ANAESTHESIA, MOVEMENT AND EMESIS

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SUMMARY

One hundred and eighty-two women undergoing dilatation and curettage were allocated randomly to receive premedication comprising temazepam, papaveretum–hyoscine or placebo. The temazepam recipients reported significantly fewer episodes of postoperative nausea. Movement was blamed by 66% of patients who identified a cause for nausea. These patients had higher scores on a motion sickness susceptibility questionnaire and were more likely to have been treated previously for nausea or vomiting. It may be possible to identify susceptible patients before surgery.

KEY WORDS


Postoperative nausea and vomiting are common and distressing symptoms and particularly affect women. Emetic properties are possessed by both opioids, which precipitate motion-induced nausea in conscious patients [1], and nitrous oxide, a weak opioid agonist [2], which also increases middle ear pressure [3]. This supports suggestions that motion sickness susceptibility predisposes to postoperative nausea and vomiting [4]. We have studied the influence of premedication and of movement on postoperative nausea and vomiting, to identify provoking factors, to determine the predictive value of motion sickness susceptibility and to reduce the incidence of postoperative nausea and vomiting by using a short acting benzodiazepine as premedication.

METHODS AND RESULTS

Following Ethics Committee approval, informed consent was gained from 182 healthy women undergoing elective dilatation and curettage. None was receiving emetic or antiemetic drugs and none was pregnant. Premedication comprised random allocation to receive placebo, oral temazepam 20 mg (10 mg if patient < 70 kg) or papaveretum 20 mg and hyoscine 400 μg (10 mg and 200 μg, respectively, if patient < 70 kg). A double dummy technique was used: each patient received both tablet and injection from coded packs 1 h before surgery. Anaesthesia was induced by one of two anaesthetists, with thiopentone 5 mg kg⁻¹ and 67% nitrous oxide in oxygen and halothane (as required during induction, 1% for maintenance of anaesthesia and discontinued after cervical dilatation). Manual ventilation of the lungs was avoided. After surgery, patients breathed 100% oxygen for 1 min before transfer in the left lateral position to the recovery room where they breathed oxygen-enriched air. When awake, patients were allowed to lie supine or laterally as they chose, before each was transferred to the same ward.

Simple observations of postoperative nausea and vomiting were made by the nurses in the recovery room, and in the ward between 4.5 and 6 h and again 20–24 h after surgery. A dedicated observer, using a structured format, recorded the incidence of nausea, retching and vomiting, and asked patients what they believed had caused these symptoms. Identification of causes was spontaneous, not from a list of suggestions. The normal practices of bed rest for the first 6 h after surgery, and of allowing sips of clear fluids


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TABLE I. Premedication groups, patient details (mean (SD)) and incidence of postoperative nausea and vomiting. \( ^*P = 0.03 \) compared with placebo, \( P = 0.05 \) compared with papaveretum–hyoscine

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Temazepam</th>
<th>Papaveretum–hyoscine</th>
</tr>
</thead>
<tbody>
<tr>
<td>( n )</td>
<td>61</td>
<td>65</td>
<td>56</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>43.1 (11.9)</td>
<td>44.5 (14.9)</td>
<td>43.1 (11.7)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>66.7 (12.6)</td>
<td>62.6 (12.8)</td>
<td>64.5 (12.6)</td>
</tr>
<tr>
<td>Patients with nausea (%) (95% confidence limits)</td>
<td>36 (25–49)</td>
<td>22 (13–34)*</td>
<td>34 (22–48)</td>
</tr>
</tbody>
</table>

TABLE II. Postal questionnaire. Factors associated with postoperative nausea and vomiting and motion sickness quotient (MSQ) (median and interquartile ranges, theoretical maximum score = 360). \( ^*P < 0.05 \) compared with other groups

<table>
<thead>
<tr>
<th>Nausea</th>
<th>No nausea</th>
<th>Movement blamed</th>
<th>Not blamed</th>
</tr>
</thead>
<tbody>
<tr>
<td>( n )</td>
<td>82</td>
<td>16</td>
<td>28</td>
</tr>
<tr>
<td>MSQ</td>
<td>21 (0–71)</td>
<td>69 (9–137)*</td>
<td>23 (0–57)</td>
</tr>
<tr>
<td>Previous treatment for nausea and vomiting (%)</td>
<td>10</td>
<td>38*</td>
<td>13</td>
</tr>
</tbody>
</table>

approximately 2 h after return to the ward were not altered.

