COMPARISON OF PULSE OXIMETERS: EFFECTS OF VASOCONSTRICTION AND VENOUS ENGORGEMENT

C. J. WILKINS, M. MOORES AND C. D. HANNING

Pulse oximetry is an emerging standard in clinical monitoring [1]. Since the first description of the technique [2], many instruments have been introduced commercially. All work on the principle of absorption spectroscopy, but there are differences in probe design and sampling frequency, and in the constants and algorithms used to process the data [3]. These differences may affect instrument performance.

Pulse oximetry relies upon the presence of a pulsating vascular bed. It is likely that signal detection would be impaired in the presence of vasoconstriction or venous congestion, conditions which may occur readily in clinical practice. Previous evaluations of pulse oximeters [3–8] have not specifically examined this problem.

The aim of this study was to evaluate five commercially available pulse oximeters as clinical monitors of oxygenation in the presence of these factors.

SUBJECTS AND METHODS

Ethics Committee approval was obtained and all subjects gave informed consent to be included in the study, which was performed in two parts.

Part I

The instruments tested in this part were the Ohmeda 3700, the Novametrix 500 (Novametrix Medical Systems Inc.), the Criticare Systems Inc. 501, and the Accusat (Datascope Corporation). Ten healthy volunteers (eight male) aged 25–40 (mean 29) yr took part. All had satisfactory tests of ulnar collateral blood flow. Subjects rested comfortably on a couch and standard finger probes from the test instruments were applied in random order to the fingers of one hand; the thumb was not used. Cards marked with each of the 24 possible combinations of probes and fingers were placed in unmarked envelopes and one drawn for each subject (thus not all combinations were used). A Biox III (Ohmeda) applied to the subject’s ear was used for safety purposes and to ensure uniformity of \( \Delta S_AO_2 \) changes between periods of low oxygen breathing. The outputs of the instruments were fed to a chart recorder calibrated according to the manufacturer’s instructions.

SUMMARY

The effects of cold-induced vasoconstriction and venous occlusion on the detection of induced hypoxaemia by four pulse oximeters were examined in 10 volunteers. In three further subjects vasoconstriction was maintained until at least one instrument failed to detect the induced hypoxaemia. Time taken to detect hypoxaemia was increased for all instruments to between two and three times the instrument’s own control value for both vasoconstriction and venous engorgement (\( P < 0.01 \)). There was highly significant variation in detected minimum saturation between the instruments (\( P < 0.001 \)). One instrument failed to detect the full extent of desaturation under the experimental conditions and was more likely to fail completely to detect desaturation than the other test instruments when influenced by vasoconstriction (\( P < 0.05 \)). Significant impairment in the performance of all the instruments tested occurred in the presence of normal pulse signals. The duration of detected reductions in oxygen saturation was not significantly affected.
Control readings

Two episodes of hypoxaemia were induced by the inhalation of 10% oxygen in nitrogen from pre-filled Douglas bags, via a closely fitting non-rebreathing mask after a 5-min period for baseline observations. Each episode lasted 2 min, but was terminated if the Biox III indicated that arterial oxygen saturation (SaO₂) had decreased to 79% or less. Subjects breathed room air for 3 min between episodes of hypoxaemia.

Venous congestion

A standard sphygmomanometer cuff applied to the upper arm was inflated to 40 mm Hg to induce venous congestion. After 5 min, two further episodes of hypoxaemia were induced as before.

Vasoconstriction

The subject’s forearm was placed into a water-filled plastic envelope cooled overnight in a domestic refrigerator (fig. 1). The oximeter probes were allowed to protrude from the envelope to avoid their direct cooling. After 5 min, two further episodes of hypoxaemia were induced as before.

The nature of any alarm or warning signal from the instruments was noted at all stages of the experiment and was acted upon as appropriate.

Analysis of data

The second of the two hypoxaemic episodes for each of the three experimental conditions (control, venous congestion and vasoconstriction) was analysed. Data for minimum SaO₂, detection time (time from start of breathing 10% oxygen to the first downward deflection on the SaO₂ trace) and duration of the reduction in SaO₂ to less than resting values were obtained from the permanent record.

No assumptions were made regarding the distribution of the data, and all statistical tests used were for non-parametric data. Data from the Biox III (ear probe) were not included in the analysis because it was not the purpose of the study to compare the performance of ear and finger probes.

For each of the categories of data, the variation between the three sets of experimental conditions and between the four test instruments was analysed by the Kruskal–Wallis one-way analysis of variance using Oxstat. Wilcoxon signed rank tests were used to test for differences between the experimental conditions for each instrument. It was possible to use a test for paired data because each instrument acted as its own control within each subject. Differences between instruments were examined using the same test, but the results of these are reported only where the performance
of one instrument was significantly different from that of all the others.

**Part II**

The instruments tested in this part were the same as those used in part I, except that the Lifesat 1600 (Physio-control Corporation) was substituted for the Criticare Systems Inc. 501 (CSI). A Biox III with ear probe was again used for safety purposes. Three subjects were studied, each on four separate occasions, and finger probes from the test instruments were applied to a different finger for each experimental session. Thus each of the probes was tested on each finger of each subject.

As before, hypoxaemia was induced by the inhalation of 10% oxygen in nitrogen for periods of 2 min separated by 3-min intervals. After two such episodes, the cold envelope was applied as before. The sequence of hypoxaemic episodes was continued for 1 h or until all the instruments failed, whichever was the shorter period. Failure was defined as an inability to detect a reduction in $\text{Sa}_\text{O}_2$ of greater than 5% from resting values. As before, any indicated alarm was recorded and acted upon as appropriate.

**Analysis of data**

The number of instrument failures was recorded for each instrument and the data analysed by a Chi-squared test with Yates' correction for small numbers.

**RESULTS**

**Part I**

All instruments detected a decrease in $\text{Sa}_\text{O}_2$ for all episodes of hypoxaemia and continued to display normal pulse signals throughout the experiment. There were no significant differences between instruments in resting $\text{Sa}_\text{O}_2$ or in minimum $\text{Sa}_\text{O}_2$ between experimental conditions (as indicated by the Biox III).

**Detection time (table I)**

Analysis of variance revealed highly significant differences between the three sets of experimental conditions ($P < 0.001$).

**Control conditions.** There were no important differences between the test instruments.

**Venous congestion.** The mean detection time of all the test instruments was increased significantly to between two and three times each instrument's own control value ($P < 0.01$) (table I). In the most extreme case, detection time was increased from a control value of 120 s to 405 s.

**Vasoconstriction.** The mean detection time of all the instruments was increased significantly, usually to between two and three times the instrument's control value ($P < 0.01$). In one case the detection time was increased from a control value of 95 s to 275 s.

There were no significant differences in detection times amongst the test instruments for both vasoconstriction and venous congestion.

**TABLE I. Detection time (s) for induced hypoxaemia for each instrument for the three experimental conditions (mean (range)), n = 10. **P < 0.01; ***P < 0.001 for comparisons with own control (Wilcoxon signed rank test)**

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Control</th>
<th>Venous congestion</th>
<th>Vasoconstriction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ohmeda 3700</td>
<td>58.4 (14–120)</td>
<td>159.7 (48–405)**</td>
<td>120.3 (53–225)**</td>
</tr>
<tr>
<td>C.S.I.</td>
<td>66.3 (14–130)</td>
<td>152.5 (35–300)**</td>
<td>140.6 (40–290)**</td>
</tr>
<tr>
<td>Novametrix</td>
<td>57.3 (14–95)</td>
<td>122.0 (35–155)**</td>
<td>121.1 (67–275)**</td>
</tr>
<tr>
<td>Accusat</td>
<td>52.9 (10–90)</td>
<td>129.5 (30–230)**</td>
<td>95.2 (42–210)**</td>
</tr>
</tbody>
</table>

