Uses and abuses of pulse oximetry

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It is extraordinarily difficult to guess the state of a patient’s arterial oxygenation subjectively. The ability to detect decreased haemoglobin oxygen saturation depends on many factors including circulatory state, skin pigmentation, haemoglobin concentration, ambient light colour and intensity. The introduction in the early eighties of pulse oximetry allows reasonably accurate objective assessment of haemoglobin oxygen saturation of arterial blood (Spo2), cheaply, non-invasively, and with a very low morbidity.

Apart from the objective indication of Spo2, pulse oximetry may be described as ‘fail-safe’ in that the technique fails and therefore alarms if the pulsatility of the pulse waveform decreases to below a critical level. Further information may be gained by the display of a plethysmograph trace which enables the differentiation between the pulse waveform, and artefacts. Indeed this author would go so far as to say that no pulse oximeter should be used unless a plethysmograph trace is displayed.

Knowledge of the principles of the technique allows one to understand the limitations and use the information displayed safely. The absorption spectra of normal adult haemoglobin in its saturated and desaturated states are shown in fig 1. Also shown are the absorption spectra of two common dyshaemoglobins, carboxyhaemoglobin and methaemoglobin, which lead to erroneous oxygen saturation values being indicated by conventional pulse oximetry as described later. It can be seen from the spectra of oxygenated and deoxygenated haemoglobin that there is a change in absorption dependent upon the amount of oxygen being carried by the haemoglobin molecule. It must be made clear that these spectra are for pure haemoglobin solutions unlike the natural state in vivo. In living tissue it is necessary to separate the change in energy absorption due to any change in oxygen saturation from all other energy absorbants at those wavelengths. This separation is done by assuming that, over short periods of time in peripheral tissue, absorption of the energy will be constant by all tissue components except that due to the pulsation of arterial blood.

Energy from light emitting diodes (LEDs) of wavelengths 660 nm and 940 nm is projected through peripheral tissue (finger, toe, ear lobe, bridge of nose). The emerging energy, after absorption by all the tissue layers including arteries and arterioles, is detected by a semiconductor sensor. To allow a single site for the transducer, the LEDs are energised alternatively at a rate of around 1 kHz. The signal from the sensor is electronically separated into plethysmograph signals for each of the wavelengths. These signals are pulsatile due to the variability of the arteriolar cross sectional area and the change in axis of erythrocytes with cardiac cycle. In simple terms the absorption due to tissues other than the arterial system are constant and therefore can easily be eliminated electronically. The ratios of the absorptions at the two wavelengths are applied to an electronic ‘look-up’ table, giving the Spo2 value. The signal is averaged over a few seconds to eliminate beat-to-beat changes in saturation and to reduce artifacts. In general the more expensive the pulse oximeter, the more complex the algorithms used by its microprocessor to reduce artifacts.

The advantage of pulse oximetry is that it is easily applied and has a much more robust probe than transcutaneous blood gas monitoring. However, it must be remembered that it is indicating percentage haemoglobin oxygen saturation in arterial blood and not partial pressure of dissolved oxygen, although these are related by the oxygen dissociation curve (ODC). The ODC is changed by a number of other variables as shown in fig 2 and also varies with other types of haemoglobin. P50 is an abbreviation for the partial pressure of oxygen at which the haemoglobin is 50% saturated and is a useful...
shorthand term for quantifying shifts in the ODC.

Safety in the use of pulse oximetry depends upon a knowledge of the limitations of the technique. The limitations are classified in the table. The limitations may be classified in two ways namely as to whether they are technical or physiological limitations and whether they are safe or potentially dangerous. Safe limitations may be defined as conditions when the pulse oximeter is not indicating the correct value of SpO₂ but the user is warned that the value may be inaccurate. Dangerous limitations are those where the device appears to be operating satisfactorily but is indicating the wrong value of saturation.

