Original articles

Pulse oximeter and transcutaneous arterial oxygen measurements in neonatal and paediatric intensive care

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SUMMARY Pulse oximeter (SaO2P) measurements were compared with direct arterial line oxygen saturation (SaO2) from co-oximeters in 92 instances in 43 patients, and with arterial line oxygen measurements (PaO2) in 169 instances in 81 patients. The mean (SD) absolute difference between SaO2P and SaO2 was 2.6% (2.4) after attempt to correct for the co-oximeter falsely measuring a proportion of fetal haemoglobin as carboxy haemoglobin. For 19 infants and children ≥5 months old, who have very little fetal haemoglobin, the mean (SD) absolute difference of 27 comparisons was 1.8% (2.1). Comparison of SaO2P and PaO2 measurements in 46 instances when PaO2 was <6-67 kPa showed SaO2 to be <90% on 40 occasions. In 24 instances when PaO2 was ≥13.3 kPa the SaO2P was ≥98% on 22 occasions. In 23 infants undergoing neonatal intensive care, transcutaneous oxygen monitors were compared with arterial PO2 measurements in 60 instances. The mean (SD) absolute difference between PaO2 and transcutaneous oxygen measurements was 1.60 kPa (1.73). Ten of the 60 comparisons had differences >2.67 kPa and three >5.33 kPa (maximum 8.40 kPa).

Pulse oximetry is a clinically useful technique for managing oxygenation but further studies are needed to confirm its safety in premature infants at risk of retinopathy of prematurity.

Information concerning the adequate uptake, transport, and unloading of oxygen is vital in the management of infants and children undergoing intensive care. While indwelling arterial lines can be used to obtain these data, insertion of catheters may be difficult and cause discomfort and serious side effects; thrombosis and blood loss may occur. Samples taken by stab give unreliable data and the procedure is painful. Correction of the anaemia caused by repeated blood sampling is usually achieved by transfusion of adult haemoglobin. By changing the position of the oxygen dissociation curve, these transfusions may compromise uptake of oxygen in the lungs if there is a low alveolar PO2. To overcome the problems inherent in repeated blood sampling heated transcutaneous PO2 monitors (TcPO2) have been widely adopted. Although overall correlations between TcPO2 and arterial PO2 (PaO2) are high,¹⁻⁴ a considerable proportion of the comparisons reported have shown differences between transcutaneous and arterial line measurements. This is particularly important when these monitors have been used for clinical rather than research purposes.⁵⁻⁶ Lack of agreement between TcPO2 and PaO2 measurements is particularly likely in infants older than 8 weeks² and when skin perfusion is poor.⁸⁻⁹ To compensate for these errors it is the policy in some intensive care units, including ours, to validate TcPO2 measurements using samples from arterial line.

In other units, however, TcPO2 values are regarded as reliable enough to direct changes in inspired oxygen concentrations and mechanical ventilator management without such validation.

In this study we report our experience with pulse oximetry (S¹O²P) and TcPO2 measurements in the monitoring of oxygenation in neonatal and paediatric intensive care.
Patients and methods

A total of 85 infants and children were studied. Twenty-four were undergoing treatment in the neonatal intensive care unit at St Mary's Hospital. Their ages ranged from 1 to 47 days, gestational age at birth from 24 to 40 weeks, and birth weights from 690 to 3970 g. Most of them (99%) were receiving additional inspired oxygen, 70% were being ventilated, and 12% were receiving continuous positive airways pressure (CPAP).

The remaining 61 infants and children were undergoing treatment at the Brompton Hospital; 53 were receiving paediatric intensive care and eight undergoing cardiac catheterisation. Their ages ranged from 1 day to 13 years; 53 had congenital heart disease and eight had major respiratory disorders.

