DIAGNOSTIC TESTS

Oximetry

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SYNOPSIS

The oxygen stores of the body are small, so life-threatening hypoxaemia can develop very rapidly with few clinical signs. The availability of robust and reliable pulse oximeters has revolutionised the safe monitoring of patients with unstable cardiorespiratory conditions, and those having medical and surgical procedures. While oximetry is now best practice in these circumstances, care must be taken in interpreting the results. There are confounding factors that may produce an erroneous signal and physiological factors that will affect the interpretation of the result. In the absence of these factors, the instruments are accurate detectors of arterial oxygen saturation, in the range between 100% and 70% with varying but reasonable performance down to 55%. The basic principles of operation are important to understand so that physiological interpretation is adequate and erroneous results can be identified.

Index words: hypoxaemia, haemoglobin, oxygen.

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Introduction

Patients at risk of hypoxaemia may need continuous monitoring of their oxygenation. Blood gas analysis requires arterial puncture and only measures the oxygenation at the time of the sample. By measuring oxygen saturation (instead of partial pressure) pulse oximetry enables non-invasive monitoring. The continuous measurement of the pulse rate is a bonus.

The technology supporting clinical oximetry has been available for more than 80 years, but pulse oximeters have only been commercially available for about 20 years. Early oximeters required a cumbersome heated probe to 'arterialise' blood in the ear lobe. They were also difficult to calibrate and notoriously unstable. Nowadays relatively cheap and reliable oximeters have revolutionised the *in vivo* monitoring of patients' oxygenation during a wide range of critical clinical situations.[1]

Definitions

SaO_2	% of total haemoglobin combined with oxygen
SpO_2	Saturation as measured by pulse oximeter. This is a 'functional' saturation ignoring abnormal haemoglobin species such as carboxyhaemoglobin and methaemoglobin.
PaO_2	Partial pressure of oxygen in arterial blood (mmHg or kPa). The relation between SaO_2 and PaO_2 is shown in Fig. 2.
CaO_2	The content (in mL/100 mL blood or mol/L of blood) of oxygen in blood. The ordinate of Fig. 2 can be calibrated as content yielding the same shape curve.

Physiological principles

While oximeters may be used to assess the efficiency of pulmonary gas exchange, at least in relation to oxygen uptake, they are more suited to assessing the adequacy of tissue oxygen delivery. Measuring oxygen in the arterial blood is important because serious acute hypoxaemia is notoriously difficult to detect clinically and by the time clinical cyanosis develops, the patient is usually in a parlous state. The oxygen stores of the body are small so the viability of many tissues is critically dependent on continuous delivery of an adequate oxygen supply. Oxygen delivery is proportional to the blood flow and arterial oxygen content (CaO_2 [mL O_2 per 100 mL blood]). For the whole body:

oxygen delivery = cardiac output (Q) x CaO_2 x 10

These variables are difficult to measure directly and rapidly, however CaO_2 is linearly related, at least over relatively short periods of time, to the saturation of haemoglobin in arterial blood (SaO_2). As oximeters provide a rapid and reliable *in vivo* measure of SaO_2 this variable can be substituted for CaO_2. This has been a very valuable advance, as long as the principles and sources of error are understood.

Technical aspects

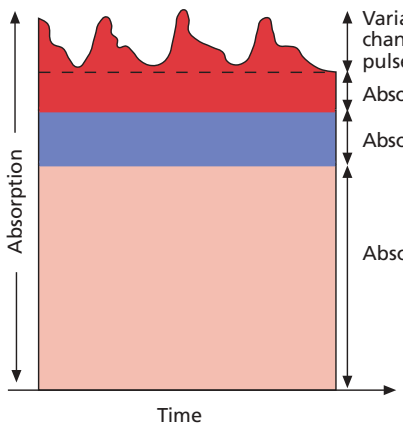
A pulse oximeter detects the change in transmission of two wavelengths of light across a capillary bed, usually in the finger. The sensor is placed on the nail with the light source against the finger pulp. The detectors can be small because they are only receiving two wavelengths, one to detect oxygenated haemoglobin (O_2Hb) and one to detect reduced haemoglobin (HHb). The absorption of light is related to the expansion of the capillary bed with the pulse (Fig.1). By comparing the light transmission through the pulsatile 'arterialised' capillary blood with the non-pulsatile venous blood the oximeter can calculate the haemoglobin saturation. Saturation is calculated as:

O_2Hb / [O_2Hb + HHb]

This is the so-called 'functional saturation', and is expressed as a percentage.[2]

Fig. 1

Absorption of light transmitted through the finger during pulse oximetry



It has been suggested that pulse oximeters should be called 'pulse spectrophotometers'. This would emphasise that they are inferring oxygen saturation from the well-known colour change between oxygenated and reduced haemoglobin and that, despite the elegant use of the pulse form to separate arterial blood from the other light absorbing structures in the finger, there are sources of error inherent in this technique which need to be appreciated.

While the vast majority of devices in clinical use measure transmitted light, newer devices are being designed to measure the light reflected off pulsating tissue surfaces. These devices are being used in perinatal monitoring and in patients whose peripheral perfusion may be compromised, as in open heart surgery. Reflectance devices are currently hampered by poor signal-to-noise ratio and the need to detect very small pulsatile signals, but advances in technology are likely to overcome these difficulties.

Sources of error

The results of pulse oximetry can be affected by technical problems and physiological factors.

Calibration problems

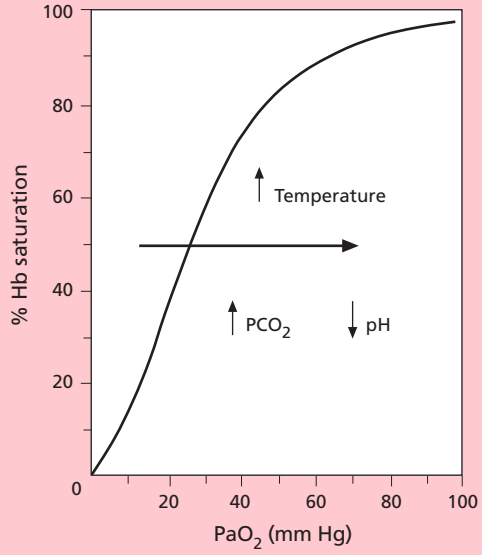
The machines do not need regular calibration by the operator and the probes and electronics are extraordinarily robust. The machine will not display a result and will warn if it cannot detect an adequate pulse signal. It is fitted with an alarm, which can be set at low (or high) saturation levels as desired. Original calibration by the manufacturer is based on the empirical relation between *in vivo* pulse oximetry (SpO_2) and the SaO_2 measured on simultaneously sampled arterial blood in a CO-oximeter.

CO-oximeters, so called because they measure carboxy- or CO haemoglobin, are now fitted to all modern blood gas analysers. They use multiple wavelengths of light to detect the four different forms of haemoglobin. The calibration process generally relies on data generated from healthy volunteers made hypoxaemic to generate SaO_2 values between 70% and 100%. When the SaO_2 is reduced to between 70% and 40% the pulse oximeters become significantly inaccurate, particularly below 55%, and fail to track rapidly developing profound hypoxaemia.[3] However, it can be argued that the accurate detection of falls between 85% and 70% is of most use in clinical monitoring.

Fig. 2

Oxyhaemoglobin dissociation curve

The relation between oxyhaemoglobin saturation and partial pressure of oxygen in blood can be changed by factors such as temperature and acid-base balance. An increased partial pressure of carbon dioxide (PCO_2), increased temperature or reduced pH move the oxyhaemoglobin dissociation curve to the right. The ordinate can be calibrated as oxygen content (CaO_2), yielding the same shape curve.