**Mechanical artefacts** are caused by any movement of the probe on the extremity. The technique is especially sensitive to this problem in the vicinity of magnetic fields emitted by electronic equipment. With such equipment, nothing metallic should be in the field area. Mechanical artefacts are obvious when the oximeter displays a plethysmograph trace. *Electromagnetic interference (EMI)* may also cause malfunction that is obvious if a trace is displayed and always leads to an alarm situation. The most common source of EMI in hospital practice is radio frequency surgical diathermy. However, a recent problem is the susceptibility of all electromedical equipment to the electromagnetic radiation from cellular telephones. Although the emissions from cellular telephones are of low power, when they are in close proximity the electric field emitted is often strong enough to disrupt microprocessor function. A special class of EMI is the intense magnetic field in the vicinity of magnetic resonance imaging or nuclear magnetic spectroscopy equipment. With such equipment, nothing metallic should be in the high field area. Special pulse oximeters have been developed in which both the LEDs and the photodetector are housed in the case of the apparatus and the energy is led to the patient and back to the photodetector by optical fibres.

Pulse oximetry is *pulse dependent*; the technique requires an adequate pulse volume and may be said to be fail-safe as all devices warn of an inadequate pulsatile signal. With ever-advancing computer software, modern pulse oximeters are better able to function with irregular rhythms.

It is essential that the dangerous limitations of the technique are understood. Pulse oximeters are *type calibrated* by the designers and manufacturers using fit healthy young adults with normal adult haemoglobin who are desaturated by stages from 100% down to a minimum of 80%. It would be unethical to desaturate below 80% and therefore any values of SpO₂ are less likely to be as accurate as those above 80% as calibration <80% is by extrapolation.

The calibration of pulse oximeters is done against in vitro arterial blood samples tested in a co-oximeter, which is a spectrophotometer dedicated to assessing haemoglobin oxygen saturation. SpO₂ should never be compared with saturation values indicated by blood gas analysis as saturation in this case is derived from the measurement of pH and carbon dioxide and oxygen tension.

An oximeter is considered to be in error when the saturation reading differs from the co-oximeter value by more than ±2%.

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inspired oxygen concentration and failure or disconnection of mechanical ventilation. Placement of the probe centrally (tongue, cheek) rather than peripherally (finger, toe) may halve the delay in indication of desaturation.¹

Extraneous energy sources, especially bright visible or infrared light may flood or overload the semiconductor detector. If the pulse oximeter does not alarm to indicate flooding it may display a value of 85%. This is because a ratio of red/infrared of one is equivalent to an SpO₂ of 85%. A similar problem which has been called the penumbra effect often occurs with small infants and children. In this case the pulse oximeter may over-read or under-read due to there being a different path length of tissue for each of the wavelengths. This occurs with very small fingers or when the LEDs are projecting tangentially through the tip of a digit. For this reason, probes especially designed for use with babies and children have been produced and should always be used.

The most serious limitations of pulse oximetry are related to the fact that as currently available devices only use two wavelengths the displayed value is always based on a calibration curve for normal adult haemoglobin. Current pulse oximeters are unable to detect dysaehaemoglobins and will therefore produce erroneous results without warning. Co-oximeters are safe to use in the presence of abnormal haemoglobins because they use more than two wavelengths; one machine uses as many as 17. Multiwavelength co-oximeters actually indicate the proportions of the common dysaehaemoglobins.

Of the dysaehaemoglobin, carboxyhaemoglobin is the most dangerous as it is fairly common and makes the pulse oximeter over-read. The absorption spectrum of carboxyhaemoglobin is shown in fig 1. Carboxyhaemoglobin is caused by the inhalation of even very small quantities of carbon monoxide. Common sources of carbon monoxide are the internal combustion engine, conflagrations, barbecues, tobacco smoke, and inadequately ventilated combustion of coal or gas in heating systems. It must now be considered negligent to use pulse oximetry on patients who have been at risk of carbon monoxide inhalation. For every 1% of carboxyhaemoglobin circulating, the pulse oximeter over-reads by approximately 1%. Fifty per cent of cigarette smokers have carboxyhaemoglobin concentrations of >6%; those involved in accidental inhalation may have much higher concentrations.