A pulse oximeter sensor (Nellcor) was used to obtain non-invasive measurements of arterial oxygen saturation. In this technique, light of two wavelengths is transmitted through a tissue bed from the surface of the skin. On the opposite side of the tissue bed there is a photodetector which senses the transmitted light. Within the oximeter a light plethysmograph is constructed for each arterial pulse waveform in the tissue bed and calculations made of the light absorption with each pulse. Thus background colour change is ignored (except in terms of the intensity of the light source), and only pulsatile (arterial) light absorption measured. The technique is independent of skin colour, thickness, or texture. The response time is affected if the application site is cold, even if pulsations are present, and the instrument cannot work in the presence of very low arterial blood pressures. Audible alarms can be set for low saturation readings and for high or low heart rates. The amplitude of the light plethysmograph used to calculate each saturation value is displayed on the front of the machine as a linear array of diodes emitting light. The calculation of saturation by the oximeter was expressed as an average over a particular time period; an average of two to three seconds of data was taken in this study, during which time the arterial waveforms had to be of adequate quality—that is, resemble blood pressure waveforms and be of adequate amplitude compared with baseline noise. For most of our comparisons the analog output of each arterial waveform was examined on an oscilloscope.

The pulse oximeter sensor was attached to the child's finger, toe, or lateral side of the hand or foot in a position which matched the arterial line, thus avoiding possible errors caused by intracardiac or ductal shunts. The sensor was covered with a dark blue mitten or sock to reduce the effect of ambient light, particularly during phototherapy.

PaO₂ measurements from the arterial line samples were made on autocalibrating blood gas analysers (Corning 178 and Radiometer ABL) within two minutes of sampling. Calibrations were checked daily by chemical pathology technicians against standard reference solutions at three levels. Samples of blood from arterial lines were analysed for oxygenated and reduced haemoglobin, carboxy-haemoglobin and methaemoglobin on co-oximeters (Corning 2500 or IL282), also within two minutes of sampling. These instruments were calibrated against control solutions once daily.

Pulse oximeter measurements were compared with arterial line PO₂ concentrations on 169 instances in 81 patients, and with arterial line saturation measurements corrected for methaemoglobin and carboxyhaemoglobin on 92 instances in 43 patients.

Measurements of TcPO₂ (Hewlett Packard 78850A, Radiometer TCM20, Kontron 820, and Novametrix 850) were made at skin temperatures of 44°C, and the sites for comparisons were chosen to avoid inconsistencies that could result from a right to left shunt. All TcPO₂ sensors were applied by trained nurses, and were recalibrated before each application (at four hourly intervals) for each patient. No infant was allowed to lie on the sensor. TcPO₂ monitors were used only in the neonatal intensive care unit.

Measurements of TcPO₂ were compared with arterial line PO₂ measurements in 60 instances on 23 infants (mean age 10.5 days, maximum 47 days).

When comparing alternative methods of measurement, correlation coefficients may be of interest but in many cases are misleading, so they were not included. The absolute differences were analysed on the assumption that measurements from arterial line samples are more accurate than any other method.

The pulse oximeter measures oxyhaemoglobin as a proportion of the total functional haemoglobin present—that is, only oxygenated + reduced haemoglobin. Co-oximeters also measure methaemoglobin and carboxyhaemoglobin, and these must be taken into account when comparing pulse oximeter and arterial line sample measurements. With samples from infants younger than 5 months, a variable is the unknown amount of oxygenated fetal haemoglobin present. Although this is a functional haemoglobin and is treated as such by the pulse oximeter, part of the fetal haemoglobin is falsely estimated as carboxyhaemoglobin by both co-oximeters. The proportions of fetal and adult haemoglobin, together with as yet undetermined factors, have been shown to affect the relation
between this measured carboxyhaemoglobin and true carboxyhaemoglobin.12 13 To overcome this problem comparisons of SaO2 in this study have been shown separately for infants above and below 5 months of age. After 5 months, fetal haemoglobin concentrations will be low.14

For all measurements a correction formula described by Brown15 and recommended by the manufacturers of the co-oximeters was used to provide values for functional SaO2. This formula is: functional SaO2 (%) = Fractional SaO2 × 100/100 – (carboxyhaemoglobin + methaemoglobin); fractional SaO2 = oxygenated haemoglobin/(oxygenated haemoglobin + reduced haemoglobin + carboxyhaemoglobin + methaemoglobin).

Results

Comparisons of SaO2P and arterial SaO2 were made on 65 occasions in 24 infants less than 5 months old, and on 27 occasions in 19 infants and children over 5 months of age. Fig 1 shows the results of all comparisons, and fig 2 those for infants and children over 5 months of age. The variability between the two values, measured as the mean (SD) absolute difference for all patients, was 2-6% (2-4). Twelve of 92 comparisons (13%) showed differences ≥5% (maximum 10%). For 19 infants over 5 months old the mean (SD) absolute differences of 27 comparisons was 1-8% (2-1) and only four (15%) showed absolute differences ≥5% (maximum 7%).

