Dexmedetomidine for Recurrent Emergence Delirium after Failed Treatment with Propofol and Midazolam

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Introduction
Emergence agitation (EA) is a dissociative state of inconsolability that occurs after anesthesia with a reported incidence as high as 80% (1). Although widely studied, the cause of EA remains unknown, and methods aimed at its prevention and treatment are not universally effective. In the following case report, combining sevoflurane with an infusion of dexmedetomidine in a child with a history of severe EA proved to be effective at preventing EA.

Case report
An 11 year old child with a history of Ewing’s Sarcoma presented for placement of a central line under general anesthesia. All previous anesthetics had been complicated by cases of severe EA. Several anesthetic regimens including the administration of preparative midazolam, and propofol based total intravenous anesthesia (TIVA) had failed to prevent the occurrence of EA. On this particular occasion, the decision was made to use a combination of sevoflurane and a dexmedetomidine infusion for maintenance anesthesia. Preoperative vital signs and laboratory values were age appropriate. Midazolam premedication was administered and induction of anesthesia was performed with propofol and fentanyl. A laryngeal mask airway was inserted, followed by sevoflurane administration at 1 MAC and a dexmedetomidine infusion at a rate of 0.3 mcg/kg/hr. A ‘deep extubation’ was performed at the conclusion of the case. There were no symptoms of EA throughout the recovery period.

Discussion
In this case report dexmedetomidine was effective in preventing EA in a child who had a history of severe EA despite previous attempts at prophylaxis with midazolam and propofol TIVA. Midazolam and propofol had been used during prior anesthetics because of their reported benefits of reducing the incidence of EA (2, 3).

Recent studies have shown administration of the α2 –adrenoceptor agonist dexmedetomidine is associated with a reduced incidence of EA. In a prospective, double blind, randomized control study, a rapid IV bolus of 4 mcg/ml of dexmedetomidine at a dose of 0.5 mcg/kg/hr was associated with a reduced incidence of EA (4).

In another study, a dexmedetomidine infusion at 0.2 mcg/kg/hr decreased the incidence of EA when compared to placebo (5). Other studies have found dexmedetomidine to be better at preventing EA than midazolam or propofol (6, 7).

In summary, our case report adds to the literature suggesting that dexmedetomidine may be considered for the prophylaxis against EA. The side effect profile of dexmedetomidine including excessive sedation, hypertension, hypotension and bradycardia need to be taken into consideration during use of the drug.

References