and may therefore offer advantages in situations similar to that reported by Ludbrook et al. Additionally, anaesthetists prefer TCI over a manual infusion scheme[5].

The role of TCI is still evolving, and it is too early to fully endorse it for management of challenging cases such as the one reported. However, I feel TCI does merit consideration.

DAVID R. BALL
Aberdeen Royal Infirmary, U.K.

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Propofol infusion for the difficult airway: Reply

Thank you for the opportunity to reply to Dr Ball's letter regarding the use of slow administration of propofol at induction in the patient with a potentially difficult airway. Dr Ball makes two separate points. The first is that the synergistic effects of concurrently administered drugs may increase the sensitivity of patients to propofol. We agree that there will potentially be an altered dose-response relationships in this setting (although, one needs to consider a mechanism of altered re-distribution in the types of studies conducted in the paper cited by Dr Ball), and this was evident by the low dose of propofol required to induce anaesthesia in this patient. This was the rationale behind the choice of a slow infusion, which allowed close assessment of the patient in an environment of relatively slowly changing levels of anaesthesia. This therefore allowed induction without major risk of "burning any bridges", in a manner akin to the use of inhalational induction in this setting.

With respect to the second point regarding the use of TCI systems for induction with propofol, we agree that the role of these systems in the induction process merits consideration. However, we disagree with the statement that these systems provide precise control of sedation and anaesthesia. Rather, they provide precise control of the blood concentrations of propofol. Sedation and anaesthesia are related to the brain concentrations of propofol, and these are poorly related to its blood concentration during induction[1]. We believe that the optimal induction of anaesthesia with propofol will require consideration of the determinants and time-course of its brain concentrations[2,3], and that this analysis is lacking from the commercially available TCI systems. We are currently analysing the implications of this deficiency.

G. LUDBROOK
Royal Adelaide Hospital, S.A.

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Fatal outcome after propofol sedation in children

We read with interest the recent article by Plötz et al[1] who describe another fatality in a child on a paediatric intensive care unit (PICU) in association with propofol sedation. This latest case shows striking similarities to a series of previously reported fatalities and "near-misses"[2].

The child presented with an upper respiratory tract infection necessitating endotracheal intubation, and was sedated with high-dose propofol. Within 48 hours he developed a metabolic acidosis, arrhythmias, progressive myocardial failure, a high temperature and an increased concentration of creatine phosphokinase. Contrary to previous cases however, the authors postulated a diagnosis of malignant hyperthermia, treated the child with dantrolene and thus were able to demonstrate a remarkable improvement of the clinical situation. Unfortunately this was overshadowed by a diagnosis of brain death due to prolonged hypotension.

This case report warrants commenting on for several reasons.

Dantrolene sodium acts primarily by affecting calcium flux across the sarcoplasmic reticulum of