POSTOPERATIVE HYPOXAEMIA: COMPARISON OF EXTRADURAL, I.M. AND PATIENT-CONTROLLED OPIOID ANALGESIA

R. G. WHEATLEY, I. D. SOMERVILLE, D. J. SAPSFORD AND J. G. JONES

SUMMARY

Arterial oxygen saturation (SaO₂) was analysed continuously before and for 24 h after lower abdominal surgery in 30 patients breathing air using one of three postoperative analgesic regimens: i.v. diamorphine using a patient-controlled analgesia system (PCAS), extradural diamorphine or i.m. morphine. Hypoxaemia was defined as SaO₂ < 94% for more than 6 min h⁻¹. Before operation there was no difference between the three analgesia groups assessed by the duration when SaO₂ was less than 94%. After operation the pattern of SaO₂ vs time distribution was either stable, with little variation from hour to hour with no hypoxaemia, or unstable with large variation with 30% of patients hypoxaemic. Thus three patterns of SaO₂ distribution were seen in the postoperative period: stable without hypoxaemia (4/10 PCAS, 0/10 extradural, and 1/10 i.m. patients), unstable without hypoxaemia (4/10 PCAS, 5/10 extradural and 7/10 i.m. patients) and unstable with prolonged nocturnal periods with SaO₂ < 94% for a mean of 17.7 min h⁻¹, 95% confidence limits (CL) 10–25 min h⁻¹, (2/10 PCAS, 2/10 i.m. and 5/10 extradural patients). Before operation, the unstable group with hypoxaemia spent longer at < 94% SaO₂ (mean 4.8 min h⁻¹, 95% CL 1.0–8.6 min h⁻¹) than the stable group (mean 0.4 min h⁻¹, 95% CL 0.17–0.61 min h⁻¹) and this was a predictor of postoperative hypoxaemia. Hypoxaemia occurred in all analgesia groups, but extradural diamorphine tended to cause longer periods. Some patients at risk of postoperative hypoxaemia may be predicted by preoperative monitoring of SaO₂ although extradural diamorphine boluses were associated with hypoxaemia in patients with normal preoperative values.

KEY WORDS

Analgesia: postoperative. Analgesics: extradural diamorphine, i.m. morphine, patient-controlled analgesia. Hypoxaemia: pulse oximetry

Opioid analgesia is a well recognized cause of ventilatory depression [1–5], but the frequency of this complication, even with conventional methods of administration, is disputed [6]. This dispute has arisen because the respiratory effects of analgesic regimens have often been based on intermittent observations of ventilatory rate or PaCO₂. For example, a study of 195 patients receiving intrathecal or extradural morphine [7] following Caesarean section revealed an incidence of ventilatory depression—defined as a breathing rate of less than 11 b.p.m.—as 1% in both groups. However, we have previously shown that a slow rate of ventilation was an unreliable and late sign of impaired postoperative ventilatory control being correlated with hypoxaemia in only one of 32 hypoxaemic patients [2]. In the same study an analysis of continuous measurements of oxygen saturation (SaO₂) showed that episodes of hypoxaemia (SaO₂ < 85%) occurred in 10 of 16 sleeping patients receiving continuous i.v. infusions of morphine in the postoperative period.

The aim of this study was to use continuous pulse oximetry to examine the effect on SaO₂ of three different postoperative analgesia regimens: diamorphine via a patient-controlled analgesic...
system (PCAS), diamorphine via the extradural route, and conventional intermittent i.m. analgesia. In contrast with earlier work [2, 8] in which i.v. morphine was used in older patients undergoing upper abdominal or hip surgery, we studied a younger group of patients having lower abdominal surgery.

PATIENTS AND METHODS

Patient selection

Patients gave informed consent to the study, which was approved by the Hospital Ethics Committee. The study comprised two parts: Part I was a reference study and Part II was the postoperative study.

The aim of Part I was to establish reference data for the distribution of SaO₂ in young healthy patients studied overnight. Nine patients were included in this part of the study.

