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Influence of pulse oximeter lower alarm limit on the incidence of hypoxaemia in the recovery room†

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Summary
In a prospective, randomized study, we have investigated the effects of two arbitrary pulse oximeter lower alarm limit (LAL) settings (90% = group 90, \( n = 320 \) and \( 85% = \text{group 85, } n = 327 \)) on the incidence of hypoxaemia in the recovery room. In group 90, we calculated the theoretical effect of elimination of transient episodes of low pulse oximeter oxyhaemoglobin saturation (\( \text{SpO}_2 \)) by introducing a time delay between the onset of the alarm condition and triggering of the alarm. When only hypoxaemic episodes lasting more than 1 min were included, \( \text{SpO}_2 \leq 90\% \) occurred in 11% of patients in group 90 and in 20% in group 85 (relative risk (RR) 1.84, confidence interval (CI) 1.26–2.68; \( P < 0.01 \)). Hypoxaemia \( \leq 85\% \) occurred in 2% of patients in group 90 and in 6% in group 85 (RR 3.10, CI 1.32–7.28; \( P < 0.01 \)). In group 90, 1007 alarms (33% false) occurred, whereas in group 85, 395 alarms (28% false) occurred. Introducing a theoretical delay of 15 s in group 90 between crossing the alarm threshold and triggering the alarm would have reduced the number of alarms by 60%. The results of the study suggest that decreasing the alarm limit in an attempt to reduce frequent false alarms may lead to an increase in more relevant episodes of hypoxaemia and setting the LAL at 85% cannot be recommended routinely. The effects of LAL on the incidence and duration of hypoxaemia have not been evaluated. In a prospective, randomized study, we have investigated the effects of two arbitrary LAL settings (\( \text{SpO}_2 \) 90% and 85%) on the incidence and duration of hypoxaemia in the recovery room. As an alternative to a LAL of 85%, the effect of maintaining the LAL at 90% and elimination of short-lasting alarms by introducing an alarm delay (a delay between \( \text{SpO}_2 \) crossing the alarm threshold and triggering of the audible alarm) was calculated.

Patients and methods
We studied consecutive patients who entered the recovery room of a regional (non-teaching) hospital after general or regional anaesthesia over a 1-month period. The study was approved by the hospital must perform multiple tasks. An average frequency of pulse oximeter alarms in the recovery room of once every 8 min has been reported. Another study showed that only 10% of alarms were clinically relevant. Frequent clinically insignificant alarms have the potential to frustrate personnel, distract attention from more important tasks and can even result in unnecessary therapeutic interventions, neglect of true alarms or permanent disabling of the alarm.

It is obvious that decreasing the pulse oximeter lower alarm limit (LAL) should reduce the number of alarms. However, it is uncertain if this would reduce its efficacy as a monitor of impending severe hypoxaemia. For sudden severe events there might be little difference. For example, when pulse oximeter oxyhaemoglobin saturation (\( \text{SpO}_2 \)) decreases rapidly after the onset of respiratory arrest, the triggering of an alarm by an \( \text{SpO}_2 \) of 90% or 85% may be only marginally different. None the less, many clinicians argue that decreasing the alarm limit might increase the incidence of hypoxaemia. The effects of LAL on the incidence and duration of hypoxaemia have not been evaluated. In a prospective, randomized study, we have investigated the effects of two arbitrary LAL settings (\( \text{SpO}_2 \) 90% and 85%) on the incidence and duration of hypoxaemia in the recovery room. As an alternative to a LAL of 85%, the effect of maintaining the LAL at 90% and elimination of short-lasting alarms by introducing an alarm delay (a delay between \( \text{SpO}_2 \) crossing the alarm threshold and triggering of the audible alarm) was calculated.

Key words
Pulse oximeter alarm setting and hypoxaemia

Ethics Committee and each patient gave informed consent to participate in the study. There were no exclusion criteria for age, ASA status or type of operation. Patients who required postoperative mechanical ventilation were excluded from the study. Institutional guidelines for anaesthetic and post-anesthetic care did not change during the study. No bedside observer was present to verify the validity of the plethysmographic signal or to record the actions by the recovery room nurse.

