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Letter to the Editor

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Reply to letter "A fatal adverse event upon adenotonsillectomy in a child. Are Brugada syndrome and propofol real accomplices?"

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Dear Editor,

We would like to thank you for giving us the opportunity to respond to the points raised in Dr Flamee's letter. We would also like to thank Dr Flamee and his colleagues for their interest in our report and for taking the time to express their opinion.

As per the emergency department note, no active bleeding was noted and the oropharynx appeared patent during intubation, findings which argue against life-threatening airway obstruction as the cause of this child's presentation. In addition, serum electrolytes were obtained and measured within the normal range (sodium 149 mEq/L, potassium 4.9 mEq/L and, calcium 10.8 mg/dL). Therefore, dyselectrolytemia was also unlikely a cause for this child's electrocardiographic abnormalities and malignant ventricular arrhythmias. A limited echocardiogram was performed in the emergency department during atrial rhythm and showed normal biventricular size, low normal biventricular function, and no evidence of pericardial effusion.[1] We also measured plasma troponin concentration at presentation which was only mildly elevated (0.06 ng/mL; normal < 0.03 ng/mL). The echocardiographic finding of low normal biventricular function which is common after cardiopulmonary arrest and only mild elevation in serum troponin concentration noted in this patient argue against severe myocardial damage from prolonged severe hypoxemia and ischemia. Moreover, cardiopulmonary arrest, which leads to global myocardial ischemia, is likely to produce generalised electrocardiographic abnormalities than changes localised to the right ventricular outflow tract region, which favour Brugada syndrome.

Propofol has complex pharmacokinetics which include an initial distribution phase with a half-life of 1.8–9.5 minutes, a second redistribution phase with a half-life of 21–70 minutes, and a terminal elimination phase with a half-life of 1.5–31 hours.[2] It remains unclear whether propofol and/or one of its metabolites is responsible for the reported adverse cardiac electrical effects. Moreover, cardiac electrical abnormalities have persisted well beyond the expected duration of effect for propofol,[3,4] and complete elimination period for several other drugs[5] suggesting permanent disruption of relevant ion channels or downstream proteins could underlie this observation.

The authors have cited trials in which no adverse electrical effects were noted after propofol administration in patients with Brugada syndrome.[6,7] Of note, the electrographic findings were only assessed 3 to 20 minutes after propofol administration with the underlying assumption that hypnotic effect of propofol correlates with its cardiac effects, which may not be true.[6,7] The majority of patients in these studies were adults who are often treated with medications that have the potential to alter autonomic tone or have antiarrhythmic effects, such as beta-blockers, calcium channel blockers, digoxin, quinidine, or other antiarrhythmics to name a few. In addition, data pertaining to administration of parasympatholytic agents such as atropine prior to induction with propofol, which can modulate the expression of Brugada phenotype, were not reported. While extrapolation of adult data to paediatric age group patients is sometimes unavoidable, it is fraught with a number of challenges due to fundamental differences between the two populations and should therefore be done with caution.[8]

We are in agreement with the authors that altered autonomic tone in the post-operative period could have contributed to the observed electrocardiographic findings and Brugada-related malignant arrhythmias in this patient and have stated so in our report.[1] We are also cognizant of the fact that it is difficult to ascribe causality based on a single patient report; however, we do believe that reports like ours inform the need for systematic evaluation of cardiac electrical effects of anesthetics such as propofol in the young.

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Conflict of interest. None.

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