Part II comprised 30 similar patients, scheduled for general anaesthesia and lower abdominal surgery, studied before and for a 24-h period after surgery. The 30 female patients studied in Part II were of low anaesthetic risk (ASA grade I or II), aged 20–70 yr. Their current therapy, smoking habits, weight and height were recorded.

Oxygen saturation measurements

Continuous monitoring of SaO₂ was carried out using a Nellcor N-100 pulse oximeter which was interfaced with an Opus, IBM-compatible microcomputer [9]. The oximeter sensor was attached to the patient’s great toe. Recordings of SaO₂ were made every 10 s and stored by the computer so that 360 samples were obtained every 1 h. Over a 24-h period, approximately 8000 recordings of SaO₂ were made and the computer was programmed to analyse and display this large amount of data in three ways:

(i) Percentage and distribution of recorded SaO₂. The values recorded during the whole observation period were displayed as percentages within a range of SaO₂. For example:

Total number of oximeter readings 8240
Values < 85% saturation 1 (0%)
Values > 85% < 90% saturation 2 (0%)
Values ≥ 90% < 94% saturation 62 (0.8%)
Values ≥ 94% saturation 8175 (99.2%)

This is not a very convenient method for reviewing trends during a monitoring period, so a graphic representation was developed.

(ii) Graphic representation of the pattern of SaO₂ distribution. The computer was programmed to produce a curve of SaO₂ distribution vs percentage of each hour (fig. 1) similar to that described previously [8]. An array of curves was produced, “stacked” to show the whole study period hour by hour as a compressed array of curves [9]. This is shown for the nine subjects in Part I of the study (fig. 2). Each curve was analysed to determine the median value of SaO₂. The Kolmo-

![Fig. 1. Method of displaying SaO₂ distribution during each epoch. In this example SaO₂ is sampled every 10 s and 300 samples are plotted. The most frequent SaO₂ value was 97%, 40% of the epoch being spent at that saturation.](image-url)
gorov–Smirnoff test was used to calculate the significance of changes between curves obtained in each epoch.

(iii) Hypoxaemic episodes. A significant episode of severe hypoxaemia was defined as $S_{a}O_2 < 85\%$ for longer than 20 s. The duration of any such episodes and their timing were noted for each observation period.

Procedure

The nine patients in Part I were studied overnight for a minimum period of 9 h.

The 30 patients in Part II, the main study group, were monitored for 1–11 h before administration of an oral benzodiazepine for premedication and for a 24-h period after operation. These patients were anaesthetized with a standardized general anaesthetic sequence of propofol 2 mg kg$^{-1}$, vecuronium 0.1 mg kg$^{-1}$, nitrous oxide and enflurane. Twenty patients had intraoperative analgesia provided by 0.5 % bupivacaine 15–20 ml after the insertion of a lumbar extradural catheter. Another 10 patients who did not have extradural analgesia received intraoperative i.v. morphine 5–10 mg. On completion of surgery and when spontaneous ventilation had been re-established, the trachea was extubated and the patient transferred to the recovery room breathing 40% oxygen from a Ventimask for 30–40 min. The patients were allocated to three groups of 10. The 20 patients who had received extradural analgesia were allocated randomly to groups A and B; the non-extradural patients were group C.

Group A patients self-administered i.v. diamorphine at a maximum rate of 1 mg every 20 min using a Graseby Patient Controlled Analgesia system (PCAS). All the patients studied were able to use the PCAS within 1 h of the end of surgery.

