases.

Recent clinical trials in the wake of the COVID-19 pandemic have supported these theoretical concerns. A recent systematic review concluded that pulse oximetry can overestimate SaO_2 in patients with darkly pigmented skin and that the magnitude of the error is likely to be greater at lower SpO_2 values.[18]

Anaemia

A change in haemoglobin content compared to the normal healthy adults used for calibration can theoretically alter the red to infrared path length ratio and create a bias in R.[3] The amount of light scatter is influenced by the number of red cells in the light path.[5] In anaemia, reduced scattering shortens the optical path length and decreases total absorption. Under desaturation conditions, the increase in absorption due to deoxyHb will be overestimated. The result is an undetected decrease in the slope of the SpO_2 vs R relationship about a point near 80% SpO_2.[3] Hence, for a given value of R, SpO_2 will be underestimated, with the difference between reported and actual values increasing as SpO_2 decreases.

In clinical studies, hypoxaemic patients have been found to have falsely lower SpO_2 readings relative to their actual SaO_2 values, but only in severe anaemia.[19] Anaemia alone (without hypoxaemia) has not been found to cause errors in SpO_2 readings.[20]

Errors due to the use of only two wavelengths of light

Two wavelengths of light can only identify two absorbing substances. If there are more than two substances within the arterial blood absorbing light at the wavelengths used, this additional absorption cannot be isolated. The increased absorption will then be erroneously attributed to one of the two substances being measured.

Carbon monoxide poisoning

Carbon monoxide (CO) is a colourless, odourless gas formed by the incomplete combustion of carbon-containing fuels. CO has 210 times greater affinity for haemoglobin than oxygen. Carboxyhaemoglobin (COHb) results from the combination of CO with heme iron. In healthy non-smokers, HbCO levels are 1-2% due to endogenous production by the metabolism of heme-containing compounds and their presence as a low-level pollutant in the urban environment. Chronic smokers have levels ranging from 3-8%, increasing to 10-15% immediately post-smoking. CO poisoning occurs when symptoms of toxicity appear, generally at levels above 10%, escalating in severity with increasing concentration.[21]

The pulse oximeter assumes that all Hb present is either oxyHb or deoxyHb. Other haemoglobin species (MetHb and COHb) can only be measured using two additional wavelengths (as occurs in a laboratory with arterial blood samples).

The extinction coefficient of COHb at the red wavelength is similar to oxyHb. At the infrared wavelength, COHb absorbs virtually no light.[5] Most equipment textbooks state, "this results in an erroneously high reading". However, an increase in red absorption relative to infrared absorption will result in an increase in R, which corresponds to a decrease in SpO_2 (Figure 4). High COHb does result in an erroneously high reading, but this is not the explanation.

In fact, the haemoglobin oxygen dissociation curve is shifted to the left in the presence of high levels of COHb. Due to this shift, concentrations of deoxyHb are also significantly decreased because the O_2 that is bound is unable to be released at the tissue level.[21]

This decrease in deoxyHb results in a decrease in red light absorption. This effect will be larger than any increase in absorption due to the presence of COHb as the extinction coefficient (absorptive capacity) of deoxyHb is 10 times greater.[5] Infrared absorption will also decrease, but to a lesser extent, due to the lower extinction coefficients for both deoxyHb and oxyHb. Hence, R will decrease, and SpO_2 will falsely increase.

Methaemoglobin

Normal haemoglobin contains iron in the ferrous (Fe^{2+}) state. Methaemoglobin (MetHb) occurs when the ferrous iron is oxidised to its ferric (Fe^{3+}) form, which prevents oxygen from binding to the affected heme molecule. Clinically, increased levels of MetHb may occur in certain congenital conditions and as a result of exposure to oxidising compounds such as benzocaine, prilocaine, nitrates, aniline and dapsone. Additionally, MetHb impairs tissue oxygen delivery by causing a left shift of the oxyHb dissociation curve, impairing oxygen offloading by the other three heme molecules in the affected haemoglobin.[22]

Methaemoglobin readily absorbs both red and infrared light (extinction coefficients at both 660 and 940 nm are high). Consequently, significant levels of methaemoglobin will increase both red and infrared absorption. In situations where there is equal absorption of both types of light, the R value will trend towards 1. Saturations will, therefore, trend towards 85%, which may result in either an over or underestimation of the actual saturations depending on the clinical scenario.[5]

Sulfhaemoglobin

Sulfhaemoglobin (SHb) is formed by the irreversible oxidation of Fe^{2+} to Fe^{3+} and the binding of sulfur atoms to the porphyrin ring of the haemoglobin molecule.[5] Similar to MetHb, affected heme molecules in SHb are unable to carry oxygen. However unlike MetHb, the molecular changes in SHb result in a rightward shift of the oxyHb dissociation curve, which facilitates oxygen offloading at the tissues. As a consequence, the level of tissue hypoxia is generally less severe with SHb compared with MetHb.[23] Formation of SHb occurs with excess exposure to sulfur-containing compounds such as dapsone, sulphonamides, sumatriptan, sulfamethoxazole and phenazopyridine.[23] Clinically, this presents as blue-grey skin discolouration.

