Cardiovascular Collapse in an Infant Undergoing Sevoflurane Induction

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Introduction: Sevoflurane has by and large replaced halothane as the inhalational induction agent of choice for pediatric anesthesia. Like halothane, sevoflurane is non-pungent and non-irritating to the airway; however, since it is less soluble in blood and tissue than halothane, it induces more rapidly. As an induction agent, sevoflurane has also been associated with fewer cardiovascular events than halothane, perhaps because it causes milder interference with SA node automaticity and less myocardial depression. Despite such advantages, however, induction with sevoflurane may still lead to untoward cardiac effects. This case report describes a cardiovascular collapse during anesthesia induction with oxygen (O₂), nitrous oxide (N₂O) and sevoflurane.

Case Presentation: A 3-month-old, 2.36 kg, ASA III, ex-premature male with a complicated medical history consisting of Grade IV intraventricular hemorrhage, cerebral atrophy, broncho-pulmonary dysplasia, nephrotic syndrome and hypothyroidism was scheduled for an inguinal hernia repair. The child had undergone a PDA repair at 22 days of age and a laryngoscopy/bronchoscopy at 2 months of age. Intravenous agents had been used to induce general anesthesia in both cases without any reported complications. Preoperative medications included levothyroxine, metoclopramide and chlorothiazide.

The patient was induced with 2 to 8% sevoflurane along with a 50/50% combination of O₂/ N₂O. Mask ventilation was easy, with a normal CO₂ waveform and a 100% SpO₂. However, as a 22-gauge intravenous catheter was placed into the right arm, the heart rate suddenly dropped from approximately 130 beats per minute to sinus bradycardia in the 40s. Blood pressure and the CO₂ tracing could no longer be detected. Atropine was administered, N₂O and sevoflurane were discontinued, CPR was initiated and the patient was intubated with a 4.0 uncuffed endotracheal tube (ETT). As a result, spontaneous circulation immediately returned, vital signs normalized, a physiologic end-tidal CO₂ tracing reappeared and SpO₂ levels returned to 100%. After discussion with the surgeon, the decision was made to proceed with the surgery. The remainder of the case proved to be uneventful, and blood gas analysis showed no evidence of either end organ damage or hypoperfusion. Upon follow-up the next day, the patient was noted to be at his pre-operative mental and physical baseline.

Conclusion: Although sevoflurane has been shown to have a safer cardiovascular profile than halothane, this case demonstrates that it is not a totally benign agent when used for induction of general anesthesia. Moreover, sevoflurane has been shown to have greater hemodynamic effects on infants than on older children. In critically ill infants, such cardiovascular instability is likely to be even more profound. A possible explanation for this observation is that while sevoflurane causes a less pronounced myocardial depression than halothane, it also causes a greater drop in peripheral vascular resistance. Therefore, when considering a patient with an immature heart and a depleted volume status, this drop in peripheral vascular resistance may be the reason that profound cardiovascular instability is observed in some critically ill infants. We believe that more research focusing on sevoflurane’s effect on this subgroup of patients is indicated.

References:
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