Influence of motivation of care providers on the incidence of postoperative hypoxaemia in the recovery room†

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Summary
We have studied the influence of motivation of care providers on the incidence and duration of postoperative hypoxaemia in the recovery room. In a prospective, switch-back designed cohort study, we have compared the incidence of low pulse oximeter saturation values ($SpO_2$) during pre-intervention, intervention and post-intervention phases. Low $SpO_2$ values were classified as either hypoxaemia ($SpO_2 \leq 90\%$, minimum duration 1 min) or artefact. Pulse oximetry trend data from 1350 patients, 450 in each group, were analysed. During the intervention phase, motivation was increased by adding an explicit instruction to prevent and treat low $SpO_2$ values and making personnel aware that they were being studied (Hawthorne effect). The incidence of hypoxaemia decreased significantly from 17.8% during the pre-intervention phase to 11.6% during the intervention phase (relative risk (RR) 0.65, 95 % confidence interval (CI) 0.47–0.90; $P<0.01$). The incidence of severe hypoxaemia ($SpO_2 \leq 85\%$, 1 min) decreased from 7.8% to 3.3% (RR 0.43, CI 0.24–0.76; $P<0.01$). The number of patients who had severe hypoxaemia for more than 5 min decreased from 13 to 1 (RR 0.08, CI 0.02–0.36; $P<0.01$). In the post-intervention period, the incidence of hypoxaemia returned to pre-intervention values. The results of this study suggest that motivation of care providers to prevent and treat low $SpO_2$ is an important determinant of postoperative hypoxaemia in the recovery room. (Br. J. Anaesth. 1996;77:453–457)

Key words

Patients and methods
The study was performed in a 650-bed community hospital. Six full-time anaesthetists (minimum experience 6 yr post-graduation) are on staff. There are 10 operating rooms and a nine-bed recovery room. Neurosurgical procedures or cardiac surgery are not performed. Neither the types of case nor the anaesthetic technique changed over the three study phases. Patients undergoing operations under general, spinal or extradural anaesthesia, who were

In a recent study the average frequency of pulse oximeter alarms ($SpO_2 \leq 90\%$) being sounded in the recovery room was once every 8 min, with 77% of alarms being false. We suspected that a very high incidence of false alarms could encourage recovery room nurses to accept low $SpO_2$ values as “normal” or “inevitable” and decrease their motivation to treat hypoxaemia.

This prospective cohort study was designed to test if motivation of care providers plays a role in preventing hypoxaemia. The study had a switch-back design (pre-intervention, intervention, post-intervention). Care providers (recovery room nurses and anaesthetists) were unaware of $SpO_2$ data collection in the pre-intervention period. In the intervention period an explicit instruction to maintain $SpO_2 > 90\%$ accompanied each patient, and care providers were purposely made aware that they were being studied. The null hypothesis was that the intervention would not change the incidence or duration of hypoxaemia. The post-intervention period served to ascertain if any possible change in the incidence of hypoxaemia occurring during phase 2 persisted after the incentives to prevent and treat low $SpO_2$ values were removed.
monitored for at least 10 min in the recovery room, were eligible to enter the study. There were no exclusion criteria based on age or clinical condition of the patient.

For each of the three study phases, $Sp_O_2$ data from 450 consecutive patients were retrieved from pulse oximeter memory (3–4 weeks for each phase).

During phase 1, anaesthetists and nurses were unaware of $Sp_O_2$ data collection. The default lower alarm limit of the pulse oximeter was 90%. There were no specific written guidelines regarding treatment of postoperative hypoxaemia. This study phase was designed to determine the incidence of hypoxaemia during routine postoperative care. After analysis of these data, care providers were informed of the incidence and duration of postoperative hypoxaemia, and the study design was explained. No attempt was made to link hypoxaemia to individual anaesthetists or nurses.