Considering the light absorption characteristics of SHb, the peak absorption of SHb occurs at 630 nm. At 660nm, absorption is significantly higher than metHb, oxyHb and deoxyHb. Absorption at 940 nm is not reported. Clinically, patients have low SpO_2 despite normal SaO_2 on laboratory testing, suggesting that the additional 660 nm absorption falsely increases the R value, resulting in falsely low SpO_2 readings. However, it is possible that absorption at 940 nm is also high, and R trends toward 1, since many clinical case reports state SpO_2 readings of 80-84%.[5]

Abnormal Hb species

Uncommon Hb variants have been linked to spuriously low SpO_2 readings. These include Hb Lansing, Hb Bonn, Hb Koln, Hb Hammersmith, and Hb Cheverly. The proposed mechanism in these instances appears to be related to high absorbance at 660 nm, resulting in falsely low SpO_2.[5]

Dyes

Methylene blue, patent blue V, and isosulfan blue (an isomer of patent blue used in the US) are injected subcutaneously during surgery to trace the lymphatic drainage of the region.

There are conflicting reports on the contribution of these compounds to SpO_2 errors when injected into tissues as intended. There is also contention about which dyes cause more significant errors.[24] The explanation for this is likely dependent on the extent of systemic absorption as IV injection has been demonstrated to cause errors with all blue dyes.[25–27]

Methylene blue has peak absorption at 665 nm. The patent blue V peak is at 637 nm, and isosulfan blue at 638 nm. All blue dyes exhibit significant absorption at 660 nm and very little absorption at 940 nm. The additional absorption at the 660 nm wavelength, while 940 nm absorption is unchanged, results in an increase in the value of R. An increase in R will result in a SpO_2 reading which is erroneously low.[5]

Indocyanine green and indigo carmine have absorption peaks around 800 nm and 600 nm, respectively. They do have some absorbance at 660 nm, but it is significantly lower than the blue dyes discussed above. Intravenous administration has demonstrated a decrease in saturation readings of only 3-4% with indocyanine green, and no change with indigo carmine.[25]

Nail polish

The majority of colours of nail polish do not interfere with pulse oximetry.[28] Some shades of blue, green, brown, purple and black nail polish absorb light at 660 nm. From first principles, you might expect this to result in artefact and a decrease in saturation readings. However, since nail polish is non-pulsatile, the increased absorption should not alter the AC absorbance, which is what is used to calculate R. There will, however, be a systematic change in red to infrared path length ratio compared to the calibration volunteers who were nail polish free and this may lead to a systematic bias in results.

Clinical studies of whether this is associated with a significant change in SpO_2 readings have been conflicting.[29] A recent meta-analysis suggests that blue, black and brown nail polishes are associated with a small decrease in SpO_2 (less than 2%), which is within the accepted margin of error of pulse oximetry values.[28]

Errors due to isolation of the pulsatile signal

Poor perfusion

Low perfusion interferes with the device's ability to correctly isolate the AC from the DC component of the absorbance signal. Under normal conditions, this is only 1-5% of the total signal.[30]

When the pulsatile component of the absorbance signal is very small relative to the background signal (low AC to DC ratio), the pulse oximeter amplifies the signal more.[31] This allows the pulse oximeter to determine saturations from a range of patients who generate different amplitudes of pulsatile absorbance.

With very high amplification (for example, vasoconstriction), there is an increase in the likelihood of an artefact being analysed as part of the pulsatile signal.[31] This is analogous to how the edges of an object in a photo become blurred when you zoom in. In this scenario, you have difficulty pinpointing where the exact edge of the object is. In the same way, the device has difficulty isolating where the AC component begins and ends.