Pulse Oximetry Monitoring

At the beginning of each day the LAL of each pulse oximeter (Criticare 504, Criticare Systems, Inc., Waukesha, WI, USA) was set randomly at either 90% (group 90) or 85% (group 85). The averaging epoch of each pulse oximeter was set at 3 s. A minimum value of the signal is required by the pulse oximeter to calculate \( \text{SpO}_2 \), or else a loss of signal message is displayed. The display of this device shows the plethysmographic waveform, \( \text{SpO}_2 \), heart rate and alarm limits. A loud continuous audible alarm sounds when \( \text{SpO}_2 \) decreases to or below the level of the LAL. It is possible to temporarily disable the alarm sound for a maximum of 2 min. Patients arriving from the operating room were allocated to the next available bed. Monitoring started immediately after arrival of the patient in the recovery room and was continued until the time of discharge. The sensor of the pulse oximeter was applied to a finger of the hand opposite to the arterial pressure cuff. Supplementary oxygen was administered by a nasal cannula to all patients during the initial postoperative period.

Analysis of Alarms, Artefacts and Hypoxaemia

Heart rate, \( \text{SpO}_2 \), and alarm status, together with time of day, were stored every 5 s in the memory of the pulse oximeter and transferred at the end of the day to a personal computer. Data for the first and last 3 min of monitoring were excluded to eliminate artefacts resulting from positioning and removal of the probe.

Episodes with \( \text{SpO}_2 \leq 90\% \) were classified as hypoxaemia or artefact. An episode was classified as artefact if one or more of the following criteria were met:
- transient outlier: a single datum point (5 s resolution) differing by more than 4% from the surrounding data;
- loss of signal (as indicated by the pulse oximeter) occurring either 0–20 s before or 0–20 s after the episode of low \( \text{SpO}_2 \);
- a step change in \( \text{SpO}_2 \) of more than 10% in 5 s; and
- in an attempt to increase further the detection of artefacts, all other episodes with \( \text{SpO}_2 \leq 90\% \) were inspected visually by the author (A. T. R-L., blinded for the group from which the data originated). If there was simultaneous jitter of \( \text{SpO}_2 \) and heart rate, a movement artefact was suspected and data were classified as artefact.

The number of episodes of hypoxaemia for the two thresholds (\( \text{SpO}_2 \leq 90\% \) and \( \leq 85\% \)) was counted. Episodes where \( \text{SpO}_2 \) decreased to values \( \leq 90\% \) were differentiated from those in which \( \text{SpO}_2 \) remained above 85% (see example A in fig. 1) and from those where it decreased to less than 85% (example B in fig. 1). The time difference between \( \text{SpO}_2 \) crossing the 90% threshold and the 85% threshold (interval \( 90\% - 85\% \)) was calculated (example B in fig. 1).

The incidence of hypoxaemia (proportion of patients with at least one episode of hypoxaemia lasting \( \geq 1\) min) for three thresholds (\( \text{SpO}_2 \leq 90\%， \leq 85\% \) and \( \leq 80\% \)) was determined. For each patient the cumulative duration of hypoxaemia (only episodes \( \geq 1\) min) for each of the thresholds was calculated.

Alarms were classified as either true positive (generated by hypoxaemia) or false positive (generated by artefact). In group 90 the effect on the number of alarms of introducing various theoretical delays (0–60 s, 5-s steps) between the onset of the alarm condition and triggering of the alarm was calculated.

Data are presented as counts and percentiles. Differences between the two groups were analysed using chi-square and Mann–Whitney tests. \( P<0.05 \) was considered significant. In group 85 the incidence of hypoxaemia was also expressed as relative risk (RR) compared with group 90. The confidence interval (CI) for relative risk was calculated with the Mantel–Haenszel chi-square statistic.

Results

During the 1-month period, we studied 647 patients; 320 patients were allocated to group 90 and 327 patients to group 85. Table 1 presents descriptive data for patients, anaesthesia and post-anaesthetic care. There were no differences between groups in...
Table 1 Data on patients, anaesthesia and post-anaesthetic care. Data are counts or median (10–90th percentiles). Group 90 = lower alarm limit of the pulse oximeter set at 90%, group 85 = lower alarm limit of the pulse oximeter set at 85%.

<table>
<thead>
<tr>
<th>Criteria for hypoxaemia</th>
<th>Group 90 (n = 320)</th>
<th>Group 85 (n = 327)</th>
<th>RR (CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>SpO₂ &lt;90%</td>
<td>34 (11)</td>
<td>64 (20)</td>
<td>1.84 (1.26–2.69)</td>
<td>0.001</td>
</tr>
<tr>
<td>SpO₂ &lt;85%</td>
<td>6 (2)</td>
<td>19 (6)</td>
<td>3.10 (1.32–7.28)</td>
<td>0.009</td>
</tr>
<tr>
<td>SpO₂ &lt;80%</td>
<td>1 (0.3)</td>
<td>6 (2)</td>
<td>6.13 (0.92–37.5)</td>
<td>0.06</td>
</tr>
</tbody>
</table>

The number of episodes of desaturation ≤90% (without applying a criterion for the minimum duration of the episode) was 674 in group 90 and 938 in group 85. SpO₂ decreased to or below 85% (see example B in fig. 1) in 104 desaturations in group 90 and in 197 desaturations in group 85. Table 3 shows the interval 90–85%, the time required for oxyhaemoglobin saturation to decrease from 90% to 85%. The majority of desaturations less than 85% had an interval 90–85% less than 20 s in both groups. A longer interval 90–85% (≥20 s) occurred significantly more often in group 85 (P<0.001).