The mean actual (signed) difference for infants under 5 months old was +2-05%, and for those over 5 months +0-74% (in both instances the mean for the co-oximeter exceeded the mean for the pulse oximeter).

Fig 3 shows the pulse oximeter values plotted against arterial line PaO2 measurements made on 169 instances in 81 patients. Some of the variability shown in this figure may be due to a range of dissociation curves showing differing displacements to the left, typical in preterm babies with high concentrations of fetal haemoglobin. Comparisons of SaO2P and PaO2 in 46 instances when PaO2 was <6-67 kPa showed SaO2P <90% on 40 occasions,
while in 24 instances when PaO$_2$ was $\geq$13.3 kPa the SaO$_2$P was $\geq$98% on 22 occasions.

TcPO$_2$ and arterial line PaO$_2$ were compared on 60 instances in 23 infants (fig 4). The mean (SD) of the absolute differences between TcPO$_2$ and PaO$_2$ was 1.67 kPa (1.73). Ten of the 60 comparisons (on six different infants and four different machines) showed differences of $>2.67$ kPa. Three comparisons on three different infants showed differences $>5.33$ kPa (maximum 8.40 kPa).

Discussion

When care is taken to use only high quality arterial waveforms, pulse oximetry is a useful, non-invasive technique and represents a considerable advance in monitoring oxygenation in infants and children undergoing intensive care. Our experience is similar to that of others, but this study has highlighted the problem of measuring fetal oxygenated haemoglobin saturation in infants under 5 months old when validation of SaO$_2$P is seriously limited by the inaccuracy of the co-oximeters. After an empirical correction for the false measurement of a proportion of oxygenated fetal haemoglobin as carboxyhaemoglobin, the mean absolute difference between SaO$_2$P and SaO$_2$ was 2.6%. When comparisons were limited to an age at which fetal haemoglobin concentrations were low, the mean absolute difference was 1.8% and exceeded 4% in four of 27 comparisons.

Recently corrections according to the proportions of fetal haemoglobin have been published by Cornelissen et al.$^{13}$ and Ryan et al.$^{12}$ Nevertheless, we suggest that in future studies the base line measurement would be better obtained by using a non-spectrophotometric method of measuring arterial blood sample oxygen saturation—for example, from direct measurement of oxygen content.

Over the steep part of the dissociation curve, the most important part of the reading in ill patients with problems associated with tissue oxygenation or the uptake of oxygen from the lungs, the quantity of oxygen transported in the blood, and the availability of oxygen are more usefully reflected by oxygen saturation than by PaO$_2$. In the presence of fetal haemoglobin where the curve is shifted to the left, SaO$_2$ may be adequate at relatively low values of PaO$_2$, and increasing the PO$_2$ may not increase the unloading of oxygen to the tissues. With fetal haemoglobin present oxygen saturation may be 95% at a PaO$_2$ of 6-67 kPa. When the curve is shifted to the right, as in acidosis, with increasing temperature, or following transfusion of adult blood, optimal availability of oxygen to tissues may require relatively higher PaO$_2$ values. Under these circumstances a PaO$_2$ of 12.7 kPa may be required to provide 95% saturation, the saturation being only 70% at a PaO$_2$ of 6-67 kPa.

As far as clinically important hypoxaemia is concerned, analysis of our data shows that in 46 instances with a PaO$_2$ <6-67 kPa, oxygen saturation was <90% on 40 occasions. Figure 3 shows that the steep part of the curve begins at about 90%. We suggest that values less than this should be avoided.