Phase 2 of the study, the intervention period, was conducted immediately after completion of phase 1. An explicit written instruction to maintain $Sp_O_2 > 90\%$ accompanied each patient, but no specific instructions regarding the methods of maintaining normoxaemia were given. Anaesthetists and nurses were now aware that pulse oximetry data from the next 450 patients would be retrieved and analysed. In this way a positive bias on performance, resulting from the knowledge of being studied (Hawthorne effect) was purposely introduced. After completion of phase 2, data were analysed and the results presented to care providers. From that time, the written instruction to prevent hypoxaemia was omitted from the postoperative orders.

Phase 3 started 2 months after completion of phase 2. There was no written instruction to prevent hypoxaemia in the postoperative orders. Anaesthetists and nurses were again unaware that pulse oximetry data were collected.

The hospital Ethics Committee advised that only patients in phase 2 were required to give informed consent, as data obtained in phases 1 and 3 consisted of anonymous pulse oximeter trend data and no randomization was involved.

RECOVERY ROOM AND DATA ACQUISITION

One nurse takes care of a maximum of two postoperative patients at a time. If necessary, postoperative mechanical ventilation is available for two patients. Supplementary oxygen is not administered during transport from the operating theatre to the recovery room. In the recovery room supplementary oxygen is administered by nasal cannula to all patients during the initial postoperative period. The minimum duration of stay for an individual patient is ordered by the responsible anaesthetist at the time of arrival.

Routine pulse oximetry monitoring had been established 4 yr previously. A Criticare 504 pulse oximeter with system software version 3.1 (Criticare Systems, Inc., Waukesha, WI, USA) was used for monitoring $Sp_O_2$. This monitor shows a plethysmographic waveform and does not apply autoscaling. Signal averaging has a default value of 12 s. Monitoring started immediately after arrival of the patient and was continued until the time of discharge. The probe of the pulse oximeter was applied to a finger of the hand opposite to the arterial pressure cuff. The $Sp_O_2$ lower alarm limit used at the start of monitoring was 90%. This level was the default lower alarm limit on all oximeters for the past 4 yr. It was chosen to reflect a threshold that would undisputedly suggest a need for therapy. A loud audible alarm sounds when $Sp_O_2$ decreases below the lower alarm limit or when the pulse oximeter is unable to measure $Sp_O_2$ because of a low signal-to-noise ratio or disconnection of the probe from the finger.

$Sp_O_2$ and pulse frequency, together with time, were stored every 5 s in the memory of the pulse oximeter. At the end of the day, when the last patient had been discharged to the ward and personnel had left the recovery room, these data were transferred from pulse oximeter memory to a personal computer for off-line analysis.

DATA ANALYSIS

Individual $Sp_O_2$ trend arrays were validated before analysis. Episodes with $Sp_O_2 \leq 90\%$ were classified as either hypoxaemia or artefact. The algorithm used to exclude episodes with artefact used the following criteria: (1) transient outlier: a single datum point (5-s resolution) changed more than 4% from the surrounding data; (2) low signal-to-noise ratio: loss of signal occurred 20 s before until 20 s after the episode of low $Sp_O_2$; and (3) decrease in $Sp_O_2$ of more than 10% within 5 s. Two arbitrary levels of hypoxaemia were defined and for each patient the cumulative duration of hypoxaemia was calculated. Hypoxaemia was defined as episodes of $Sp_O_2 \leq 90\%$ lasting at least 1 min. Severe hypoxaemia was considered to be present when $Sp_O_2$ values were at or below 85% for at least 1 min.

In each study phase the following were determined: number of patients who were hypoxaemic at any moment during pulse oximetry monitoring in the recovery room; number of patients who arrived hypoxaemic in the recovery room; and number of hypoxaemic patients, after excluding data recorded during the first 3 min of monitoring in the recovery room.

STATISTICAL ANALYSIS

Data are presented as counts and percentiles. Differences between phases were analysed using chi-square and Kruskal–Wallis tests. $P<0.05$ was considered significant. Group size was calculated to achieve 85% power to detect a 50% decrease in the incidence of hypoxaemia. The incidence of hypoxaemia during phase 2 was expressed as relative risk to phase 1. The confidence interval for relative risk was calculated using the Mantel–Haenszel chi-square statistic.

