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Quick response code

Sustained intraoperative bradycardia revealing Sengers syndrome

Sir,

We read with interest the article by Zarrouki *et al*. revealing the characteristic manifestations of Sengers syndrome in a 6-month-old infant.[1] The authors encountered bradycardia unresponsive to atropine in the infant immediately following induction of anaesthesia. It was associated with decrease in end-tidal carbon dioxide ($EtCO_2$) as low as 8 mmHg. Following resuscitation and completion of surgery, transthoracic echocardiography showed hypertrophied and apical hypokinesia of the left ventricle responsible for the event.

We believe that even though the child had a predisposing cardiac condition, high doses of propofol triggered the event. It is not clear from the text as to why the authors administered propofol when sevoflurane was already used for induction of anaesthesia. The authors administered an initial dose of propofol 3 mg/kg (equal to 18 mg for a 6 kg infant) followed by another 30 mg totalling to 48 mg i.e. 8 mg/kg for a 6 kg infant which is much higher than the commonly used induction dose (2.5–3 mg/kg) in children <2 years of age.

Propofol is a known cardiac depressant with a potential to cause severe bradycardia due to resetting or inhibition of the baroreceptor reflex mechanism.[2] Propofol also attenuates the heart rate response to atropine in a dose-dependent manner.[3] Moreover, younger children have high vagal tone, and another mechanism of bradycardia might also have been the manipulation of the laryngopharynx in a light plane of anaesthesia. Although the authors have not mentioned any readings of blood pressure, we presume that the drop in $EtCO_2$ was most probably due to systemic hypotension caused by high doses of propofol. Nevertheless, the authors managed the case well and have enabled the readers to be aware of the consequences of Sengers syndrome during the perioperative period.

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Conflicts of interest

There are no conflicts of interest.

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