To assess motion sickness susceptibility, 3–6 months later patients were sent a postal questionnaire which used a well validated method [5] to derive a quotient from the frequency of nausea and vomiting, provoked by different forms of transport. The questionnaire included questions on nausea and vomiting during pregnancy, causation by other stimuli including migraine, and treatment received for any cause of nausea and vomiting during the previous 12 months.

The three premedication groups were similar (table I). Emetic events were reported by 12% of patients in the recovery room, 31% at the first ward visit and 13% at the second ward visit. Nausea with vomiting or retching accounted for 56% of reports and nausea alone for the remainder. No patient reported vomiting or retching without nausea, and the incidence of symptoms was greatest at the first ward visit of the dedicated observer, so we analysed the incidence of nausea at this visit. Fifty-five of the 182 patients reporting nausea (with or without other emetic symptoms). Age and time within menstrual cycle had no influence. The incidence of nausea for the premedication groups was compared (Fisher's exact test). In the group receiving temazepam, 14 of 65 patients (22%) suffered nausea, compared with 22 of 61 (36%) in the placebo group \( (P = 0.03) \) (table I). The temazepam group also fared better than the papaveretum–hyoscine group which contained 56 patients, of whom 19 (34%) reported nausea \( (P = 0.05) \). There was no difference between the placebo and papaveretum–hyoscine groups.

Of all 55 patients reporting nausea, 33 volunteered a cause and, of these, 22 blamed movement whether passive or active. One hundred and twenty-six correctly completed postal questionnaires were returned (response rate 67%). Patients who had reported movement as provoking nausea had significantly higher motion sickness quotients (Mann–Whitney U test) (table II). There were too few patients to determine any association with morning sickness of pregnancy, migraine or previous postoperative nausea and vomiting.

COMMENT

In this study, we have found that temazepam as premedication reduced the incidence of reported postoperative nausea, that movement provoked it in many patients, and that those with a history of motion sickness or previous treatment for nausea and vomiting were particularly vulnerable. Our
study eliminated the influence of surgical and anaesthetic technique and it is unlikely that the results were caused by confounding influences.

The relatively high incidence of nausea associated with the use of papaveretum-hyoscine is not surprising, as morphine is an emetic. The cause of the unexpectedly high incidence in the placebo compared with the temazepam group is open to speculation: it may be a chance occurrence; higher level of arousal may predispose to nausea and vomiting; temazepam may have antiemetic properties, and as these events were all within 8 h of premedication, its amnesic properties may play a part. The response rate to the questionnaires was inevitably reduced as they were sent 3-6 months after surgery, with the aim of avoiding the influence of suggestions from other patients and staff, and to limit the impact of memories of postoperative nausea and vomiting. Despite this, the survey provides evidence that patients who had identified movement as provoking their symptoms had a specific susceptibility to motion sickness. Although they were also more likely to have received treatment for previous episodes of nausea and vomiting, these patients were not more susceptible to emesis caused by any other identified stimulus. The identification of causes of nausea is admittedly somewhat subjective, but the conclusion that movement, whether passive or active is a stimulus is difficult to avoid, and traditional exhortations not to disturb patients recovering from anaesthesia are sensible. Morphine may also cause postoperative nausea and vomiting by sensitizing the vestibular apparatus to the effect of movement [6], but our groups were too small to study the interaction of premedication and movement. In addition, all patients in our study received nitrous oxide which, in addition to increasing middle ear pressure during its uptake, is itself an opioid agonist. Movement during recovery from anaesthesia should perhaps be limited, but its relationship to postoperative nausea and vomiting needs further study.

REFERENCES