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Hemodynamic and Ventilatory Changes Following the Intraoperative Administration of Dexmedetomidine: Sevoflurane vs. Desflurane

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Introduction: Dexmedetomidine is a highly selective central α_2-adrenergic agonist which produces anxiolysis, amnesia, sedation, potentiation of analgesia, and sympatholysis. In addition to its other clinical applications, it has been shown to significantly decrease the incidence of emergence agitation following general anesthesia with either sevoflurane or desflurane. As there may be the potential for increased or synergistic hemodynamic or respiratory depression when dexmedetomidine is combined with inhaled anesthetic agents, we prospectively compared the hemodynamic and respiratory effects of a single dose of dexmedetomidine administration in pediatric patients anesthetized with either desflurane or sevoflurane.

Methods: The study was approved by the institutional IRB. Infants and children scheduled for general anesthesia with endotracheal intubation were considered eligible for inclusion. Patients were held NPO for 4-6 hours. Premedication with midazolam in acetaminophen was followed by inhalation induction with sevoflurane in 100% oxygen. Following anesthetic induction, intravenous access was obtained and endotracheal intubation was facilitated with 0.2 mivacurium. Following endotracheal intubation, fentanyl (1.5-2 µg/kg) was administered and anesthesia maintained with either sevoflurane or desflurane (0.7-1 MAC) and monitored using the Bispectral Index (BIS). When there was spontaneous return of neuromuscular function, spontaneous ventilation was allowed. Once this was accomplished, dexmedetomidine (0.5 µg/kg) was administered over 5 minutes (time 0 to 5). To evaluate hemodynamic and ventilatory effects, heart rate (HR), systolic blood pressure (sBP), diastolic blood pressure (dBP), and end-tidal CO_2 ($ETCO_2$) were recorded prior to the administration of dexmedetomidine (time 0 or baseline), at the completion of the dose (time: 5 minutes), at 5 minutes post dexmedetomidine administration (time: 10 minutes), and 10 minutes post dexmedetomidine administration (time: 15 minutes). The lowest hemodynamic (HR, SBP, DBP) value, and highest $ETCO_2$ value, between time 0 and time 15 were noted. Demographic data (age and weight) between the 2 groups were compared using a non-paired t-test while gender between the 2 groups was compared using a contingency table and a Fisher's exact test. Within group hemodynamic changes (HR, sBP, dBP), and ventilatory ($ETCO_2$) changes were compared using a Donnett test where the values at 5, 10 and 15 minutes were compared to the control value (time 0). Between group hemodynamic and ventilatory changes at 0, 5, 10, and 15 minutes were compared using a two-way analysis of variance with a Bonferroni correction for multiple comparisons. The lowest HR, sBP, dBP, and the highest $ETCO_2$ following the administration of dexmedetomidine in patients anesthetized with sevoflurane were compared to patients anesthetized with desflurane using a non-paired t-test. All data are presented as the mean ± SD with $p<0.05$ considered significant.

Results: A total of 50 patients were enrolled in the study (22 sevoflurane, 28 desflurane). There were no differences in the demographics between the 2 groups. Sevoflurane: age 57 ± 39 months, weight 22.2 ± 12.2, gender (M/F) 12/10. Desflurane: age 63 ± 29 months, weight 22.8 ± 13.8, gender (M/F) 18/10. In patients anesthetized with both sevoflurane and desflurane, there was a statistically significant decrease in HR at 5, 10, and 15 minutes following the administration of dexmedetomidine when compared to baseline. In patients anesthetized with sevoflurane, the HR values at 0, 5, 10 and 15 minutes were 137 ± 23, 115 ± 20, 109 ± 21, and 104 ± 19 beats/min respectively. In patients anesthetized with desflurane, the HR values at 0, 5, 10 and 15 minutes were 142 ± 19, 127 ± 20, 124 ± 2, and 119 ± 19 beats/min respectively. The lowest recorded HR during the 15 minutes following dexmedetomidine administration was 103 ± 20 in with sevoflurane vs. 116 ± 19 beats/minute with desflurane ($p=0.0231$). The lowest sBP for 15 minutes following dexmedetomidine in patients anesthetized with sevoflurane was 92 ± 13 mmHg ($p<0.01$ compared to baseline of 104 ± 16 mmHg) and the lowest sBP following dexmedetomidine in patients anesthetized with desflurane was 97 ± 11 mmHg ($p<0.01$ compared to baseline of 108 ± 11 mmHg and p=NS, desflurane vs. sevoflurane). There were no statistically significant changes in dBP and $ETCO_2$ when comparing baseline values to those obtained following the administration of dexmedetomidine.

Discussion: We noted no clinically significant hemodynamic or ventilatory changes following the administration of dexmedetomidine to patients anesthetized with either sevoflurane or desflurane. However, the decrease in HR was greater in patients anesthetized with sevoflurane when compared to desflurane. Given the negative chronotropic effects of sevoflurane, the HR changes following dexmedetomidine can be expected to be greater in patients anesthetized with sevoflurane when compared to desflurane. Although we noted no clinical consequences of these effects, the potential for HR decreases should be considered in sub-populations who may not tolerate this relative bradycardia.