In group 90, 1007 alarms (674 true, 333 false) occurred, whereas in group 85, 395 alarms (285 true, 110 false) occurred. Figure 2 shows the number of alarms in group 90 that would have occurred with various delays from 0 to 60 s between the onset of the alarm condition (SpO₂ = 90%) and triggering of the alarm. As 60% of alarms lasted 15 s or less, introducing a 15-s delay in group 90 would reduce the number of alarms by the same amount as changing the LAL from 90% to 85% (group 90 with 15 s delay—398 alarms (71% true, 29% false) and group 85 with no delay—395 alarms (72% true, 28% false)).

Table 3 Time required for oxyhaemoglobin saturation to decrease from 90% to 85%. Group 90 = lower alarm limit of the pulse oximeter set at 90%, group 85 = lower alarm limit of the pulse oximeter set at 85%, SpO₂ = oxyhaemoglobin saturation, interval 90–85% = time difference between SpO₂ crossing the 90% threshold and the 85% threshold.

<table>
<thead>
<tr>
<th>No. of desaturations</th>
<th>Group 90</th>
<th>Group 85</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SpO₂ &lt;85% lasting less than 1 min</td>
<td>≤85% lasting more than 1 min</td>
</tr>
<tr>
<td>Interval 90–85%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–15 s</td>
<td>70</td>
<td>5</td>
</tr>
<tr>
<td>20–55 s</td>
<td>17</td>
<td>0</td>
</tr>
<tr>
<td>≥1 min</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Total No. of desaturations &lt;85%</td>
<td>95</td>
<td>9</td>
</tr>
</tbody>
</table>
Discussion

Lowering the LAL from 90% to 85% is an effective means of reducing the number of audible alarms in the recovery room. However, with the LAL set at 85%, the incidence of hypoxaemia lasting more than 1 min increased. Although the clinical relevance of this finding is unknown, most clinicians would consider episodes of hypoxaemia of less than 85% lasting greater than 1 min potentially harmful. Therefore, based on the results of this study, setting the LAL at 85% cannot be recommended routinely.

Postoperative hypoxaemia has been implicated as a contributing factor for myocardial ischaemia, wound infection and cognitive dysfunction. However, a large prospective study failed to demonstrate significant effects of pulse oximetry on outcome. To increase the clinical relevance of a possible effect of the chosen LAL, we studied episodes with a minimum duration of at least 1 min. The six times higher incidence of severe hypoxaemia ≤80% in group 85 was not statistically significant (P=0.06). To exclude a type II error (a difference exists, but was not found in this study) a calculated group size of more than 2000 patients would be needed to achieve 80% power to detect a 50% reduction. This suggests that groups larger than the 320 patients we studied would have shown a statistically significant increased incidence of hypoxaemia ≤80% in group 85 also.

An alarm should initiate an intervention to reverse the ongoing desaturation. In addition, the underlying cause of the desaturation must be treated. In group 85 the majority of desaturations less than 90% resolved without triggering an alarm. However, the total number of these desaturations (in which the lowest \(S_\text{PO}_2\) remained >85%, see example A in fig. 1) was 40% higher than in group 90. This suggests that the alarms resulted in therapeutic actions that corrected the underlying cause and prevented subsequent hypoxaemia.

In this study the speed of desaturation (as measured by the interval\(^{90-85\%}\)) was rapid in the majority of patients in both groups. When \(S_\text{PO}_2\) decreases rapidly, changing the LAL from 90% to 85% delays the onset of the alarm only marginally. None the less, the incidence of desaturation less than 85% was higher in group 85. As can be seen in table 3 the speed of desaturation was more gradual in those patients who became hypoxaemic for more than 1 min in group 85. The interval\(^{90-85\%}\) appears to be a crucial period in the institution of therapy to prevent prolonged hypoxaemia when desaturation is more gradual. This suggests that an early alarm is more effective in preventing longer and more severe desaturations than an alarm that sounds when \(S_\text{PO}_2\) has decreased to less than 85%.

