The effect of premedication on oxygen saturation during the post-premedication period in 20 Chinese children undergoing elective surgery

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Summary
Peri-operative continuous pulse oximetric data were studied in healthy Chinese children randomly allocated to receive either pethidine 1 mg kg⁻¹ and atropine 0.02 mg kg⁻¹ intramuscularly 90 min prior to surgery (n = 10), or midazolam 0.5 mg kg⁻¹ and atropine 0.02 mg kg⁻¹ orally, 120 min before surgery (n = 10). Data were collected during the night before surgery, after premedication and for 8 h post-operatively. The pulse oximeter (Nellcor N-200E) output was retrospectively evaluated using Satmaster™, a computer programme which permits storage, retrieval, signal evaluation and compilation of oximetric data. There was no significant difference in the frequency, duration, or magnitude of desaturation episodes recorded during the post-premedication period compared to the desaturation episodes which occurred in the same child during normal sleep, on the night before surgery. Furthermore, there was no significant difference in the desaturation data between the two premedicant regimens. No child recorded a genuine desaturation less than 80% for longer than 15 s at any time during the study. Neither regimen significantly depressed saturation in otherwise healthy children presenting for minor surgical procedures.

Keywords: ANAESTHESIA, paediatric; PREMEDICATION, atropine, midazolam, pethidine; MEASUREMENT TECHNIQUES, pulse oximetry.

Introduction
The pulse oximeter has become a vital instrument in the detection of peri-operative hypoxaemia [1]. Previous studies of the effect of premedication on arterial oxygenation using intermittent blood gas sampling have reported conflicting results [2–4] and few workers have employed continuous pulse oximetry to examine the preoperative period [5,6]. Continuous oximetric data acquisition should overcome the limitations of intermittent data sampling and permit a more accurate comparison of the effect of drug regimens on oxygenation of children undergoing minor surgery. However, our previous work suggests that retrospective evaluation of peri-operative desaturation episodes may be misleading and necessitate the use of a computer programme such as Satmaster™ [7].

The aim of this study was to compare the effect of two premedication regimens on arterial oxygen saturation in the post-premedication period and the influence, if any, of premedication on the incidence of desaturation episodes during the first post-operative 8 h period, employing continuous, pulse oximetry in children undergoing minor surgery.

Methods
Twenty ASA grade I, Chinese children aged 1–8 years undergoing elective orthopaedic or a minor general surgical procedure were investigated. The study was approved...
Fig. 1. Plan of the study, showing periods of recording and inspired O₂ concentrations.

Results

Both groups in the aesthetic direct larynngoscopy and tissue prolapse group conformed with an interval scale of 0-28 min. The period of observation was 132.5 min.

There was no significant difference in the outcome of the investigation. The saturation of the target group after the intervention was compared with the control group. The statistical significance of the results was calculated using the Mann-Whitney rank-sum test and the Wilcoxon signed-rank test.
Only two patients (one from each group) recorded five or more desaturation episodes per hour with an $\text{SpO}_2$ $<$ 95% and $>$ 15 s duration, following premedication, and no patient recorded a genuine desaturation less than 80% for longer than 15 s at any time during the study. The minimum $\text{SpO}_2$ recorded after premedication in either group was 85% and the longest duration of a particular episode was 75 s to a lowest saturation of 91%.

**Discussion**

This study demonstrates that in otherwise healthy children, the use of two common premedication regimens employing either pethidine or midazolam, does not significantly depress oxygen saturation. These findings are at variance with previous studies in healthy children, one of which showed that both i.m. and intranasal (i.n.) midazolam (0.2 mg kg$^{-1}$) caused a significant decrease in arterial oxygen saturation from baseline values [5]. However, Rose and colleagues, using i.n. midazolam, demonstrated no change in oxygen saturation levels 15 min after administration [11]. In another study, premedication with rectal midazolam (0.35–0.45 mg kg$^{-1}$) lowered oxygen saturation 30 min after administration when compared with a placebo [6]. These apparently conflicting results can be explained by our ability to exclude movement-induced artefactual desaturation data before determining the incidence of genuine desaturation episodes [12]. Patient movement resulted in an overestimation of desaturation time by 75% in this study, confirming similar findings in patients following spinal surgery [8].