Physiological factors (Table 1)

The presence of abnormal haemoglobins disturbs the relation between SaO_2 and CaO_2. 'Functional saturation' ignores the possible presence of methaemoglobin, carboxyhaemoglobin and other abnormalities of haemoglobin. These abnormal forms will not carry oxygen normally and will add to the denominator of the O_2Hb / [O_2Hb + HHb] ratio. Usually abnormal haemoglobins only comprise a few percent of the total, even in heavy smokers who have increased concentrations of carboxyhaemoglobin. However, common drugs such as paracetamol and sulfa drugs can induce the formation of methaemoglobin. Anaemia will also reduce the oxygen content without changing the calculated functional saturation.

There will be difficulties relating the SpO_2 to PaO_2 if the position of the oxyhaemoglobin dissociation curve has been shifted by influences such as acid-base balance and carbon dioxide tension. If the SpO_2 is above 92% the partial pressure of oxygen (PaO_2) can change rapidly with very little change in saturation (Fig. 2). This latter physiological feature limits the

Table 1

Physiological factors which affect interpretation of a saturation measurement (when assessing oxygen content or partial pressure)

- Saturation above 92%
- Dyshaemoglobinaemia, commonly presence of methaemoglobin and/or carboxyhaemoglobin
- Anaemia
- Shift of the oxyhaemoglobin dissociation curve (e.g. acidosis, alkalosis, hyper- or hypocapnia)

usefulness of pulse oximetry in, for example, the assessment of pulmonary gas exchange efficiency and the detection of hyperoxia in preterm babies. Measurement of the partial pressure of arterial oxygen (PaO_2) from *in vitro* samples or transcutaneous electrodes may be preferable for monitoring hyperoxia in the 'flat' part of the dissociation curve.

Pulse oximetry has also been an enormous boon in intensive care units. However, measurements can be difficult to obtain in low perfusion states or where inotropes such as dopamine are being used to sustain blood pressure.

Confounding factors (Table 2)

Abnormal haemoglobins and anaemia are 'physiological' confounders but these abnormalities can also affect the accuracy of the measurements. In animal experiments, SpO_2 decreases as methaemoglobin increases up to 35%, and SpO_2 increases as carboxyhaemoglobin increases up to 70%. Modest concentrations of these haemoglobins will not substantially change SpO_2 which is a functional saturation. Anaemia has to be severe (50 g/L) before it interferes significantly with the measurement.

Abnormal dyes and pigments such as methylene blue (used to treat methaemoglobinaemia) and severe hyperbilirubinaemia may interfere. In most clinical circumstances, these disturbances will not be present to a significant degree, but they need to be kept in mind. Strong superficial pigments such as nail polish must be removed and signal failure may occur in black patients although careful positioning on the less pigmented nail bed usually overcomes this problem. Venous pulsation may confuse the signal, reducing the displayed saturation, particularly where a tourniquet is applied above the probe or in the presence of right heart failure or tricuspid incompetence. Excessive motion of the probe and strong incident light can also cause an erroneous or inadequate signal. Motion artifact is also a problem in many longer-term settings where movements can be interpreted as a pulse.

Table 2

Confounding factors causing an inaccurate or unobtainable SpO_2 measurement *in excess* of any physiological effect

- Methaemoglobin and carboxyhaemoglobin
- Anaemia
- Dyes and pigments
- Low perfusion
- Venous pulsations
- Motion artifact
- Excessive incident light

The pulse oximeter does not measure partial pressure of oxygen in arterial blood (usually expressed as mmHg) and the relationship between SpO_2 and PaO_2 is complex.

Clinical applications

Reliable pulse oximeters are now indispensable in all emergency departments, intensive care units (adult and neonatal) and operating theatres. Their use is considered to be good practice for procedures requiring sedation or instrumentation of the respiratory tract ranging from cardiac catheterisation to endoscopy and bronchoscopy. Their use in these procedures has uncovered quite alarming transient hypoxaemia requiring the use of supplemental oxygen. It is desirable to maintain the SpO_2 above 90%.[4]

The routine use of pulse oximeters in operating theatres and recovery rooms has coincided with a dramatic decrease in perioperative morbidity and mortality although, interestingly, a cause and effect relation has not been confirmed.[5] Clearly, disastrous errors such as incorrect connections in anaesthetic machines can be quickly recognised.

