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Review

Principles of pulse oximetry and its clinical application in neonatal medicine

Win Tin*, Mithilesh Lal

The James Cook University Hospital, University of Durham, Middlesbrough TS4 3BW, UK

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SUMMARY

Pulse oximetry is one of the most commonly used monitoring devices in clinical medicine. It was first introduced to neonatal medicine in the mid-1980s to monitor oxygenation and guide therapy, and it is now used widely in the delivery room during resuscitation. More recently, it is utilized to screen for congenital heart disease. Pulse oximetry is based on the variation in the ratio of the light absorbances of tissues during systole and diastole. It has become the mainstay of non-invasive continuous oxygen monitoring but with a wide variation in clinical practices and without good research evidence. This article provides a brief historical overview of pulse oximetry development, its principles, advantages and limitations, and the clinical applications in neonatal medicine.

1. Principles of pulse oximetry

Assessment of arterial oxygen saturation (SaO_2) by pulse oximetry is based on the Beer–Lambert law (also known as Beer–Lambert–Bouguer law) that relates the attenuation of light to the properties of the materials through which the light is travelling; and photoplethysmography, a non-invasive optical technique used to detect blood volume changes in the microvascular bed of the tissue [1].

Light-emitting diodes in conventional pulse oximeters transmit two light energies, 660 nm (red light) and around 940 nm (infrared light) to the semiconductor photodetector placed around an extremity. The red and the infrared lights are used because of the differences in the light absorption by oxyhemoglobin (HbO_2) and deoxyhemoglobin (Hb). In the red wavelength region, HbO_2 absorbs less visible light than Hb, whereas it is reversed with the infrared wavelength (Fig. 1).

During systole, the transmitted light intensity decreases through tissue because of increases in the absorption of light by the different hemoglobins in the arteries and arterioles. During diastole, there is an increase in the transmitted light intensity through the tissue because of the decrease in absorption. Conventional pulse oximeters compare the ratios of absorbances of the two wavelengths of light during systole and diastole. Built-in algorithms convert the logarithmic ratios of the maximum and minimum transmission of red and infra-red lights to pulse oxygen saturation (SpO_2) [2].

There is a small discrepancy between the measurement of arterial oxygen saturation by an invasive method and by pulse oximetry; hence, the former is denoted as SaO_2 and the latter as SpO_2.

2. Historical perspectives

The relationships between the absorption of light, the strength of the light source, and the light path length were first described by Johann Heinrich Lambert, a Swiss mathematician, physicist, astronomer, and philosopher in 1760. This was further investigated by August Beer, who published his findings in 1852 as the Beer–Lambert law. Felix Hoppe-Seyler first described the optical absorption spectra of the red blood pigment and the two different absorption bands in 1864, and his recognition that the binding of oxygen to red blood cells was a function of haemoglobin, creating the compound oxyhemoglobin, was one of the major landmarks of pulse oximetry [2–4].

The analysis of tissue oxygenation by spectrophotometry was introduced by Ludwyn in 1931 and the development of the first device capable of continuously monitoring oxygen saturation of human blood was published by Karl Matthes in 1935. In 1940, Glen Millikan, an American physiologist, built the first portable device for monitoring pilots during high-altitude flight (the Millikan oximeter). This system underwent several modifications and was used mainly in aviation. In 1949, Earl Wood developed an oximeter

* Corresponding author. Tel.: +44 (0)1642 854834; fax: +44 (0)1642 854874.
E-mail address: Win.tin@stees.nhs.uk (W. Tin).

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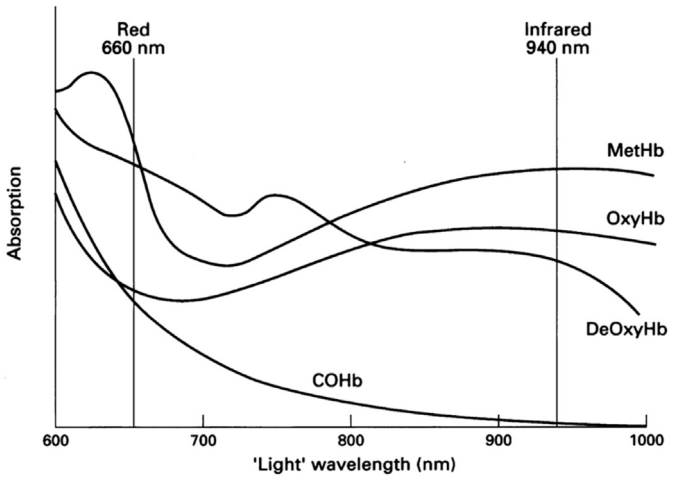

Fig. 1. Absorption spectra of oxyhemoglobin (OxyHb) and deoxyhemoglobin (DeOxyHb) of normal adult hemoglobin, as well as methemoglobin (MetHb) and carboxyhemoglobin (COHb). (Reproduced from Moyle [10] with permission by Archives of Disease in Childhood.)

that could be placed on the ear for continuous oxygen saturation monitoring using two wavelengths of light, and in 1964, Robert Shaw, an American surgeon, developed an oximeter using eight wavelengths. Hewlett–Packard marketed these in 1970 but the units were large, heavy, and expensive and were not suitable for bedside clinical monitoring.