RESULTS

Patient data, anaesthesia data and duration of postoperative monitoring are shown in table 1. Median duration of stay in the recovery room in phase 2 was 6 min longer ($P<0.01$).

The audible $Sp_O_2$ alarm was triggered 766 times during phase 1 (mean 2.1 times per hour of monitoring), 411 times during phase 2 (1.2 per hour) and
588 times during phase 3 (1.5 per hour). In 53% of patients, the \( \text{SpO}_2 \) signal was lost transiently at some time during monitoring. There were no differences in the incidence or duration of “no signal conditions” (median duration 40 s) between the three phases of the study. The incidence of artefacts was reduced in phase 2 (table 2).

Table 2 shows the number of hypoxaemic patients in each phase. In phase 1, 17.8% of patients experienced hypoxaemia. During phase 2 it decreased to 11.6% (relative risk (RR) 0.65, 95% confidence interval (CI) 0.47–0.90; \( P < 0.01 \)). More severe hypoxaemia (\( \text{SpO}_2 \leq 85\% \)) was noted at some time in 7.8% of patients during phase 1; in phase 2 it decreased to 3.3% (RR 0.43, CI 0.24–0.76; \( P < 0.01 \)). The reduction in postoperative hypoxaemia during phase 2 was not persistent: the incidence of hypoxaemia in phase 3 was not significantly different from phase 1. The number of patients who arrived hypoxaemic in the recovery room was less in phase 2 compared with phase 1. When data recorded during the first 3 min of monitoring were excluded from analysis, the reduction in hypoxaemia in phase 2 compared with phase 1 remained.

Median duration of hypoxaemia was not different between the three study groups. However, the 90th percentile of the duration of severe hypoxaemia was 19 min in phase 1, 3.5 min in phase 2 and 21 min phase 3 (fig. 1). Severe hypoxaemia lasting more than 5 min occurred in 13 patients during phase 1 compared with in one patient during phase 2, and in six patients during phase 3 (RR 0.08, CI 0.02–0.36; \( P < 0.01 \)).

**Discussion**

We have found that an explicit instruction to maintain \( \text{SpO}_2 > 90\% \), combined with the knowledge of being studied, may reduce the incidence of postoperative hypoxaemia by as much as 50%. The recurrence of hypoxaemia 4 months after intervention further supports the hypothesis that motivational factors of care providers is an important determinant of the incidence of postoperative hypoxaemia.
hypoxaemia. With this sample size the study lacked sufficient statistical power to determine if some residual effect of the intervention was present in phase 3, and it is possible that a larger study population might have shown such an effect. However, a larger study population would have required a longer data collection period, during which time changes in recovery room personnel could not be avoided.

Subjects who are aware of being studied behave differently, and usually performance increases (Hawthorne effect). In several studies on the incidence of postoperative hypoxaemia, the investigators used bedside observers to record the actions of personnel and to verify that a valid pulse oximeter signal was present. However, the presence of an observer might introduce a Hawthorne effect and the possibility exists that studies involving direct observation of personnel do not reflect the actual situation during routine postoperative care. The aim of the pre-intervention phase was to document existing practices. Therefore, anaesthetists and nurses were not informed that pulse oximetry data were collected and no observers were present to document patient care. Although this study design precluded matching of pulse oximetry data from individual patients with specific therapeutic interventions, the absence of observers made it possible to document real practice and to introduce a positive study bias during the intervention. No specific instructions regarding particular methods to prevent or treat hypoxaemia were given. In this way we tried to ensure that any change in the incidence of hypoxaemia was the result of an action initiated by an individual anaesthetist or nurse, for example administration of reversal drugs or stimulation of the patient, rather than adherence to a specific set of changed postoperative guidelines.

