CORRESPONDENCE

TRANSDERMAL HYOSCINE AND POSTOPERATIVE NAUSEA AND VOMITING

Sir,—Koski and colleagues [1] found no difference in the incidence of nausea and vomiting in patients receiving transdermal hyoscine or placebo before operation. This is at variance with the findings of others [2].

A number of points warrant mention. In the study of Koski, different types of surgery were performed, whereas only patients having gynaecological surgery were studied by Uppington, Dunnet and Blogg [2]. It has been noted that the incidence of nausea and vomiting differs depending upon the type of surgery undertaken—for example, 40–70% for intra-abdominal, 20–50% for gynaecological and 15% for surface/limb operations [3].

The dose of fentanyl given may have differed from that given by Uppington and colleagues, who used 3–5 µg kg⁻¹. What was the dose range in the current study?

The assessment of nausea and vomiting, although estimated on a similar scale, was made by an unspecified number of recovery nurses in the Koski study whereas, at most, only three observers made assessments in the study of Uppington and colleagues.

Finally, the fluid regimen differed in the two studies: 2–5 ml kg⁻¹ h⁻¹ (Koski) and 5–10 ml kg⁻¹ h⁻¹ (Uppington).

The aetiology of nausea and vomiting is multifactorial [3]. The study by Koski and colleagues failed to demonstrate a difference between transdermal hyoscine and placebo. However, the differences highlighted above should be noted before dismissing the value of transdermal hyoscine as a prophylactic against nausea and vomiting for some types of surgery.

G. A. FRANCIS
London

REFERENCES

Sir,—In our study, 53% of the operations were gynaecological laparotomy, 28% upper abdominal and the remainder mostly thyroid or breast surgery. We studied carefully the incidence of nausea and vomiting within the subgroups and found no statistically significant difference between the placebo or hyoscine groups.

The dose of fentanyl was 3–5 µg kg⁻¹, as in the study by Uppington and colleagues [1].

The possible bias between the nurses’ estimation of nausea and vomiting was avoided by the standardized monitoring method. Nausea was recorded using the following grading: 3 = vomiting patient; 2 = patient experiencing and spontaneously complaining of feeling nauseas; if no nausea was noted, the nurses asked the patients: “Do you feel nauseated?” A positive answer was graded as 1 and a negative answer as 0.

The fluid infusion in our study was given in such a manner as to maintain normovolaemia and fulfill the normal daily fluid requirement.

E. M. J. KOSKI
Kuopio, Finland

REFERENCE

UNPREDICTABLE SPINAL ANAESTHESIA

Sir,—One of the criticisms that continue to be levelled against the use of plain 0.5% bupivacaine for spinal anaesthesia is unpredictable spread of anaesthesia and, in particular, inadequately low blocks in some patients. The work of Bannister, McClure and Wildsmith, using the smallest concentration of glucose (0.33%), supports this view—as does previous work by Logan, McClure and Wildsmith [1, 2]. However, they unjustly malign plain 0.5% bupivacaine as an agent for spinal anaesthesia. When it is administered to patients in the seated rather than the lateral position which they have used, adequate levels of analgesia are attained readily with plain 0.5% bupivacaine [3]. Furthermore, plain is more predictable than hyperbaric 0.5% bupivacaine when used in this way [3].

Undoubtedly, both plain and hyperbaric solutions of bupivacaine are valuable agents for spinal anaesthesia. However, appropriate positioning for lumbar puncture is required to obtain the best results from both agents.

R. P. ALSTON
Glasgow

REFERENCES
Sir,—Dr Alston’s findings show that plain and hyperbaric bupivacaine produce equally predictable levels of sensory block when given intrathecally to the seated patient [1]. Our study showed that predictability may be obtained with lumbar puncture in the lateral position without unacceptably high blocks [2]. Many practitioners find the lateral position more convenient, especially in patients with restricted mobility, rather than sitting and maintaining that position for 2 min as recommended by Dr Alston.

Clearly, both techniques may be used as appropriate to achieve the desired effect.

J. BANNISTER
Dundee

REFERENCES

EXTRADURAL SOMATOSTATIN

Sir,—The report by Desborough and colleagues [1] of the use of extradural somatostatin in postoperative pain was read with some concern. Currently there is a body of peer-reviewed literature which suggests that somatostatin 1–14 administered spinally in three different species may have an irreversible, deleterious effect on spinal tissue [2–7] at doses less than those which produce analgesia.

Animal studies might be deemed sufficiently different (multiple dosing, higher concentration, intrathecal route) as not to be relevant to human studies. I could accept this rationale if the mechanisms of toxicity were known, and excluded in humans. Indeed, I should be enthusiastic about a rationale if the mechanisms of toxicity were known, and not to be relevant to human studies. I could accept this (multiple dosing, higher concentration, intrathecal route) as being supportive of continued human investigation.

A small additional query relates to the nature of the molecule used, its vehicle and source. Somatostatin is the name given to a family of peptides that range from 14 to 28 amino acids and have different properties. These peptides may be diluted with salts and other products from the synthesis and therefore, the source and vehicle should be quoted.

T. L. YAKSH
San Diego

REFERENCES

Sir,—Our study was carried out between July 1987 and April 1988 after Ethics Committee approval was granted on July 20, 1987. None of the publications concerning neurotoxicity were available before our submission to the Ethics Committee or during the study.

The Ethics Committee and the patients were informed that somatostatin had been given by the extradural and intrathecal route in human studies and had been reported to be effective in the treatment of acute and chronic pain without side effects. The study proceeded with the full knowledge and support of Serono U.K., the suppliers of the somatostatin 1–14 which was a freeze-dried, lyophilized preparation diluted in 0.9% sodium chloride.

We agree that the studies quoted by Professor Yaksh deal with neurotoxicity resulting from intrathecal administration of somatostatin in different species and cannot be related directly to human studies using extradural somatostatin. None of the patients in our study suffered any neurological sequelae.

However, we pointed out in our discussion the more recent work by Gauman and Yaksh [1] and Mollenholt and colleagues [2] and we stated “possible toxic effects suggest that its further use in clinical practice is unjustifiable”.

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REFERENCES