Group B patients received extradural diamorphine in doses of 3.6 mg in saline 9 ml administered by the anaesthetist or senior nursing
TABLE I. Demographic data (mean values (range)). * Derived from morphine dose

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PCAS</td>
<td>Extradural</td>
<td>I.m.</td>
</tr>
<tr>
<td>(n = 10)</td>
<td></td>
<td>(n = 10)</td>
<td>(n = 10)</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>40.2 (28–51)</td>
<td>43.2 (35–52)</td>
<td>42.2 (18–68)</td>
</tr>
<tr>
<td>Wt h⁻¹ index (kg m⁻¹)</td>
<td>21.1 (17.6–28)</td>
<td>21.12 (16–31)</td>
<td>18.12 (15.2–21)</td>
</tr>
<tr>
<td>Diamorphine total dose (mg/24 h)</td>
<td>27.3 (11.0–46.0)</td>
<td>14.4 (7.2–18.0)</td>
<td>26.1 (20–36.3)*</td>
</tr>
</tbody>
</table>

staff as requested by the patient. This was repeated as necessary during the 24-h period. The total dose used was recorded.

Group C patients were those without a lumbar extradural catheter who had received i.v. morphine during surgery. After being made comfortable in the recovery area with further i.v. morphine they were returned to the ward where they received morphine 7.5–10 mg i.m. on a 4-hourly, as required, basis.

Following the patient’s return to a general ward, \( S_{ao} \) was monitored for 24 h with the oximeter probe attached to the great toe. All the recordings were made with the patients breathing room air unless the \( S_{ao} \) decreased persistently to less than 85\%, in which case oxygen by facemask was administered to the patient.

Postoperative measurements

Visual analogue pain scores were obtained 4-hourly when the patient was awake using a 10-cm visual analogue scale [10]. Any side effects such as nausea, vomiting, itching and the necessity for bladder catheterization were noted also.

RESULTS

Part I: Reference study

The mean age of patients in this group was 38.4 yr (range 24–58 yr) and wt h⁻¹ was 15.9 (range 14–19.4). The arrays of \( S_{ao} \) vs time are shown in figure 2. Each patient showed a narrow distribution of \( S_{ao} \) which in subjects Nos 1, 7 and 9 showed a small but consistent decrease in \( S_{ao} \) during the night and recovery towards the morning. The remaining subjects showed a stable pattern of distribution with similar overlapping peaked curves for each 1 h, the majority of \( S_{ao} \) values occurring in the range 96–100\%. The median \( S_{ao} \) of the nine patients was 98\%, with less than 1\% of the values less than 94\% (\( S_{ao} < 94 \% \) for 0.6 min h⁻¹). Although some of these curves in different epochs showed statistically significant shifts in distribution, the largest shift in the median value was 2\%.

Part II: Postoperative study

The demographic data of the three treatment groups are shown in table I. Surgery and anaesthesia were uncomplicated in all 30 patients. No significant differences were found with respect to age, degree of obesity or duration of surgery between the three groups. Age and wt h⁻¹ did not differ from the patients in the Part I study. The dose of diamorphine used in the extradural group was significantly less than the other two treatment groups. The doses of morphine used in the i.m. group were converted to equivalent doses of diamorphine (1.5:1) [11] in order to compare drug usage within the three groups.

Incidence and patterns of hypoxaemia

Based on the results of the Part I study, in which normal subjects spent less than 1\% of their time with \( S_{ao} \) less than 94\%, we have defined “hypoxaemia” as \( S_{ao} < 94 \% \) occurring for more than 10\% of each epoch (i.e. > 6 min h⁻¹).

Before operation there was no difference between the three treatment groups in the time spent at \( S_{ao} < 94 \% \) (table II). However, in the postoperative period the patients receiving extradural diamorphine (group B) spent significantly more time with \( S_{ao} < 94 \% \) than patients in group A or group C (table II).

Using these criteria, nine of the 30 patients in this study showed postoperative hypoxaemia. In the hypoxaemic group of patients a very unstable pattern of \( S_{ao} \) distribution was seen (fig. 3), characterized by a wide distribution of \( S_{ao} \) values and a progressive decrease in \( S_{ao} \) after operation which was particularly marked during the night. This pattern was seen in five of the 10 patients in
group B and similar but less marked patterns were seen in two patients in group A and two patients in group C.