Patients presenting to emergency departments with cardiorespiratory disorders are routinely monitored with pulse oximetry. However, it is important to identify when the additional information available from *in vitro* analysis of an arterial blood gas sample is critical for management. Patients with worsening asthma, deteriorating pneumonia or left heart failure and those with chronic obstructive pulmonary disease developing clouded consciousness on supplemental oxygen all need arterial carbon dioxide partial pressure ($PaCO_2$), pH and base excess measurements (Fig. 2).

Pulse oximeters in sleep investigation laboratories have substantially contributed to the explosion of knowledge about sleep and breathing over the last few decades. They are also extensively used during exercise testing in pulmonary function and cardiac stress test units. Here, small falls in PaO_2 in the higher range will be difficult to detect, but hazardous falls will be readily identified.

Finally, pulse oximeters have a place in the non-procedural doctor's office where the detection of acute or chronic hypoxaemia may be important – as in the assessment of patients requiring home oxygen therapy. An SpO_2 above 90% in a patient with chronic obstructive pulmonary disease is reassuring whereas a lower measurement would suggest the need for confirmatory measurement of arterial blood gases. Experience with these devices, and their widespread adoption, have emphasised their status as a truly important advance in non-invasive patient monitoring and investigation.

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Conflict of interest: none declared

Self-test questions

The following statements are either true or false (answers on page 151)

5. Carboxyhaemoglobin causes pulse oximeters to show an increase in oxygen saturation.
6. Nail polish should be removed from a patient's finger before a pulse oximeter is attached.

Book review

Therapeutic Guidelines: Antibiotic. Version 12.

Melbourne: Therapeutic Guidelines Limited; 2003.

407 pages. Price $33, students $25.30, plus postage.

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How can one review a book which has become such a common sight in general practitioners' surgeries, hospitals, and everywhere prescriptions are written, and which was the first in a wave of therapeutic guidelines in Australia?

This book has been used for the selection of antibiotics in several circumstances:

- where the practitioner has a limited knowledge of the infection they are treating
- where the comorbidity of the patient makes antibiotic selection more complex
- where there is unknown life-threatening sepsis
- where there have been previous adverse reactions to antibiotics which are the first or second choice
- in different physiological states – pregnancy, renal impairment, childhood.

The main section of the book is a set of headings of infections and infestations of all body systems with recommended first- and second-line therapy. For practitioners seeking third-line medications for a likely or known organism where there is a history of adverse reaction to the first- or second-line drugs, it is necessary to consult Table 49 which gives the likely antibiotic resistance for most organisms.

The appendices of this book are most useful. In Appendix 1 the adverse drug reactions are subclassified by their frequency which is actually given numerical status in the introduction.

Appendix 3 is a set of desensitisation protocols for antimicrobial therapy. This is extremely useful for remote and rural practice where alternative medications may not be available for several days, and in circumstances where life-threatening infections require an antibiotic to which the patient is sensitised. Appendix 10 provides a reproduction of the CARPA antibiotic guidelines which are well used by nurses and general practitioners in remote areas.

The guidelines on intravenous antimicrobial use in Appendix 6 could be an Australia-wide standard for hospitals, and home intravenous antibiotic therapy. Appendix 7 contains a guide on monitoring of blood levels with due emphasis on aminoglycosides. Appendix 8 provides a useful guide on paediatric dosing, while Appendix 9 deals with dosing during lactation and pregnancy and Appendix 11 advises on dosing in renal impairment with and without dialysis.

I have a dislike of guidelines that do not quote supporting evidence to help practitioners judge the reliability of the recommendations. To add references would swell the volume beyond pocket size, but without them the guidelines could appear to be based only on expert opinion. The detail of these guidelines also demands a more useful retrieval system than flicking through a book. Although an electronic version is available for use on a personal computer, more portability would be useful.*

Overall, this is an excellent little book. It should be owned by all prescribers in book or electronic form for quick reference.

* *Editor's note:* The supporting references are available in the electronic version of the guidelines (eTG Complete) and a palmtop format is being considered.