Monitoring arterial oxygen saturation by pulse oximetry was developed in 1972 by Aoyagi and Kishi, who realized that pulsatile changes in the ratio of red and infrared light energy absorption could be used to compute the pulse oxygen saturation (SpO_2). The device was first commercialized in 1981, and the use of pulse oximetry for continuous oxygen monitoring in newborns was first described in 1986 [5]. It was not long before the use of pulse oximetry became the mainstay of non-invasive, continuous monitoring of oxygenation in newborns [6].

3. Advantages, limitations, and practical considerations

The main advantages of pulse oximetry are that the device can be easily applied by a sensor probe that does not require calibration, heating, nor the need to frequently change its position. It also measures oxygen saturation, a basic physiologic determinant of tissue oxygen delivery. In addition, pulse oximeters have a high sensitivity to detect hypoxemia.

The safe and efficient use of pulse oximetry depends on the knowledge and understanding of its physiological and technical limitations. The limitations that are of practical importance are described below.

3.1. Detection of hyperoxemia

The main physiological limitation of pulse oximetry is the inability to detect hyperoxemia in the higher SpO_2 range (>90%) because the shape of the oxygen–hemoglobin dissociation curve. Thus, relatively small increases in SpO_2 can be associated with a large increase in PaO_2 [7,8] (Fig. 2). This is particularly important for preterm infants receiving supplemental oxygen because of their vulnerabilities to oxygen toxicity and oxidative stress [9].

3.2. Calibration and accuracy

Pulse oximeters are calibrated by the data obtained from healthy adults with normal adult hemoglobin, and correlated to arterial

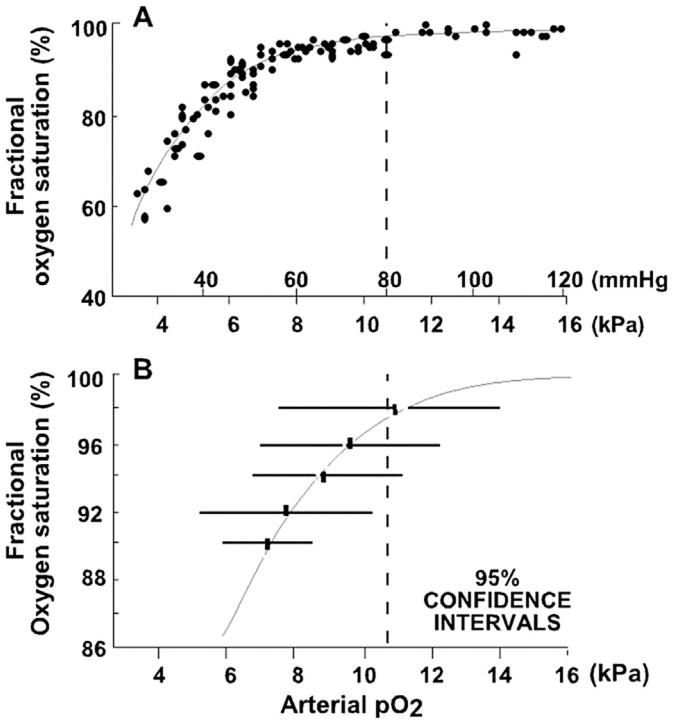

Fig. 2. Relationship between SpO_2 (display by oximeter calibrated to reflect fractional oxygen saturation) and arterial partial pressure The dashed line marks the TcO_2 above which there was increased risk of severe retinopathy of prematurity. Note that pulse oximeters calibrated to display functional saturations produce about 2% higher than those reflecting fractional saturation (Appendix 1). (Reproduced from Flynn et al. [20] with permission by BMJ Books.)

blood samples tested in vitro by a co-oximeter that actually measured SaO_2. Calibrations for SpO_2 values <80% can only be made by extrapolation and are less likely to be accurate. Most manufacturers of pulse oximeters claim an accuracy of ±2%; this is within acceptable limits [10].

3.3. Response delays

There may be response delays to indicate the change in oxygen saturation for technical reasons. This response depends on the set "signal averaging time" of the oximeter. Most pulse oximeters have a default time of 8 s to display the average value of SpO_2 measured over this period, but this can also be adjusted from 2 to 32 s in most oximeters. Long averaging times may reduce the frequency of alarms but will delay the response time and may not detect brief hypoxemic episodes [11]. Delayed responses may also result from irregularities in the pulse volume or rhythm that can slow the computer used to display the change in SpO_2.

3.4. Presence of abnormal hemoglobins

SpO_2 is overestimated in the presence of carboxyhemoglobin, and decreases in the presence of methemoglobin.

3.5. Motion artefacts

These are caused by movements of the sensor probes and poor peripheral perfusion, often leading to false alarms. Newer pulse oximeters with signal extraction technology (SET) can measure the SpO_2 during patient motion and low perfusion by separating the pulsatile arterial signal from the other signals [12]. In clinical