dren receiving low dose vs high dose propofol infusion for urologic surgery. Examining the dose related effect of propofol on CBFV in children is important to our understanding of the differences in cerebrovascular responses between children and adults. However, no details about the age distribution of children in each of the randomized groups are presented, nor are differences in age between the two groups described. This is relevant because CBFV changes with age.² The data appear to have been analyzed without consideration for the age-related differences in CBFV. We recognize that there are only six children in each group. Nevertheless without this information it is difficult to determine whether the difference in CBFV between the low dose and high dose propofol groups are valid for children between one and six years of age.

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REPLY:

We would like to thank Drs. Vavilala and Lam for their interest and comments regarding the effects of propofol on cerebral blood flow velocity (CBFV) in children.¹ The suggestion that CBFV changes with age is an accurate one, although the most rapid changes occur outside of the age range of our patient population. From birth to 18 months CBFV increases rapidly, followed by a much slower increase to peak values at around seven years of age.² As such, the age range of our study patients was in fact chosen in order to minimize age-related effects on CBFV, and we are confident that the analysis reported the variation in CBFV in response to propofol dosing, rather than age-related factors. The coefficient of variation was found to be well within acceptable values, suggesting that variability in CBFV was not likely due to age-related effects.

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High-degree atrioventricular block after the administration of atropine for sinus arrest during anesthesia

To the Editor:

We describe a case of sudden sinus arrest of six seconds' duration under general anesthesia in a patient with preoperative asymptomatic bifascicular block. Atropine 0.5 mg was ineffective in resolving the sick sinus syndrome, and the arrhythmia progressed to atrial flutter with high-degree atrioventricular block.

A 77-yr-old man with chronic renal failure was admitted for construction, under general anesthesia, of an arteriovenous fistula by compound vascular graft for hemodialysis. His electrocardiogram (ECG) showed complete right bundle branch block with left axis deviation. Anesthesia was induced with thiamylal 150 mg, vecuronium 6 mg, and fentanyl 0.1 mg *iv*. A mixture of 50% oxygen, 50% nitrous oxide, and approximately 0.8% isoflurane was given for maintenance of anesthesia. Sinus arrest of six seconds' duration occurred 110 min after induction. Intravenous atropine 0.5 mg was administered immediately but was ineffective, and his ECG showed progression to atrial flutter with high-degree atrioventricular block (Figure). Thereafter, 1 mg of epinephrine *iv* was given and sinus rhythm was restored.

Sick sinus syndrome has been reported after spinal anesthesia¹ and after induction of general anesthesia.² The authors of these two reports suggested that the balance between sympathetic and parasympathetic tone is important in the maintenance of heart rate. Parasympathetic tone appeared to be predominant in our patient. General anesthesia was induced primarily to relax the patient during a surgical procedure of long duration. Thus, an imbalance in the autonomic nervous system may have resulted in sinus arrest.

FIGURE The rhythm strip displays atrial flutter with high-degree atrioventricular block, which appeared after treatment with atropine.

Administration of atropine or isoproterenol or emergency cardiac pacing is the routine treatment for sinus arrest. Interestingly, it is known that low-dose atropine has different effects at the sinoatrial node and the atrioventricular node, which result in paradoxical bradycardia or a deterioration of atrioventricular conduction, respectively. According to a review by Das, either vagotonic stimulation of the central nervous system or peripheral inhibition of cholinesterase or both were thought to be possible causes of the paradoxical effect with low-dose atropine, but the precise explanation is still unclear.³

In our patient, atropine 0.5 mg appeared to be inadequate to block the parasympathetic influence on the heart, and it led to an augmentation of bradycardia and atrioventricular block. Therefore, we would like to emphasize the necessity of administering a sufficient dose of atropine when treating this kind of life-threatening arrhythmia.

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