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APPARATUS

Accuracy of pulse oximetry in patients with low systemic vascular resistance

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Summary

In order to assess the accuracy of pulse oximeters in patients with septic shock, we compared 80 paired readings of oxygen saturations taken from pulse oximeters and oxygen saturations obtained from co-oximetry in patients receiving intensive therapy with indwelling pulmonary artery flotation catheters. Comparison between groups with low or normal systemic vascular resistance indices showed a small (1.4%) but significant ($p < 0.001$) underreading of the saturation from the pulse oximeter in the presence of a low systemic vascular resistance. With normal or high systemic vascular resistance pulse oximeter readings correlated well with co-oximetry. We hypothesise that the main cause of this underreading is because the pulse oximeter is sensing pulsatile venous flow due to the opening of arteriovenous channels in the skin in septic states.

Keywords *Measurement techniques*; pulse oximetry. *Complications*; septic shock.

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In normal volunteers, high skin blood flow may result in the reading from a pulse oximeter being inaccurate [1]. This may be relevant in clinical conditions when skin blood flow is increased, especially in septic shock, but also with regional or conduction anaesthesia or reperfusion following ischaemic injury. Pulse oximeters are calibrated by empirical observations in normal adults and all pulse oximeters assume that the only pulsatile absorbance between the light source and the photodetector is that of arterial blood [2]. This assumption may not be correct in clinical cases where significant venous pulsation is seen [3].

Manipulation of oxygen delivery and uptake have been shown to be important in the treatment of septic shock [4]. The present study was designed to examine if there was a significant difference between oxygen saturation as measured by pulse oximetry and the 'gold standard' of co-oximetry in a population of patients with low systemic vascular resistance as compared with a population with normal or high systemic vascular resistance.

Method

Following approval by the local ethics committee, 20 patients admitted to the general intensive therapy unit, who required arterial cannulation and placement of a pulmonary artery flotation catheter, were studied. All patients were monitored with finger probe pulse oximeters integral to the bedside monitors (Tramscope 12c, Marquette Electronics Inc., WI, USA). Pulse oximeter probes were placed by the nursing staff on the hand opposite to that containing the arterial line. Patients' arms were rested at their sides and there were no impedances to blood flow. Readings of oxygen saturation as displayed by the pulse oximeter (Spo_2) were taken only when the reading was stable, of adequate intensity for reliable readings and when the heart rate from the oximeter was within ± 3 beats of that recorded by the ECG.

The pulmonary artery flotation catheters used were Edwards Swan-Ganz® CCO/SVO_2 attached to a Vigilance® Continuous cardiac output monitor (Baxter Health Care Corporation, CA, USA). Readings of central venous and arterial blood pressure were taken from suitable central venous access and radial arterial cannulae connected to the bedside monitor, the signals being regularly zeroed and calibrated. Patient height was measured as supine length, weight being estimated from the last recorded reading in the patient's notes. Body surface area was calculated from a standard nomogram

and this figure used to index values obtained for cardiac output and systemic vascular resistance.

Paired samples of oxygen saturation data were obtained in the following way: following the performance of a full set of cardiac output studies, an arterial sample was taken anaerobically for blood gas analysis and a note was made of the reading on the bedside pulse oximeter. The sample was analysed in a combined blood gas analyser and co-oximeter (ABL system 620, Radiometer, Copenhagen) within 60 s of being taken. The reading of oxygen saturation is provided from the absorption spectra of the haemolysed sample. The calibration of the co-oximeter is checked daily by technical and quality assurance staff. In a small quality control study of 20 samples comparing pulse oximetry with co-oximetry on nonseptic intensive therapy patients, the system bias was −0.38% and the precision was 2.1%.

