Emergence agitation after sevoflurane anaesthesia in children

Editor—we read with interest the article by Lee and colleagues comparing propofol and fentanyl for prevention of emergence agitation after sevoflurane anaesthesia in children. We thank the authors for their work in this very common and often very distressing scenario.

We would like to raise a few important questions regarding this study. Pain is one of the important causes for emergence agitation in children, and although the authors excluded the children in whom caudal analgesia had not worked, we think assessment of pain by appropriate scales would have been very helpful in addressing this confounding factor. Also considering the authors have used lidocaine for caudal analgesia and the patients had at least 60 min duration of anaesthesia and a further 40 min in Post Anaesthesia Care Unit (PACU), we consider assessment of pain in PACU would have been invaluable data in this study.

Propofol was used as the rescue agent for severe agitation in PACU. Considering that propofol was one of the study drugs, we think that it introduces bias into the study.

The starvation times used in this study were very long compared with the common clinical practice in the UK (where it is 6 h for solids and 2 h for clear fluids). We appreciate this could have been the standard protocol of the institution, but longer starvation periods would lead to more distress before induction, which possibly could have led to some exclusions from the study. Preoperative anxiety and distress is another significant contributing factor for emergence agitation in children. Parental stay during recovery of anaesthesia is another factor influencing emergence agitation in children. We understand the policy of the institution of not having parental presence in PACU influencing the study protocol, and we would like to commend the authors in acknowledging this factor for higher incidence of emergence agitation in Group S.

Declaration of interest
None declared.

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Comparison of propofol and fentanyl for preventing emergence agitation in children

Reply from the authors

Editor—we thank Dr Narayanasamy and Ghori for their interest in our study and would like to take this opportunity to respond to their comments.

This study’s aim was to compare the preventive effects on emergence agitation (EA) between propofol and fentanyl, which were commonly investigated by previous studies. As Dr Narayanasamy and Ghori mentioned, postoperative pain has been considered one possible aetiological factor of EA. We also recognized that the same standard of postoperative pain control for each group is essential for appropriate comparison among the drugs for preventing EA. So, the caudal block was applied identically to all the groups, and this procedure was performed by a seasoned paediatric anaesthesia specialist. In addition, cases were withdrawn if there was any doubt about the success of the caudal block. Because we performed this study under the assumption that there were no differences in the degree of analgesia among the groups, any scales regarding pain were not investigated nor described. Although we used lidocaine for caudal block, a large volume (1.2 ml kg⁻¹) was used for caudal block and additional analgesics were not required in patients who successfully completed the study. In addition, the time from caudal block to completion of anaesthesia was just 35–40 min during ~60 min of anaesthesia duration. Thus, we think that we could adequately evaluate the severity of the EA among three groups under the identical clinical conditions without the postoperative pain although we did not assess the pain by appropriate scales.

The primary outcome, which was compared in this study, was the peak EA score or scale and the rescue drug was used for behavioural control of patients with severe EA which lasted >5 min. In other words, the evaluation of the patients who were treated with the rescue agents was finished because they had already been deemed severely agitated before drug administration. Therefore, we think that
the kind of rescue drug was not a factor affecting the incidence or severity of EA among the groups and the impact of propofol as the rescue medication on the results of this study was insignificant. In addition, the proved effects and its rapid onset and duration should be considered for choosing the rescue drug because all the enrolled patients underwent ambulatory surgery. In this study, propofol was preferentially considered as the rescue drug because of its rapid pharmacokinetics and known effects on the EA.

We followed the old-fashioned policy of our institution regarding the starvation time for the safety of patients undergoing anaesthesia. We are currently discussing this issue to find an appropriate starvation time in children. However, we do not consider that long starvation time would affect our results because this is a comparative study and the three groups were under the same conditions regarding the starvation time. If we had applied a shorter starvation time, we assume that the baseline values might be affected but the differences among the groups would not. In addition, even though a long starvation time could lead to more preoperative agitation, only a small number of children had severe preoperative agitation in the present study and they were withdrawn. Thus, we believe that this issue did not affect our results.

There have been conflicting results about the preventive effects on EA of parental presence. Burke and colleagues reported that parental presence at emergence period did not decrease EA in young children. Thus, further studies are required to address this issue.

Declaration of interest

None declared.

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Differences in study results

Editor—We thank Dr Marhofer and colleagues for publishing new information on the captivating subject of the use of dexmedetomidine to prolong the duration of nerve block. We also have published a recent work using dexmedetomidine as an adjuvant to local anaesthetics for peripheral nerve blocks. We were very interested in reading their results and have noticed a few interesting differences. First, the sensory duration they report using a similar local anaesthetic is somewhat shorter than that reported in the literature and that reported in our results. Perhaps their measure of sensory block using pinprick compared with our use of all three modalities (pinprick, light touch, and ice) explains this time difference. Typically, ice recognition was the last sensation recovered by our volunteers. Having both used volunteers it is difficult to establish which modality might represent the appearance of pain in a postoperative setting. This is likely an interesting question to be resolved by a future study.

Secondly, their work, when compared with ours, seems to show that when using a lower adjuvant dose of dexmedetomidine, there is the absence of noticeable haemodynamic effects. Although healthy and young, certain subjects in our study demonstrated a noticeable decrease in systolic and diastolic arterial pressure between 60 and 480 min. Two volunteers experienced a 30% decrease in systolic arterial pressure when compared with the baseline value as compared with none in the group without dexmedetomidine. Heart rates were similar between groups except at 60 min when a significant decrease was noted.

Thus when putting our results in perspective, we obtain the following: