Validation of pulse oximetry for monitoring of hypoxaemic episodes in the late postoperative period

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Summary
We monitored 12 patients undergoing major abdominal surgery using a pulse oximeter (Nellcor N-200) and a transcutaneous oxygen tension monitor (TINA, Radiometer A/S) on the second or third night after operation. Of the shortest hypoxaemic episodes measured with the pulse oximeter (\( \leq 30 \) s duration), 78% also occurred in the transcutaneous oxygen tension measurement. Episodes of longer duration (\( \geq 1 \) min duration on the pulse oximeter) were, in 95% of cases, reflected in the transcutaneous oxygen tension measurement also. Thus postoperative episodic desaturations lasting \( \geq 1 \) min are at least 95% likely to be a real phenomenon. (Br. J. Anaesth. 1997; 78: 86–87)

Key words

Late postoperative episodic hypoxaemia is frequent during the second to fourth nights after major surgery, and may be related to development of cardiac complications. Postoperative episodic hypoxaemia has been verified previously by pulse oximetry. However, it is possible that some of the hypoxaemic episodes may have been motion artefacts rather than hypoxaemic events. Measurement of transcutaneous oxygen tension using a heated Clark electrode is not sensitive to motion, and this method is therefore a suitable reference for validating pulse oximetry monitoring of episodic hypoxaemia.

The aim of the study was to validate pulse oximetry for monitoring of episodic hypoxaemia after major surgery by simultaneous and continuous monitoring of transcutaneous oxygen tension (TcO2) and transcutaneous oxygen saturation (SpO2).

Methods and results
We studied 12 patients (six men, median age 66 (range 44–84) yr, weight 70 (55–85) kg, height 170 (160–180) cm) in the ward after major abdominal surgery. Patients were studied on the second or third night after laparotomy during the hours of 23:00–07:00. Eight patients underwent colonic resection, two gastric procedures and two operations for adhesions. The anaesthetic technique was inhalation (enflurane, 11 patients) or i.v. (midazolam/low-dose fentanyl, one patient). Nine of the patients received additional extradural anaesthesia during operation. Supplementary oxygen therapy was not given during the study night. In nine patients postoperative pain therapy was performed with continuous extradural infusion of bupivacaine 10 mg h\(^{-1}\) and morphine 4.8 mg day\(^{-1}\), and in three patients postoperative pain was treated with morphine i.m. on demand.

Arterial oxygen saturation (SpO2) was measured continuously, with a Nellcor N-200 pulse oximeter (Nellcor Inc., Pleasanton, CA, USA) using an adhesive finger probe, and transcutaneous oxygen tension (TcO2) was measured with a TINA transcutaneous oxygen tension monitor (Radiometer A/S, Copenhagen, Denmark). The TcO2 electrode was heated to 43°C and placed distally at the volar side of the same arm as the SpO2 probe. Data for SpO2 and TcO2 were printed simultaneously at bedside on an analogue recorder.

An episodic hypoxaemic event in the SpO2 tracing was defined as a sudden reduction in SpO2 of \( \geq 5\% \) from baseline and occurring within 2 min. In the TcO2 tracing a hypoxaemic event was defined as a sudden reduction in TcO2 of more than 0.3 kPa and occurring within 2 min. Events were considered to be coherent if the TcO2 event was delayed by not more than 30 s from the SpO2 event.

The study was approved by the local Ethics Committee and all patients gave written informed consent. Group data are given as median (range). For statistical analyses we used Fisher’s exact test. \( P<0.05 \) was considered statistically significant.

Duration of surgery was 175 (75–290) min. For the group of 12 patients, median SpO2 was 93 (91–96) % and TcO2 was 4.3 (3.8–5.9) kPa. A total of 481 hypoxaemic episodes were recorded on the pulse oximeter (table 1), of which 424 (88%) episodes were transmitted to the TcO2 tracing. There were no instances of episodes in the TcO2 curve not reflected in the SpO2 curve; 95% of the hypoxaemic
Pulse oximetry for monitoring hypoxaemic episodes

Table 1 Occurrence of late postoperative hypoxaemic episodes assessed by pulse oximetry and transcutaneous measurement (TcO2) in relation to duration of the desaturation episode