**TABLE II. Minimum $\text{Sa}_\text{O}_2$ (%) during breathing 10% oxygen for each set of experimental conditions (mean (range)), n = 10. *P < 0.05 for comparison with own control (Wilcoxon signed rank test)**

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Control</th>
<th>Venous congestion</th>
<th>Vasoconstriction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biox III</td>
<td>83.0 (79–86)</td>
<td>81.4 (79–85)</td>
<td>81.1 (79–84)</td>
</tr>
<tr>
<td>Ohmeda 3700</td>
<td>78.9 (72–82)</td>
<td>82.9 (74–91)</td>
<td>77.9 (72–83)</td>
</tr>
<tr>
<td>C.S.I.</td>
<td>81.6 (72–84)</td>
<td>85.0 (79–95)*</td>
<td>80.9 (77–84)*</td>
</tr>
<tr>
<td>Novametrix</td>
<td>82.6 (78–88)</td>
<td>86.4 (82–90)*</td>
<td>87.8 (80–96)*</td>
</tr>
<tr>
<td>Accusat</td>
<td>82.7 (78–85)</td>
<td>83.9 (78–89)</td>
<td>83.1 (78–89)</td>
</tr>
</tbody>
</table>
**TABLE III.** Number of instrument failures and warnings of failure for each set of experimental conditions in part II. *P < 0.05 for comparison with Ohmeda 3700 (Chi-squared test)

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Control</th>
<th>Vasoconstriction</th>
<th>Warning given</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ohmeda 3700</td>
<td>0</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Accusat</td>
<td>0</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Lifestat 1600</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Novametrix 500</td>
<td>0</td>
<td>8*</td>
<td>2</td>
</tr>
</tbody>
</table>

Fig. 2. The effect of vasoconstriction (two consecutive episodes of hypoxaemia). Note delay in response and failure of Novametrix 500.

*Minimum Sa\(_{\text{O}_2}\)* (table II)

There were no significant differences between the three sets of experimental conditions, but there was marked variability between the test instruments (*P < 0.001*).

**Control.** The minimum Sa\(_{\text{O}_2}\) was significantly less for the Ohmeda 3700 than for all the other instruments (*P < 0.05*). There were no other differences between the instruments (table II).

**Venous congestion.** The minimum Sa\(_{\text{O}_2}\) for the Novametrix 500 was significantly greater than its own control value (*P < 0.05*).

**Vasoconstriction.** The minimum Sa\(_{\text{O}_2}\) for the Novametrix 500 was significantly greater than its own control value (*P < 0.01*). The value of the Ohmeda 3700 was significantly less than those of all the other instruments (*P < 0.05*).

**Duration of detection reduction in Sa\(_{\text{O}_2}\)**

There were no significant differences between instruments or between experimental conditions.
There were 12 separate experimental sessions and the maximum number of possible failures for each instrument was therefore 12 (table III).

An example of instrument failure is seen in figure 2. Because the numbers are small, only the figures for the Biox 3700 and the Novametrix 500 were subjected to statistical analysis by chi-squared test (with Yates' modification for small numbers). The difference was significant at $P < 0.05$.

DISCUSSION

Analysis of the performance of pulse oximeters is complex. We identified three elements which we thought likely to be affected by a reduction in limb perfusion, namely detection time, duration of desaturation and minimum $Sa_O$. Other authors [3, 8] have been concerned with the absolute accuracy of pulse oximeters and have measured $Sa_O$ directly to provide the standard. The purpose of the present study was to examine the influence of venous congestion and vasoconstriction on the performance of the test instruments, rather than to determine their absolute performance, and we have no data for absolute $Sa_O$ or $Pa_O$. We used an oximeter (Biox III) applied to the subject's ear to ensure uniformity of desaturation between hypoxic tests (not as a measure of true $Sa_O$) and have made comparisons for each instrument under the influence of venous congestion and vasoconstriction with its own control value.