The problem of avoiding hyperoxaemia when using SaO$_2$ rather than PaO$_2$ measurements is of major concern in neonatal intensive care because of the risk of retinopathy of prematurity$^{21,22}$; a large and possibly harmful change in PO$_2$ can occur in the presence of small or, in the case of 100%, zero change in saturation values. In this respect, TcPO$_2$ could provide a more reliable indicator of hyperoxaemia. In practice, however, the reliability of TcPO$_2$ measurement decreases as PaO$_2$ increases.$^{1,2,5,6}$ It was reassuring to note that using pulse oximetry in the 24 instances when PaO$_2$ was $\geq$100 mmHg, the SaO$_2$P was $\geq$98% on 22 occasions. Nevertheless, in the presence of additional inspired oxygen or CPAP, our data suggest that to avoid PaO$_2$ values of $>$13.2 kPa in newborn preterm infants true arterial oxygen saturation values of $>$95% should be avoided. In our data from patients over 5 months of age the co-oximeter exceeded the pulse oximeter by 2% or less in 85% of instances with a maximum of 7%. This relatively small quantity of information from older infants does suggest that pulse oximeter values of 90% should avoid hyperoxia. Nevertheless, more investigations into the calibration and validation of pulse oximeters for use in neonates are required. Without additional inspired oxygen,
SaO₂P values in healthy normal infants are often >98%, and this safety level of 91% is only relevant when additional oxygen is being given.

In practice it is vital that the operator appreciates the number of arterial pulse waveforms (or the time) over which the pulse oximeter is averaging saturation. Measurements of saturation averaged from over a long time period should theoretically decrease errors and dampen any short term oscillations in saturation. Long term average measurements will, however, provide accurate readings only if all of the waveforms are of adequate quality. Measurements made more often over short average times should avoid this possible source of error, and are probably a safer way of using this instrument. It is essential that the quality of all waveforms used to derive a particular saturation value are adequate. Fig 5 shows the kind of artefact that may result from inadequate waveforms. The monitor used in this present study provided a bouncing light display to indicate the quality of each waveform; we consider that this may be insufficient. A monitoring oscilloscope showing the quality of the waveforms on which the calculations were based would be useful. Alternatively, the analog output of the arterial waveform signals could be displayed on ECG monitor at the bedside.

Previous investigators have examined correlations between TcPO₂ and arterial line PO₂ measurements, and the lack of reliable comparisons by clinicians as distinct from research workers has been well documented. Fanconi et al reported a mean absolute difference of 0.93 kPa (range −1.87 to +6.53), with 10 of 108 values differing by >2.67 kPa, and Kraus et al reported that about 20% of values differed by more than 2.67 kPa. Similarly our data (fig 4) shows that 10 of 60 comparisons differed by more than 2.67 kPa, and three of 60 by >5.33 kPa, (maximum 8.40 kPa). Inexplicable and clinically relevant differences between TcPO₂ and PaO₂ measurements have been identified, and our study confirms that TcPO₂ measurements must be validated frequently, and that changes in inspired oxygen must not be based on TcPO₂ alone but always checked by blood gas analysis.

TcPO₂ measurements are known to become less reliable as patients get older, and they are unsuitable for older preterm infants with chronic lung disease who are receiving additional inspired oxygen. Pulse oximetry may be particularly valuable in this group.

Potential advantages of the pulse oximeter include the rapidity of achieving a measurement after the probe has been applied, the internal calibration, the lack of skin heating, and the fact that the characteristics of the arterial waveforms indicate whether the probe has been correctly applied. This last advantage is particularly important; TcPO₂ electrodes do not have this capacity.

The major disadvantage of the pulse oximeter is its susceptibility to artefacts caused by movement. This is probably inherent in the ‘transmission’ method and may be difficult to overcome. While the babies were crying, squirming, or having seizures, when SaO₂ may well fall, the monitor was unable to identify satisfactory arterial waveforms for the measurement of saturation. Fig 6 illustrates short lived episodes of true arterial hypoxaemia during short apnoeic pauses and periodic breathing in normal preterm and fullterm infants, which may

![Graph](https://i.imgur.com/9QX5yZ.png)

**Fig. 5** Recording of healthy fullterm infant aged 3 months in mode 2 of pulse oximeter; dip in oxygen saturation to almost 60% without an apnoeic pause is due to artefact.
produce many low saturation alarms. To overcome this problem it would be necessary to be able to measure the duration of the hypoxaemia, or track a breathing movement signal, so that episodes of hypoxaemia associated with short apnoic pauses and periodic breathing could be differentiated from persistent baseline hypoxaemia, or from hypoxaemia associated with prolonged apnoea. Other problems include the difficulty of being sure that the arterial waveforms are adequate and the relatively high cost of the probe; this may tempt nursing staff to use them on more than one patient, so increasing the risk of cross infection.

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