In the remaining 21 patients who did not develop hypoxaemia, two patterns of distribution of $S_{O_2}$ were seen. Five patients demonstrated a stable pattern of $S_{O_2}$ throughout the 24-h study period (fig. 4). The curve for each epoch was peaked and almost exactly superimposed that of the previous epoch. This resembled the pattern in the majority of patients in the Part I study. This stable pattern was seen in four of the 10 patients in group A (PCAS), no patients in group B (extradural) and one patient in group C (i.m.).

In the remaining 16 patients, a mildly unstable pattern of distribution of $S_{O_2}$, with some reduction in $S_{O_2}$, was seen (fig. 4) with the $S_{O_2}$ being less than 94% for less than 1.5 min h$^{-1}$.

There were no differences in $S_{O_2}$ before operation when the three treatment groups were compared, but when the preoperative $S_{O_2}$ was analysed in groups allocated according to their postoperative pattern of hypoxaemia, there was a significantly longer period (95% confidence limits) spent at $S_{O_2} < 94%$ in the preoperative period in the unstable group with hypoxaemia (table III).

The pre- and postoperative data and the duration of preoperative monitoring in the nine hypoxaemic patients are shown in table IV.

Hypoxaemia in the PCAS and i.m. groups occurred only in those patients with preoperative $S_{O_2} < 94%$ for longer than 6 min h$^{-1}$. However, in the extradural group, three patients who developed postoperative hypoxaemia had a normal distribution of $S_{O_2}$ before operation.
FIG. 4. Two examples of the stable pattern of $S_aO_2$ in two patients using PCAS. The bottom panel shows preoperative values and the upper panel postoperative values, for a 24-h period.

**TABLE III. Duration of hypoxaemia (min h$^{-1} < 94\% S_aO_2$) (mean (95\% confidence limits)) allocated according to postoperative pattern of $S_aO_2$.

<table>
<thead>
<tr>
<th></th>
<th>Non-hypoxaemic</th>
<th>Hypoxaemic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stable ($n = 5$)</td>
<td>Unstable ($n = 16$)</td>
</tr>
<tr>
<td>Before op.</td>
<td>0.4 (0.17-0.61)</td>
<td>1.2 (0.25-2.2)</td>
</tr>
<tr>
<td>After op.</td>
<td>0.9 (0.45-1.3)</td>
<td>1.4 (0.69-2.2)</td>
</tr>
</tbody>
</table>

Episodes of profound hypoxaemia

The $S_aO_2$ recordings were analysed for episodes of hypoxaemia divided into those at less than 90\% (moderate) and those at less than 85\% (severe) which lasted longer than 20 s.

Episodes of severe hypoxaemia were rare and there was no significant difference in the incidence of severe hypoxaemic episodes in the three treatment groups. However, three patients in the extradural group (patients Nos 12, 13 and 19) had

**TABLE IV. Duration of preoperative monitoring in the nine patients with postoperative hypoxaemia.

*No preoperative data available

<table>
<thead>
<tr>
<th></th>
<th>Patient No.</th>
<th>Duration (h)</th>
<th>$S_aO_2 &lt; 94%$ (min h$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Before op.</td>
<td>Before op.</td>
</tr>
<tr>
<td>Group A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCAS</td>
<td>4</td>
<td>1</td>
<td>6.4</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>11</td>
<td>14.4</td>
</tr>
<tr>
<td>Group B</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extradural</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>2</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>2</td>
<td>5.0</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>19</td>
<td>10</td>
<td>0.5</td>
</tr>
<tr>
<td>Group C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I.m.</td>
<td>6</td>
<td>0*</td>
<td>0*</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>10</td>
<td>7.6</td>
</tr>
</tbody>
</table>
POSTOPERATIVE HYPOXAEAMIA

TABLE V. Mean (SEM) pain scores (mm) derived from a 100-mm visual analogue scale

<table>
<thead>
<tr>
<th>Time (h)</th>
<th>4</th>
<th>8</th>
<th>12</th>
<th>16</th>
<th>20</th>
<th>24</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCAS</td>
<td>31</td>
<td>20</td>
<td>21</td>
<td>18</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>Group B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extradural</td>
<td>6</td>
<td>11</td>
<td>14</td>
<td>37</td>
<td>13</td>
<td>16</td>
</tr>
<tr>
<td>Group C</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i.m.</td>
<td>47</td>
<td>26</td>
<td>26</td>
<td>16</td>
<td>29</td>
<td>18</td>
</tr>
</tbody>
</table>

