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Propofol: Bolus Induction plus Continuous Infusion in a Patient with Duchenne Muscular Dystrophy

To the Editor:—The use of propofol in children known to have Duchenne muscular dystrophy (DMD) has not been reported. A recent study using propofol in malignant hyperthermia (MH)-susceptible swine revealed it to be nontriggering.¹ There have been sporadic reports citing the probable safety of propofol in adults who are MH-susceptible.^{2,3}

The anesthesia technique often suggested for patients with DMD is nitrous oxide/opioid anesthesia, with or without tracheal intubation.⁴ However, this method often is associated with prolonged emergence. We describe here the case of a 10-yr-old boy who had biopsy-proven DMD, and who required emergency mastoidectomy for decompression of a facial nerve entrapment and palsy. The surgeons had requested that he be awakened as soon as possible after surgery to evaluate the success of the planned procedure.

Using a vapor-free anesthesia machine, we induced anesthesia with propofol $2.0 \mu\text{g} \cdot \text{kg}^{-1}$ via bolus intravenous injection. After tracheal intubation facilitated with atracurium, anesthesia was maintained with a propofol infusion of $100\text{--}150 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and ventilation with 70% nitrous oxide in oxygen. All parameters, arterial blood gases, and temperature remained stable throughout.

Upon discontinuation of the propofol infusion, the child awakened rapidly. He was observed in the intensive care unit for 24 h and subsequently discharged without complications. Serum creatinine phosphokinase concentrations were within normal limits.

We offer this technique for use in children with DMD.

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No Coronary Dilation: No Coronary Steal

To the Editor:—I read with interest the recent report¹ by Hartman and co-workers demonstrating that isoflurane does not cause intercoronary or transmural steal in their chronic dog model of steal-prone coronary anatomy. I wondered: "why not?" because a previous study in our laboratory in a similar model did find intercoronary steal with isoflurane.² Hartman's group speculated that this difference might result from their use of an awake control (whereas we used basal sedation with morphine and chloralose) or the use of repeated short-term occlusions to stimulate collateral development (whereas we used an amero-constrictor).

I offer an alternative to these explanations, based on the data Hartman *et al.* present in table 5, "Effects of isoflurane on regional myocardial perfusion." Data for flow to normal myocardium remote from the occluder and stenosis do not demonstrate coronary dilation, even at 1.9% isoflurane, when blood pressure was returned to control levels. This lack of dilation certainly accounts for a lack of steal. Therefore, the results of Hartman *et al.*'s study should be extrapolated to humans with caution, because isoflurane *does* cause coronary dilation in humans.³

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