Uniformity in the degree of desaturation was achieved, there being no differences in minimum $Sa_O$ for the three sets of conditions. It is not possible to state which of the instruments was reflecting the true $Sa_O$ most accurately, and thus the meaning of comparisons of $Sa_O$ between instruments would be questionable. We have, therefore, not made such comparisons, but have commented only on the variability amongst the group of instruments, except where one instrument was different from all of the others.

Vasoconstriction occurs commonly in patients undergoing surgery and intensive care and may be caused by cold, hypovolaemia, drugs, inadequate anaesthesia or other factors. We have attempted to mimic these conditions in the laboratory by using a surface cooling technique. The degree of vasoconstriction produced was not quantified, but all subjects found the cold stimulus unpleasant or even painful and all displayed marked digital pallor. The oximeter probes protruded from the envelope and were not subjected to direct cooling, although some indirect cooling must have occurred. None of the probes tested incorporates a heating element. This model may not exactly represent the conditions found in clinical practice, particularly if there are concomitant changes in cardiac output, but it was simple and non-invasive.

Venous congestion may be produced by awkward positioning of the patient, by venous pooling caused by gravity or by increased central venous pressure. It may not always be obvious to the anaesthetist if the affected part is obscured by surgical drapes.

This study demonstrated the significant effect of vasoconstriction and venous congestion on the performance of pulse oximeters.

Detection time was increased consistently (figs 2 and 3). A mean delay of 150 s in detecting a hypoxic insult is clearly undesirable. The most extreme delay was 405 s. These delays occurred at
a time when the instruments were displaying normal pulse signals.

The increase in detection time probably has two components. The principal component, the reduced blood flow to the fingers, is physiological. The second is related to the algorithm of the instrument: the displayed value of \( SaO_2 \) is derived from many discrete values which are weighted according to their quality and then averaged. A reduced pulse volume may influence the weighting placed on each discrete measurement of \( SaO_2 \) and thus on the displayed value. It is interesting that the Novametrix 500 continues to display a value for \( SaO_2 \) even if only one valid pulse (peak–trough pair) has been detected during the previous 25 s, and this may explain its sensitivity to reduced perfusion. This information was not available for the other instruments.

Most of the instruments gave similar values of minimum \( SaO_2 \) for the three conditions (control, venous congestion and vasoconstriction). This is in keeping with previous work [9] in which one instrument gave reliable data in spite of the infusion of vasopressor drugs. However, one instrument (the Novametrix 500) failed to detect the maximum extent of the reductions in \( SaO_2 \) when influenced by venous congestion or vasoconstriction. This tendency was confirmed in part II of the study, when the same instrument failed more often than any of the others. The same instrument has been shown by others to possess marked variability in performance [8]. More disturbingly, this instrument gave no alarm signal on six of the eight occasions when it failed. The Accusat and Lifestat 1600 both gave clear warning of poor quality signals when they failed. The incidence of instrument failure did not appear to be influenced by site of probe placement.

It is possible that an individual instrument may perform badly because it is faulty or because of sampling error in the face of variation between instruments. It has been reported that two instruments of the same type gave widely differing \( SaO_2 \) values when connected to the same patient [10]. It is impossible for the user to know that an instrument is inaccurate, because no form of calibration is required.

New versions of several of the instruments tested (including the Novametrix 500) were introduced after we began the study and may possess different performance characteristics.

We conclude that:

1. Venous congestion and cold-induced vasoconstriction considerably increased the detection time for induced hypoxaemia.
2. The presence of a normal pulse signal does not always imply that perfusion is adequate for the rapid detection of hypoxaemia.
3. There may be important differences between instruments in their susceptibility to vasoconstriction and venous congestion.
4. There was marked variation between instruments in the minimum saturation detected.

REFERENCES