Methaemoglobinemia, whose absorption spectrum is also shown in fig 1, may be congenital or, more commonly, acquired. This dysaehaemoglobin may be considered ‘safer’ as the greater the concentration of methaemoglobin, the more the indicated SpO₂ tends toward the value of 85%. Acquired methaemoglobinemia occurs when the iron of haemoglobin is oxidised from the ferrous Fe⁺⁺ to the ferric Fe⁺⁺⁺ form. There are a large number of drugs and other chemicals which may induce methaemoglobinaemia including a number of antimalarials, dapsone, EDTA, local anaesthetic agents, methylene blue, nitrates, nitrites, nitric oxide, para-aminosalicylic acid, and the sulphonamides.

Fetal haemoglobin has similar absorption spectra to adult haemoglobin and therefore pulse oximeters indicate SpO₂ within the same limits of accuracy as with adult haemoglobin.²³ This is also true of sick cell haemoglobin,⁴ although in both cases the ODC will be shifted.

Certain dyes such as methylene blue and indocyanine green, which may be administered for diagnostic purposes, have a drastic effect upon the accuracy of pulse oximeters. The accuracy recovers rapidly after bolus injection as the dye dilutes.

Bilirubin was reported to render pulse oximetry inaccurate when compared with co-oximetry. Examination of the absorption spectrum of bilirubin shows no absorption at 660 nm and 940 nm. The discrepancy between pulse oximetry and co-oximetry was due to the fact that co-oximeters operate over different wavelengths, normally visible, where bilirubin does absorb. This demonstrates how important it is to compare like with like. The latest multiwavelength co-oximeters can distinguish bilirubin and also fetal haemoglobin. The accuracy of pulse oximetry is not effected by jaundice, although the ODC will be shifted.

Pulsatile veins may cause pulse oximeters to under-read as the technique cannot tell the difference between pulsating veins and arteries. This was first noted in cases of tricuspid incompetence but recently there has been some suggestion that venules in neonates and children with hyperdynamic circulation may be more pulsatile than in adults due to the shorter length of the arteriovenous Anastamoses in the microcirculation.

Skin pigmentation and opaque nail varnish usually make pulse oximeters fail safely but there have been some conflicting reports of inaccuracies.

Pulse oximetry and the neonate

Possibly the greatest concern in the use of pulse oximetry in paediatric practice is the use of the technique on the sick neonate. In neonatal intensive care units, pulse oximetry is used both as an indicator of hypoxia and hyperoxia to protect against retinopathy of prematurity.

It is important to remember that the ODC for fetal haemoglobin is shifted to the left of the ODC for adult haemoglobin so that although the haemoglobin saturation is correctly indicated, the partial pressure of oxygen to which the tissues are exposed at a cellular level will be different.

There have been a number of papers validating pulse oximetry against transcutaneous oxygen tension measurement,⁵⁻⁷ however, to suggest that pulse oximetry is a safe alternative to protect the neonate from hypoxia and subsequent retinal damage is dangerous. Reliability of pulse oximetry in detecting hyperoxia is controversial because small changes in SpO₂ >90% are associated with relatively large changes in the arterial oxygen tension (Pao₂).⁸ Several
authors have stated that theSpo2 should be kept below 95%: '80%–90% is safe'\textsuperscript{9}, <95% ('Pao2 >90 mm Hg') [>12.0 kPa] and is safe\textsuperscript{6}; >92% 'may be associated with hyperoxia'\textsuperscript{10}; '80%–95% is safe'\textsuperscript{11}; and 'recommended goal should be 90%'\textsuperscript{12}. These authors fail to take into account the poor correlation between Spo2 and the partial pressure of oxygen in the plasma. Apart from the general factors shown in fig 2, the ODC of fetal haemoglobin is shifted to the left compared with adult haemoglobin; in the worst case, an Spo2 of 80% may correspond to a PaO2 of 4.0 kPa (30 mm Hg) with consequent hypoxic damage. However, this left shift progressively reduces as the ratio of fatal to adult haemoglobin decreases with increasing age.

Pulse oximetry has been used to provide closed loop control of inspired oxygen concentration to neonates\textsuperscript{13} using target saturations of between 94% and 96%, but yet again, the poor correlation of SpO2 to PaO2 in the upper portion of the ODC was not taken into account. Although pulse oximetry adds important safety monitoring against hypoxia, the PaO2 of the premature neonate rather than the SpO2 must be monitored to protect against hyperoxia.\textsuperscript{14}

Further reading


