Chiari and colleagues\textsuperscript{2} demonstrated significant dose-dependant sedation after administration of intrathecal clonidine, as the sole agent in the first stage of labour (using doses of 50–200 $\mu$g). Filos and colleagues\textsuperscript{3} also describe significant sedation after intrathecal clonidine (150 $\mu$g administered post-Caesarean section under general anaesthesia). These doses would be comparable to the 1 $\mu$g kg$^{-1}$ dose of intrathecal clonidine administered in the study by Lena and colleagues.\textsuperscript{4}

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.\textsuperscript{4} 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.

M. L. Wattie
S. I. Jaggar
London, UK

Editor—We were interested to read the article by Lena and colleagues\textsuperscript{1} 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.

Sedative effects of morphine and clonidine

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