<table>
<thead>
<tr>
<th>Duration of episode in pulse oximetry measurement</th>
<th>Total No. of episodes</th>
<th>Reflected in TcO2 (no. of episodes)</th>
<th>Not reflected in TcO2 (no. of episodes)</th>
<th>P (Fisher’s test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;30 s</td>
<td>30</td>
<td>7 (23%)</td>
<td>23 (77%)</td>
<td>0.00001</td>
</tr>
<tr>
<td>30 s</td>
<td>161</td>
<td>142 (88%)</td>
<td>19 (12%)</td>
<td>&lt;0.00005</td>
</tr>
<tr>
<td>Total &lt; 1 min</td>
<td>191</td>
<td>149 (78%)</td>
<td>42 (22%)</td>
<td>&lt;0.00005</td>
</tr>
<tr>
<td>1 min</td>
<td>195</td>
<td>183 (94%)</td>
<td>12 (6%)</td>
<td>&lt;0.00005</td>
</tr>
<tr>
<td>1 min 30 s</td>
<td>46</td>
<td>44 (96%)</td>
<td>2 (4%)</td>
<td>&lt;0.00005</td>
</tr>
<tr>
<td>2 min</td>
<td>13</td>
<td>13 (100%)</td>
<td>0 (0%)</td>
<td>&lt;0.00005</td>
</tr>
<tr>
<td>&gt;2 min</td>
<td>36</td>
<td>35 (97%)</td>
<td>1 (3%)</td>
<td>&lt;0.00005</td>
</tr>
<tr>
<td>Total ≥1 min</td>
<td>290</td>
<td>275 (93%)</td>
<td>15 (5%)</td>
<td>&lt;0.00005</td>
</tr>
<tr>
<td>Total No. of episodes</td>
<td>481</td>
<td>424 (88%)</td>
<td>57 (12%)</td>
<td>&lt;0.00005</td>
</tr>
</tbody>
</table>

episodes lasting ≥1 min also occurred on the TcO2 curve, whereas 5% did not (difference between rates = 90%, 95% confidence limits of difference between rates = 78–101%).

Comment

The principal finding of our study was that episodic oxygen desaturations were reflected in transcutaneous oxygen tension measurements in 95% of cases if the episodes lasted 1 min or more. It is presumed that if a hypoxaemic episode occurs in both SpO2 and TcO2 tracings it is “real”, and if it occurs only in the SpO2 curve it may (or may not) be an artefact. Thus at least 95% of episodes lasting ≥1 min were real.

Pulse oximetry needs arterial pulsation to measure oxygen saturation. Thus in circumstances when decreased pulsation occurs, for example hypothermia, hypotension and vasoconstriction decrease the ability of the oximeter to measure arterial oxygen saturation. It is not likely, however, that this was the case for the shorter lasting episodes in this study. Movement artefacts may be a significant source of error in pulse oximetry measurements. A previous study evaluating four oximeters in an experimental setting with vibration found no false hypoxaemic episodes with the Nellcor N-200 oximeter (when the SpO2 signal was read in conjunction with the ECG signal for data verification) whereas the Ohmeda 3700, the Simed S100 and the Datex Satellite Plus reported spurious hypoxaemic episodes, that is movement artefacts.

The relationship between transcutaneous and arterial oxygen tension depends on skin temperature, skin thickness, chosen place for measurement and correct preparation of the skin. Measurement of transcutaneous oxygen tension, however, is not dependent on motion. In adults, values for transcutaneous oxygen tension are normally much lower than arterial values. Thus in our study the median TcO2 was only 4.3 kPa. This method is therefore not suitable for measuring exact values for arterial oxygen tension, but sudden changes in arterial oxygen tension are reflected in transcutaneous measurements. However, there is the question of response time for the TcO2 technique. Previous studies have found that with acute changes in arterial oxygen tension, the TcO2 signal may be dampened or delayed because of the time for transport of oxygen through the skin. Based on various assumptions and the results of earlier experimental studies, this transport time has been estimated to be in the order of 30 s. In our study, when examining the actual curves for SpO2 and TcO2 changes resembled more of an “all or nothing” phenomenon; either the SpO2 event was reflected almost simultaneously in the TcO2 curve or the SpO2 event did not occur. Our limit of 30 s between the SpO2 and TcO2 events was arbitrary, as no episodes were reflected in the TcO2 curve with a delay of more than 30 s. It is therefore more likely that the discrepancy between some of the shortest SpO2 events and the TcO2 tracing was caused by motion artefacts (thus an error in the SpO2 measurement), rather than the TcO2 not reporting actual hypoxaemic events.

In summary, SpO2 desaturations lasting ≥1 min, assessed using pulse oximetry in the late postoperative period, represented, with at least 95% certainty, an actual hypoxaemic episode. Short-lasting (<30 s) episodic hypoxaemia, as assessed by pulse oximetry, may in 22% of instances or less, not reflect an actual hypoxaemic event.

Acknowledgements

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References