Because this artefact is equally present in both the red and infrared absorbance signals, both red and infrared absorbances will increase, and therefore, the ratio will approach R = 1. This gives SpO_2 trending towards 85%. SpO_2 will be either over or underestimated, depending on the clinical situation.[31]

Other factors that contribute to erroneous readings in this situation include an increase in the ratio of venous blood relative to arterial blood in the digit, and an increase in tissue oxygen extraction, such that the venous blood present will have a lower saturation. Therefore, any DC components that are erroneously included will have a lower saturation than usual. Hence, readings may be lower than 85% in some situations.[11]

Improvements in device design to address this issue include having a minimum signal-to-noise ratio below which no value will be displayed (rather than further amplification occurring). In addition, there is some evidence that reflectance-type oximeters placed on the forehead may have greater accuracy in low-perfusion states.[32]

Movement

Motion interferes with the ability to accurately isolate the pulsatile component of the signal in a similar way to poor perfusion. However, rather than reducing the AC:DC ratio, the size of the AC signal is artefactually increased.[31] Finger motion causes some of the venous and tissue components (particularly venous blood and tissue fluid) to form part of the AC signal (essentially a fake AC signal). In this situation, venous blood and tissue fluid are the components artefactually included in the AC signal, rather than the nonspecific artefact described for poor perfusion. Since the tissue and venous components have lower saturation, SpO_2 will be artefactually decreased.[11]

Solutions to this problem used in modern oximeters include data averaging, holding data, alarm delays, and the use of motion-resistant technologies such as adaptive filtering, frequency domain analysis, and cardiac gated averaging.[11]

Probe malposition or poor fit

If the device is malpositioned on the patient's digit, the light of one or both wavelengths may bypass the finger and reach the photodetector directly or without passing through pulsatile tissue. This is referred to as shunting. Unless the shunting changes the path length of each wavelength in exactly the same ratio as determined in the calibration, SpO_2 readings will be erroneous. Whether this results in an over or underestimation of the actual SaO_2 will depend on which of the wavelengths is more strongly shunted.[3]

Increased venous pulsations

Artefact from venous pulsations most commonly occurs when the venous outflow to the finger is occluded (e.g during NIBP cuff deflation), or in patients with severe tricuspid regurgitation. It can also occur when the probe is placed on the earlobe or forehead of patients in the Trendelenberg position. In this situation, part of the analysed AC signal will be from venous blood, resulting in an artefactually decreased SpO_2 reading.[5]

Ambient light

The photodetector is not wavelength-sensitive, so its input is only referenced to the wavelength by the timing of the LEDs. The LEDs cycle red, infrared, and both off at a rate of 400 Hz.[6]

If a contaminating light source were to have a particular frequency that coincided with only the red detection window or only the infrared detection window, then this would lead to an error. Such errors were more common with older models of pulse oximeters, which had a lower LED switching frequency.[33]

Due to the very high frequency of modern oximeters, this issue is no longer a common source of error with ambient operating room lighting.[34] The off period allows quantification of the amount of light that passes through the finger from external sources, and this is subtracted from the received signal when the LEDs are on. In addition, most devices have physical shielding to prevent ambient light from reaching the photodetector.

CONCLUSION

It has been more than 50 years since the invention of the pulse oximeter. It has become a universal device for monitoring patient oxygenation and has made significant contributions to improving patient safety. Despite many technological advances, assumptions remain inherent in the design of the device, which underpin significant errors that can occur. Understanding the design of the device helps us interpret the data it provides and allows us to identify and respond appropriately to these errors.

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APPENDIX 1

Using Equation 2, the ratio of pulsatile absorbance at 660 nm to that at 940 nm is expressed as:

$$R = \frac{\Delta L(\varepsilon_0 C_0 + \varepsilon_d C_d)_{660}}{\Delta L(\varepsilon_0 C_0 + \varepsilon_d C_d)_{940}}$$

(Equation 2b)

If we assume that the light path length is the distance between the LED and the photodetector and that this does not differ between the two wavelengths, then ΔL will cancel out from this equation and it becomes:

$$R = \frac{(\varepsilon_0 C_0 + \varepsilon_d C_d)_{660}}{(\varepsilon_0 C_0 + \varepsilon_d C_d)_{940}}$$

(Equation 2c)

If we assume that oxyHb and deoxyHb are the only species present in arterial blood, then their relative proportions are related to one another by:

$$F_0 + F_d = 1$$

(Equation 2d)

Where F_o is the fraction of oxyHbO_2 present, and F_d is the fraction of deoxyHb present, substituting Equation 2d into Equation 2c gives the following:

$$R = \frac{\left(\varepsilon_0 F_0 + \varepsilon_d (1 - F_0)\right)_{660} cHb}{\left(\varepsilon_0 F_0 + \varepsilon_d (1 - F_0)\right)_{940} cHb}$$

(Equation 2e)

Assuming that the concentration of Hb does not vary between the two wavelengths, this can now be cancelled out leaving:

$$R = \frac{\left(\varepsilon_0 F_0 + \varepsilon_d (1 - F_0)\right)_{660}}{\left(\varepsilon_0 F_0 + \varepsilon_d (1 - F_0)\right)_{940}}$$

(Equation 3)