TABLE VI. Number of patients experiencing side effects in the postoperative period

<table>
<thead>
<tr>
<th></th>
<th>Group B Extradural (n = 10)</th>
<th>Group A PCAS (n = 10)</th>
<th>Group C i.m. (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vomiting</td>
<td>3</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Need for catheter</td>
<td>8</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Itching</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

episodes of moderate hypoxaemia lasting 3–90 min and patient 12 developed severe hypoxaemia requiring oxygen by facemask.

Pain scores

The pain scores for the three groups are shown in table V. Group B (extradural) patients had significantly lower pain scores ($P < 0.01$, analysis of variance, unpaired $t$ test) than the i.m. groups 4 h after operation. The situation was reversed at 16 h, when the i.m. group had significantly lower scores ($P < 0.05$, analysis of variance) than the extradural group. For the rest of the observation period, there was no significant difference in the pain scores of the three treatment groups.

Side effects

These are shown in table VI. On questioning several days after surgery, all patients said postoperative analgesia had been satisfactory.

DISCUSSION

Postoperative hypoxaemia occurring after abdominal surgery may be caused by a combination of pulmonary factors such as dependent lung atelectasis, and extrapulmonary factors such as obstructive sleep apnoea secondary to the use of opioid drugs [2, 12, 13]. This study was designed to examine postoperative oxygenation and, in particular, the effect of analgesia on extrapulmonary factors, because dependent lung atelectasis is very much less marked after lower than upper abdominal surgery [13]. The reference study was used to establish our criteria for hypoxaemia. We found that the distribution of $S_{aO_2}$ was skewed, with a median $S_{aO_2}$ of 98% and with less than 1% of values of the group being less than 94% $S_{aO_2}$. Three of the nine subjects in this reference group showed $S_{aO_2}$ values < 94% $S_{aO_2}$ for longer than this mean value of 0.6 min h$^{-1}$ and we set our threshold of hypoxaemia 10 times longer than this (6 min h$^{-1}$). The patterns of $S_{aO_2}$ vs time were either stable or unstable. The unstable pattern was characterized by a greater variability from hour to hour. Because of the shape of the dissociation curve, the less the $S_{aO_2}$, the more exaggerated is the appearance of the unstable pattern (figs 3, 5).

Monitoring the 30 patients following surgery showed that the majority had an $S_{aO_2}$ immediately after operation that was indistinguishable from normal. However, during the subsequent postoperative study period, almost 30% of patients developed hypoxaemia ($S_{aO_2} < 94\%$, 10–25 min h$^{-1}$ (95% CL)) following uncomplicated lower abdominal surgery and the hypoxaemia was most marked in those patients receiving extradural diamorphine. Moderate hypoxaemia ($S_{aO_2} < 90\%$) occurred in three patients (extradural group), but severe hypoxaemia ($S_{aO_2} < 85\%$) was rare in all three groups, only one patient requiring oxygen supplementation after operation. None of the patients in the PCAS or extradural groups was given preoperative or intraoperative opioids and this may account for the paucity of episodes of severe hypoxaemia ($S_{aO_2} < 85\%$) compared with the patients in the study by Catley and colleagues [2]; these were older and received opioids as premedication, during the operation, in recovery and as a continuous infusion after operation.

