CORRESPONDENCE

TRANSDERMAL HYOSCINE AND POSTOPERATIVE NAUSEA AND VOMITING

Sir,—I refer to the recent double-blind comparison by Koski and colleagues [1] involving different types of surgery, in which transdermal hyoscine was found to be of no benefit in reducing the incidence of nausea and vomiting after operation. A recent study by Bailey and colleagues [2] concluded that transdermal hyoscine was an effective antiemetic in patients undergoing outpatient laparoscopy. This appears to be in accordance with the account given by Palazzo and Strunin [3] which described a lower incidence of nausea and vomiting in gynaecological operations than in intra-abdominal operations.

The strength of opioid analgesia used may be significant. The doses of fentanyl used by Koski [4] (3–5 μg kg⁻¹) were greater than those used by Bailey and colleagues [2] (0.5–2 μg kg⁻¹), and a previous study in which less potent analgesia was used (morphine and pethidine) [5] found transdermal hyoscine was effective even in patients undergoing different types of surgery. These limited studies on the prophylactic use of transdermal hyoscine suggest that its effectiveness may be affected by the potency of analgesia used.

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In a study just completed, we induced and maintained anaesthesia in a way similar to that described by Bailey and colleagues [2], but administered only 0.1 mg of fentanyl to inpatients undergoing laparoscopy. In recovery, only 5% of these patients vomited and no more made spontaneous complaints of nausea. Only when questioned specifically, 15% admitted feeling nauseated. In the postoperative ward, 15% of patients vomited within the first 8 h after operation. However, solid food was tolerated by 65% and liquids by 30% at the evening meal time. These initial results suggest that the tendency to nausea, vomiting, or both, may be reduced by maintaining anaesthesia mainly with inhalation anaesthetic agents and limiting the peroperative dose of analgesics.

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FAILED TRACHEAL INTUBATION

Sir,—I should like to add one or two comments to the excellent review by King and Adams [1]. White and Panjabi [2] believe that flexion/extension movements at the atlanto-occipital and atlanto-axial joints are more or less equal and that they function as a unit, “the occipito-atlanto-axial (OAA) complex”. King and Adams appear to suggest that bone structure limits movement at the OAA complex. I expect they refer to diseased patients (congenital or acquired), as flexion/extension movements of the OAA complex are not limited normally by abutment of the bones but by the tectorial membrane, which is the prolongation of the posterior longitudinal ligament inserted on the anterior rim of the foramen magnum. This accounts for those patients, with clinically poor top end movement, who have radiographically satisfactory separation of C0, C1 and C2.

We are performing a prospective study of difficult intubation in patients with diseased cervical spines. It is already quite clear that an absent atlanto–axial gap is a more powerful discriminant than an absent atlanto–occipital gap. Flexion/extension lateral radiographs are required to diagnose an absent gap [3]. A "neutral" view is acceptable if there are good gaps, but when they appear to be absent, one questions if it was a truly neutral position.

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Sir,—Thank you for the opportunity to comment on Dr Hendley’s letter.

The strength of opioid analgesia may certainly affect the incidence of postoperative nausea and vomiting. A tendency towards such a finding was noted also in our original study [1]. However, more extensive surgery was performed on the majority of patients in our study. Thus the group of patients receiving low doses of opioids was too small for statistical evaluation.
STUDIES ON MIVACURIUM

Sir,—Data published in the Canadian Journal of Anaesthesia [1] were used as a comparison group in two subsequent papers [2, 3]. One paper [2] made a comparison with a new group of patient data, whereas the other [3] reported use of a unique statistical treatment to analyse data among groups.

In the first paper [1], a more thorough treatment of the effects on both neuromuscular block and circulation caused by mivacurium during nitrous oxide-opioid anaesthesia was compared with those in patients receiving mivacurium during nitrous oxide-isoflurane anaesthesia. (Ninety patients were described in the nitrous oxide-opioid group; the first 45 patients from the nitrous oxide–opioid group were used as a comparison group in the other two papers. The last 45 patients in the paper in the Canadian Journal of Anaesthesia [1] were given doses which exceeded the ED$_{90}$.) We used data from those patients who received mivacurium 0.03–0.15 mg kg$^{-1}$ during nitrous oxide–opioid anaesthesia so as to provide comparison data for our studies during other types of anaesthesia. We failed to clarify this by appropriate references in subsequent papers.

In the third paper [3], slopes and intercepts of the dose–response curves for mivacurium during the three types of anaesthesia were compared using a $t$ test for multiple comparisons described by Winer and noted as reference No. 8 in this paper. This analysis is not found in either of the other two papers.

We were in error in failing to acknowledge that data from 45 patients were common to all three papers and that data from an additional 45 patients (anaesthetized with isoflurane) were common to two papers [1, 3]. We should have asked permission from the British Journal of Anaesthesia to use these data for comparisons in our other manuscripts. Copies of the other manuscripts should have been included for each Journal’s review at the time of original submission and they should subsequently have been cross-referenced correctly. We apologize for this error.

Finally, we have thought carefully about the sequence of events which occurred here and have tried hard to understand our actions and our intent. We had no intent to deceive. This was a large project, with 171 patients studied. The three manuscripts were all prepared and submitted at about the same time (February 21 to March 7, 1989) and we failed to ask for permissions or cross-reference them correctly. The data are true and correct responses to mivacurium during three types of anaesthesia.

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PROPOFOL: EFFECT ON THE MYOCARDIUM COMPARED WITH THE PERIPHERAL VASCULAR SYSTEM

Sir,—I read with great interest the article by Boer and colleagues [1] on the effect of propofol on peripheral vascular resistance during cardiopulmonary bypass. The authors state that propofol appeared to cause more hypotension than an equivalent dose of thiopentone, and that this may be related to the greater decrease in peripheral vascular resistance (PVR), citing my study [2] in their opening paragraph. They continue to say that the decrease in PVR was comparable to the decrease in arterial pressure, suggesting that vasodilatation may be a major factor in propofol-induced hypotension.

I believe the authors are incorrect in what they state. The study plainly revealed no significant decreases in either PVR or SVR, but significant decreases in left cardiac work index and left stroke work index of 35%. Cardiac index decreased by 18%, while mean arterial pressure decreased 23%. The data indicate myocardial depression without loss of systemic vascular resistance. Authors such as Williams and colleagues [3], Kaplan and colleagues [4] and Van Aken and colleagues [5] have also indicated that propofol is indeed a myocardial depressant drug.

Dr Boer and his colleagues are to be congratulated on performing such an interesting study and also on their statement that the results are influenced by special factors present during cardiopulmonary bypass, in particular haemodilution, hypothermia and the use of non-pulsatile blood flow. Data resulting from the use of drugs during cardiopulmonary bypass to determine their effects on the vascular bed are, indeed, difficult to extrapolate to the intact human being.

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