The route of administration of midazolam in children significantly alters bioavailability and the time to peak serum concentration, making comparison with other studies difficult [13]. Bioequivalence should be present between a 0.2 mg kg$^{-1}$ i.m. dose of midazolam and the 0.5 mg kg$^{-1}$ p.o. dose of midazolam used in this study, from 45 to 120 min after administration [13]. The peak serum concentration of midazolam occurs 15 min after i.m. administration [13] and the onset of sedation, together with the decrease in arterial oxygen saturation from baseline data, was similarly described in the study by de Santos and colleagues [5]. The peak incidence of the recorded minor desaturation episodes in our study occurred 45–105 min after administration of both pethidine and midazolam. It is therefore possible that our premedication regimen was not as sedative as those used in the Spanish study and did not depress respiration to
Table 2. Pulse oximetry data during the four study periods

<table>
<thead>
<tr>
<th>Data collection periods</th>
<th>Duration of data collection period per patient (h)</th>
<th>Mean SPO₂ (%)</th>
<th>P value (t-test)</th>
<th>Mean percentage of time with an SPO₂ &lt; 95%</th>
<th>P value (Mann Whiten U-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pethidine (n=10)</td>
<td>Midazolam (n=10)</td>
<td></td>
<td>Pethidine (n=10)</td>
<td>Midazolam (n=10)</td>
</tr>
<tr>
<td>Pre-operative night</td>
<td>11.04 (1.22) 9.63 (0.84)</td>
<td>98.9 (0.67) 98.7 (0.67)</td>
<td>0.48</td>
<td>0.2 (0.63) 1.1 (1.85)</td>
<td>0.08</td>
</tr>
<tr>
<td>After premedication</td>
<td>1.29 (0.34) 1.80 (0.31)</td>
<td>98.6 (0.84) 98.7 (0.94)</td>
<td>0.81</td>
<td>1.6 (2.96) 2.8 (5.00)</td>
<td>0.49</td>
</tr>
<tr>
<td>Recovery area</td>
<td>0.26 (0.13) 0.35 (0.19)</td>
<td>99.4 (0.70) 99.1 (1.20)</td>
<td>0.50</td>
<td>0.6 (1.30) 5.7 (12.10)</td>
<td>0.35</td>
</tr>
<tr>
<td>General ward</td>
<td>7.05 (1.05) 6.52 (1.96)</td>
<td>98.5 (0.70) 98.4 (0.84)</td>
<td>0.78</td>
<td>0.8 (1.30) 1.1 (1.40)</td>
<td>0.60</td>
</tr>
</tbody>
</table>

Data are mean (± SD).

Table 3. A pulse oximetry data before and after premedication

<table>
<thead>
<tr>
<th>Data collection periods</th>
<th>Pethidine group n=10</th>
<th>Mean % of time with an SPO₂ &lt; 95%</th>
<th>Desaturation episodes h⁻¹ with an SPO₂ &lt; 95% for &gt; 15 s</th>
<th>Midazolam group n=10</th>
<th>Mean % of time with an SPO₂ &lt; 95%</th>
<th>Desaturation episodes h⁻¹ with an SPO₂ &lt; 95% for &gt; 15 s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-operative night</td>
<td>0.2 (0.63)</td>
<td>0.36 (0.62)</td>
<td>1.1 (1.85)</td>
<td>0.65 (0.73)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>After premedication</td>
<td>1.6 (2.90)</td>
<td>1.48 (2.60)</td>
<td>2.8 (5.00)</td>
<td>1.82 (3.47)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value (Wilcoxon)</td>
<td>0.11</td>
<td>0.37</td>
<td>0.34</td>
<td>0.51</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are mean (± SD).

The same degree. Also, our study population may not match those in the other series, but a pharmacokinetic explanation of our oximetry findings is difficult to establish. The small lean body mass of Chinese children should result in a smaller central volume of distribution, higher peak serum concentration of midazolam, a more intense level of sedation of shorter duration, and consequently a higher incidence of episodic desaturation [14]. This was not the case, and, in a separate work, we have found that the serum concentration of midazolam 2 h after oral administration in Chinese children was similar to the serum concentration reported by other workers using a similar dosing regimen in Caucasian children [15]. Furthermore, none of the children in our study had any predisposition to upper airway obstruction or arterial oxygen desaturation and all were ASA class I. Peak respiratory depression in adults is observed within an hour of receiving intramuscular pethidine and there is a return towards baseline values commencing at about 2 h [16]. However, pethidine premedication did not depress ventilation in our children to a degree where significant arterial oxygen desaturation occurred, nor to a level that was clinically or statistically significantly different from each child's own sleep SPO₂.

In conclusion, we have shown that neither oral midazolam 0.5 mg kg⁻¹, nor i.m. pethidine 1.0 mg kg⁻¹ given as premedicants, significantly depressed SPO₂ in otherwise healthy children presenting for minor surgical procedures. Furthermore, raw oximetry data require careful evaluation and elimination of movement artefact before conclusions regarding the incidence of desaturation can be drawn. If analgesia is not a premedication requirement, then oral midazolam confers the advantage of avoiding the pain of an i.m. injection without compromising oxygen saturation.

References