Correspondence

Sedative effects of morphine and clonidine

Editor—We were interested to read the article by Lena and colleagues on the effects of intrathecal morphine and clonidine for coronary artery bypass grafting, in which a significant reduction of postoperative morphine requirements was demonstrated. We have some comments to make.

The sedative effect of intrathecal morphine and clonidine was deemed insufficient to prevent extubation. However, formal sedation scoring was not undertaken.

Chiari and colleagues demonstrated significant dose-dependent sedation after administration of intrathecal clonidine, as the sole agent in the first stage of labour (using doses of 50–200 μg). Filos and colleagues also describe significant sedation after intrathecal clonidine (150 μg administered post-Caesarean section under general anaesthesia). These doses would be comparable to the 1 μg kg⁻¹ dose of intrathecal clonidine administered in the study by Lena and colleagues.

Postoperative sedation could have contributed to the low use of patient-controlled analgesia in the clonidine group. Sedation scoring after extubation might also have revealed this. Any sedation may have been further compounded by addition of droperidol to the PCA, as it has well-recognized sedative actions. We wondered if use of a 5-HT antagonist would have addressed the issue of postoperative nausea and vomiting without the sedative side-effects.

Earlier extubation in the clonidine group was, as the authors note, confounded by the significantly smaller dose of intraoperative sufentanil. Whilst this is a short-acting agent and should not have affected time to extubation, we do wonder if the anaesthetist was adequately blinded to the different groups.

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Editor—We thank Drs Wattie and Jaggar for their comments concerning the side-effects of intrathecal clonidine. We agree that sedation induced by intrathecal clonidine could prevent patients from using PCA morphine and that it could be considered, at least theoretically, as a bias of the study. On the other hand, if sedation prevented use of the PCA, the VAS scores would have been higher in the clonidine + morphine group compared with the i.v. PCA morphine group, but we observed the opposite. In addition, others have demonstrated that clonidine reduced PCA morphine consumption independently of its sedative effect. In patients given i.v. morphine, sedation has been reported to be dissociated from analgesia, sedated patients occasionally having high VAS pain scores.

We were aware of the sedative effect of the droperidol used in the PCA morphine solution and consequently limited the amount given to 3 mg in 30 ml. Patients in the morphine + clonidine group should have received a mean dose of 7 mg over 24 h, but the mean dose of droperidol taken by these patients was 0.7 mg. This dose is very unlikely to have contributed to any sedation.

Clonidine’s anaesthetic and opioid-sparing effects have been documented for many years. A study design that guaranteed double blindness by using a bispectral index to which the anaesthetist was blinded, has showed a significant decrease in anaesthetic consumption in patients receiving clonidine. Consequently, the sufentanil-sparing effect observed in the intrathecal clonidine group may explain the earlier extubation but does not seem to be the cause of any bias in our study.

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