The pair of single readings was entered into a database along with the cardiovascular and blood-gas data for later analysis. Readings were separated by at least 6 h. One hundred sets of paired data were obtained and were divided into two groups based upon the systemic vascular resistance index (SVRI). A figure of 1000 dyne.s.cm^{-5}.m^{-2} was taken as the point at which the groups were separated, as below this figure patients are certainly pathologically vasodilated. Thus below 1000 dyne.s.cm^{-5}.m^{-2} patients were placed in the study or 'septic' group, at or above this figure, in the control group. If any doubt arose as to the validity of the reading, either due to technical or recording problems, the result was not used in the analysis.

Power studies suggested a figure of 85 paired sets of data to have a 90% chance of detecting a 1% difference. Statistical analysis included Kolmogorov–Smirnov (K-S) analysis for normal distribution and then Student's *t*-test, Mann–Whitney *U*-test or χ^2 test were used as appropriate. Levels of significance were set at $p = 0.05$.

Results

Readings were taken from 20 patients over 10 weeks of study. Table 1 illustrates the wide range of clinical conditions encountered. Seven readings from two patients were excluded due to problems with the monitoring systems and a further 13 readings were excluded because the recorded data were incomplete. Thirty-four of the remaining 80 paired samples fulfilled the criteria for the study group. Results are summarised in Table 2.

The groups are not significantly different in age, sex, weight or height. The mean SVRI was 604 (range 337–998) dyne.s.cm^{-5}.m^{-2} in the study group and 1385 (1010–5116) dyne.s.cm^{-5}.m^{-2} in the control group, while the mean cardiac indices were 5.8 (4.3–11.6) l.min^{-1}.m^{-2} and 3.4 (1.8–5.0) l.min^{-1}.m^{-2}, respectively. Kolmogorov–Smirnov analysis of the differences between pulse oximeter and co-oximeter readings gave $p = 0.78$, thus we assumed the difference to be normally distributed. Oxygen saturation as measured by the co-oximeter (Sa_{O_2}) was significantly higher than that measured by the pulse oximeter (Sp_{O_2}) in the study group by a mean 1.4% ($p < 0.001$), while there was no significant difference in the control group. Sa_{O_2} values (taken as 'true' oxygen saturation) were, on average, lower in the study group (94.6% vs. 96.3%, $p < 0.05$). Other differences between study and control groups were that study patients were likely to be more acidaemic (pH 7.35 vs. 7.40, $p < 0.05$) and were more likely to be receiving adrenaline (47% vs. 26%, $p < 0.05$) and noradrenaline (44% vs. 20%, $p < 0.05$). Levels of carboxyhaemoglobin and methaemoglobin were measured, but were insignificant (< 1%) on all samples.

Table 1 Patients on the intensive therapy unit needing pulmonary artery flotation catheters over the study period.

Patient	Diagnosis	Age	Sex
1	Pneumonia, respiratory failure	63	F
2	ESRF, sepsis	50	F
3	Pneumonia, respiratory failure	30	M
4	Faecal peritonitis	52	M
5	Cardiac arrest, ARF	51	F
6	Pneumonia, septic shock	71	M
7	Hepatic failure, pneumonia	42	M
8	AAA repair, pneumonia	73	M
9	ESRF, faecal peritonitis	50	M
10	AAA repair, ARF	57	M
11	ESRF, biliary peritonitis	49	M
12	Nephrectomy, septicaemia	73	M
13	Wegeners granulomatosis, ARF, pneumonia	46	M
14	AAA repair, ARF	70	M
15	Pneumonia, myocardial infarction, ARF	73	F
16	Cardiorespiratory failure	65	M
17	AAA repair	66	M
18	AAA repair	79	M
19	Hepatorenal failure	28	M
20	Cholecystitis, peritonitis	67	F

ESRF, end-stage renal failure; ARF, acute renal failure; AAA, abdominal aortic aneurysm.