The positive study bias introduced during phase 2 not only influenced postoperative care, but also altered intraoperative care. Anaesthetists were aware of the ongoing data collection and there was a substantial reduction in the incidence of hypoxaemia on arrival in the recovery room. Anaesthetists may have modified their anaesthetic technique, for example they may have administered a lower dose of opioid which may decrease the incidence of postoperative respiratory depression. Another possible explanation for the reduction in hypoxaemia on arrival might be delayed transport from the operating theatre to the recovery room, with treatment of early hypoxaemia in the operating room rather than in the recovery room. The occurrence of hypoxaemia during transport from the operating to the recovery room has been reported previously. The results of this study showed that increased motivation to prevent hypoxaemia reduced the incidence of hypoxaemia during transportation from 3% to less than 1%.

When the contribution of hypoxaemia on arrival in the recovery room is included in the calculation of the incidence of hypoxaemia during the entire recovery period, it could obscure the possible influence of actions initiated by recovery room nurses. Therefore, we have recalculated the incidence of hypoxaemia after excluding data from the first 3 min after arrival. During these 3 min the effect of supplementary oxygen or stimulation of the patient on \( S_{PO_2} \) or both, would be expected to become apparent. The reduction in hypoxaemia remained after exclusion of data recorded during the first minutes after arrival, suggesting that both anaesthetists and nurses contributed to the decreased incidence of hypoxaemia.

When pulse oximeter data are not validated by verifying the presence of a plethysmographic signal of sufficient quality, the incidence of hypoxaemia is overestimated because of inclusion of episodes with erroneously low \( S_{PO_2} \) values. In one study 9% of pulse oximetry data obtained during emergence from anaesthesia appeared to be invalid. In other studies, up to 75% of low \( S_{PO_2} \) episodes during postoperative care in the recovery room and ICU were false. In this study, we used computer analysis of the individual trend data arrays in an attempt to eliminate artefacts. Extra attention to correct application of the finger probe or successful reduction in patient movement in phase 2 decreased the incidence of artefacts by 50%. None the less, the possibility remains that part of the reduction in hypoxaemia during phase 2 may be attributed to a decrease in the number of non-rejected artefacts; however, if application of the artefact algorithms before analysis of hypoxaemia would be insufficient to eliminate all artefacts (and a large number of artefacts remained), the result would be to dilute the apparent effect of the intervention. Most \( S_{PO_2} \) artefacts in the recovery room are of short duration, and are ignored when a minimum duration criterion is in effect. Pan and Gravenstein found that excluding discrepant data less than 12 s decreased the frequency of artefacts by 63% and less than 30 s by 93%. In this study we chose to exclude low \( S_{PO_2} \) episodes lasting less than 60 s in order to reduce the influence of artefacts and to focus attention on clinically more relevant episodes of hypoxaemia.

When used as a physiological monitor, the pulse oximeter should direct the attention of personnel to patients who are at risk for prolonged hypoxaemia. Severe hypoxaemia lasting more than 5 min occurred significantly less often during phase 2, suggesting that patients who were at risk for prolonged severe hypoxaemia gained specifically from the increased attention to early recognition and treatment of hypoxaemia. We can only speculate why pulse oximetry monitoring did not always result in timely interventions to prevent prolonged hypoxaemia during phase 1. Pulse oximetry has often been introduced in the recovery room without clear guidelines on the type of interventions that are required when low \( S_{PO_2} \) values are present. The frequent occurrence of low \( S_{PO_2} \) values without a direct negative effect on recovery outcome may have resulted in complacency. A second explanation might be that some of the low \( S_{PO_2} \) values that reflect true hypoxaemia are incorrectly interpreted as artefact. This study cannot differentiate between the relative contribution of the Hawthorne effect and the impact of the explicit instruction to prevent desaturation. However, in several studies that have shown a reduction in postoperative hypoxaemia by pulse oximetry, personnel were observed continuously and motivated explicitly to reduce hypoxaemia.

In conclusion, the results of this study suggest that motivation of care providers to prevent and treat low \( S_{PO_2} \) values is an important determinant of the incidence of postoperative hypoxaemia in the recovery room.
References