The periods of hypoxaemia occurred usually at night and were noticed to lessen after 06:00. It was noted also that desaturation was evident in some patients in both groups during the afternoon of the day following surgery and may have been related to the patient’s afternoon rest period. We have shown previously that all hypoxaemic episodes occurred whilst the patient slept [2] and others have shown that sleep disturbs ventilatory control [14–16]. It was not the intention of the study to relate hypoxaemia to sleep, so no
measurements of EEG were made and sleep and sleep stages could not be recognized. Nevertheless, the most profound hypoxaemia occurred between midnight and 06:00. The mechanism for this nocturnal hypoxaemia is unknown, but we have shown previously that the majority of hypoxaemic episodes were associated with obstructive rather than central apnoea [2]. There is a synergistic effect of opioids and sleep leading to an impairment of ventilatory control [2, 16].

An incidental finding which needs further study was the ability, in some subjects, to predict the likelihood of postoperative hypoxaemia by preoperative monitoring of $S_{aO_2}$. A value of $S_{aO_2} < 94\%$ for more than 4 min $h^{-1}$ was a strong predictor of postoperative hypoxaemia, irrespective of which analgesic regimen was used. In the PCAS and i.m. groups, hypoxaemia developed only in those patients with preoperative $S_{aO_2} < 94\%$ in excess of 4 min $h^{-1}$. However, three of five patients in the extradural group who developed postoperative hypoxaemia had normal preoperative values. The period of preoperative monitoring varied from 1 h to 11 h and, as the study developed, the importance of an extended period of preoperative monitoring became apparent. Further work is required to establish if a short period of $S_{aO_2}$ monitoring before operation in surgical patients would be as valuable as 8–10 h of overnight $S_{aO_2}$ monitoring.

The choice of a safe, effective method of pain relief on general wards remains unresolved. Although conventional i.m. analgesia in this study appeared to cause minimal desaturation with satisfactory pain scores, there is considerable evidence to suggest that pain relief is inadequate when i.m. opioids are used routinely. Patient-controlled analgesia in this series produced satisfactory results in terms of the visual analogue score, with minimal side effects and minimal respiratory disturbance in the majority of patients. Although there was no significant difference in the pain scores, the nursing staff were impressed both by the mobility of the patients using the PCAS and their ready acceptance of the technique. In this study, we examined the use of PCAS in patients with lower abdominal surgery who had received intraoperative extradural analgesia and in whom dosage was limited by a long lock-out time of 20 min. Because of those factors the dose of diamorphine used was small (26.8 mg) and it might be expected that patients after upper abdominal surgery without regional anaesthesia and with a shorter lock-out period would use
greater doses of opioid drug. Bolus administration of extradural opioid provided analgesia comparable to that obtained by patients in the PCAS group. However, its use was associated with an unstable pattern of $SaO_2$ with a high incidence (5/10) of prolonged hypoxaemia, even in three patients with normal preoperative $SaO_2$, and with side effects such as urinary retention, nausea and vomiting. In contrast with previous reports [3] suggesting a delayed onset of ventilatory depression with extradural opioids, we found that the decrease in $SaO_2$ occurred within 6 h of the end of surgery. There was a tendency for desaturation to recur on the afternoon of the day following surgery.

The consequences of this degree of hypoxaemia in young healthy patients are not known in detail, but it has been shown that an $SaO_2 < 85\%$ produced an abnormality of cognitive function [17]. In patients with cerebrovascular or coronary artery disease, and particularly in those with anaemia or with cardiovascular instability, this degree of hypoxaemia could have serious effects on organ function. In such patients postoperative oxygen therapy should be used routinely and for much longer periods of time than normal. In such high risk patients, provided they are not reliant solely on hypoxic ventilatory drive, it would seem advisable to monitor $SaO_2$ using pulse oximetry and to administer oxygen if $SaO_2$ decreases to less than 90\%.

Ventilatory rates were recorded by the nursing staff responsible for these patients. No patient had a recorded breathing rate of less than 10 b.p.m. despite hypoxaemia in 30\% of the subjects studied. This confirms the results of earlier work [2] that continuous oximetry (in patients breathing air) is a much more sensitive index of respiratory depression than simply measuring the frequency of ventilation. Of equal importance is the finding that continuous oximetry used in the preoperative period may be a predictor of postoperative hypoxaemia.

REFERENCES