Discussion

Pulse oximeters do not measure oxygen saturation. They measure the difference in absorption of two wavelengths of light as it passes through a blood-filled tissue and rely on 'look-up' tables derived from data obtained from normal volunteers to give a reading of saturation. They aim to estimate arterial saturation by analysing the portion of the absorption that is continuously changing, representing arterial pulsation. Pulse oximeters are prone to several

Table 2 Comparison of cardiac calculations, oximetry values, blood gas parameters and inotropes between the two groups. Presented as mean (SD) or median [range].

	Low SVRI group	Normal/high SVRI group	Significance
n	34	46	
Age; years	51 [28–73]	63 [28–79]	ns
Male:female	28 : 6	37 : 9	ns
Weight; kg	74.44 (10.14)	78.82 (14.20)	ns
Height; cm	169.67 (7.26)	172.53 (8.93)	ns
SVRI; dyne.s.cm^{-5}.m^{-2}	604 [337–998]	1385 [1010–5116]	$p < 0.001$
Cardiac index; l.min^{-1}.m^{-2}	5.8 [4.3–11.6]	3.4 [1.8–5.0]	$p < 0.001$
Spo_2; %	93 [88–97]	96 [90–99]	$p < 0.05$
Sao_2; %	94.7 [88.9–98.2]	95.3 [92.2–98.7]	ns
Sao_2–Spo_2	1.4 (1.1)	0.5 (2.0)	$p < 0.001$
Patient temperature; °C	38.4 (1.1)	38.1 (1.1)	ns
Sample pH	7.36 (0.08)	7.40 (0.09)	$p < 0.05$
Sample po_2; kPa	10.86 [7.9–14.4]	11.77 [8.7–18.8]	ns
Sample pco_2; kPa	5.4 (1.0)	5.0 (0.8)	ns
Sample base excess; μmol.l^{-1}	−2.9 (6.0)	−1.3 (5.3)	ns
Bilirubin; μmol.l^{-1}	28 [14–93]	56 [14–139]	ns
Dopamine	28/34	36/46	ns
Dobutamine	12/34	21/46	ns
Adrenaline	16/34	12/46	$p < 0.05$
Noradrenaline	15/34	9/46	$p < 0.05$

well known and extensively researched problems producing unreliable readings, including extremes of oxygen saturations [5], significant carboxyhaemoglobinaemia, methaemoglobinaemia and the presence of dyes and pigments [6].

Co-oximetry is accepted as the 'gold-standard' in studies on pulse oximetry. Following haemolysis of heparinised blood, differential absorption spectra are obtained using several (often four or more) wavelengths of light to measure all forms of haemoglobin. Saturation is expressed as the percentage of oxygenated haemoglobin over total haemoglobin, giving a reading of true arterial saturation. Bias, or systematic error, and precision, or random error, are as would be expected within this system; bias means that Sao_2 consistently reads 0.38% higher than Spo_2, and precision of 2.1% correlates well with the often quoted 2% for pulse oximeters as a whole.

Other factors which could result in differences between Sao_2 and Spo_2 include temperature, pH and presence of bilirubin. Only pH was significantly different between the two groups but this is taken into account during the original calibration of pulse oximeters and is included in the 'look up' table conversion factors [7].

Our observed lowering of the estimate of arterial saturation displayed by the pulse oximeter in patients with low systemic vascular resistance correlates with the findings of previous studies where venous pulsation is increased [1, 3]. The low systemic vascular resistances we observed were caused by septicaemia complicated on occasion by other disease (i.e. hepatic failure) or therapy (prostacyclin used with continuous veno-venous haemofiltration). In septicaemia, the cardiac output may increase by a factor of two or more and is usually accompanied by marked peripheral vasodilatation. Arterioles are maximally dilated, possibly resulting in pulsatility being transmitted to the capillary beds. Arteriovenous shunts may also open resulting in pulsatility of the venous flow. As the pulse oximeter is analysing the pulsatile component of the absorption it will therefore incorporate a systematic error giving a lower saturation than the true arterial value.

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