A high frequency of false alarms could be a legitimate argument to reduce the LAL with the aim of increasing specificity. Unfortunately, many technical problems (dislocation of the sensor, peripheral vasoconstriction, patient movement) produce artefactual rapid \(S_\text{PO}_2\) decreases to values of 80–90%. Previous studies have shown that as many as 50% of pulse oximeter alarms may be false. In group 90 an average of one artefact per patient was present and 30% of the total number of alarms were false. We cannot exclude the possibility that, despite our artefact rejection algorithm and inspection of individual \(S_\text{PO}_2\) data, several artefacts remained undetected. However, the majority of episodes with \(S_\text{PO}_2\) ≤90% were transients which were excluded from calculations of hypoxaemia by the requirement of a minimum duration of 1 min. The criterion of longer than 1 min duration appears to be a valid cut-off point for the detection of true desaturation. Stausholm and colleagues found that 95% of low \(S_\text{PO}_2\) episodes for more than 1 min were reflected in decreases of transcutaneous oxygen tension measurements. Recent improvements in signal extraction technology may decrease the impact of motion artefact on the quality of the pulse oximeter signal and reduce the frequency of artefacts even further.

Monitors with a low frequency of false alarms should increase the acceptance by recovery personnel of a higher LAL setting.

Most manufacturers of pulse oximeters give the user the option to average several beats to prevent jitter of the display and unnecessary triggering of the alarm by transients. The benefit of averaging a larger number of beats is the reduction of random variability and diminished susceptibility to transients. As a consequence there is a temporal delay between actual and displayed \(S_\text{PO}_2\) and delaying triggered of the alarm. Introducing a time delay between the detection of a low \(S_\text{PO}_2\) and triggering of the audible alarm is an alternative way of reducing the frequency of alarms. Makivirta and Koski found that doubling the pre-alarm delay from 5 to 10 s reduced the mean alarm rate by 26%. In our study a time delay of 15 s between the occurrence of the alarm condition and triggering of the audible alarm would have provided a similar reduction in the number of alarms as a decrease of the LAL from 90% to 85%. One advantage of introducing a time delay as opposed to lowering the LAL could be that hypoxaemia is detected earlier in patients whose \(S_\text{PO}_2\) decreases more gradually. For example, in group 85, for 27 of the 40 desaturations (≤85%, ≥1 min) the alarm would have been triggered earlier if an LAL of 90% and a 15 s delay of the alarm had been used. A second advantage might be improved attention to longer lasting episodes of mild hypoxaemia with \(S_\text{PO}_2\) values of 85–90%. In contrast, an alarm delay might result in later detection of severe hypoxaemia if the rate of desaturation is very fast. Because the majority of desaturations had an interval\(^{90-85\%}\) of less than 20 s, detection of hypoxaemia and institution of appropriate treatment would have been delayed by several seconds. This potential negative effect would have to balanced against the advantage of having a more specific alarm as a result of elimination of the majority of false alarms.

Uncertainty concerning the optimal LAL has to be acknowledged. To estimate the best cut-off point for a specific test a receiver operator characteristic curve (ROC) is often used. This curve visualizes the trade-off between sensitivity and specificity. The sensitivity and specificity of individual \(S_\text{PO}_2\) data points can be assessed (for example, by comparison
with co-oximetry of arterial blood samples). However, it is impossible to calculate “sensitivity” or “specificity” for a postoperative pulse oximeter monitoring session. The number of false negative episodes (hypoxemia present, no $\text{Sp}_\text{O}_2$ alarm) is unknown and it is illogical to count the number of true negative episodes (no hypoxemia, no alarm) because, by definition, each alarm is preceded by a true negative episode. The only variable that we could calculate with some confidence (based on the rejection algorithm) was the positive predictive value (number of true alarms divided by the total number of alarms). The positive predictive value was approximately 70% in both groups although the frequency of alarms was three-fold higher in group 90. Therefore, this value cannot be used to determine the optimal LAL.

In conclusion, the results suggest that recovery room personnel rely on $\text{Sp}_\text{O}_2$ alarms to identify and treat hypoxemia rather than observation of the patient and the pulse oximeter $\text{Sp}_\text{O}_2$ display. Decreasing the alarm limit in an attempt to reduce frequent false alarms may lead to an increase in clinically relevant hypoxaemia. Maintaining the LAL at 90% and instituting a 15 s time delay before triggering the audible alarm is an equally effective approach to reduce the frequency of alarms that might not hamper rapid detection of hypoxaemia.

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**References**


