Dexmedetomidine for Emergence Agitation after Sevoflurane Anesthesia in Preschool Children Undergoing Day Case Surgery: Comparative Dose-Ranging Study

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Abstract

Background: The present study was designed to compare the efficacy and safety of two different doses of 0.3 and 0.5 µg/kg dexmedetomidine on emergence agitation (EA) after sevoflurane based anesthesia in children undergoing day case surgery.

Methods: 60 pediatric patients ASA physical status I or II, aged 3-6 years old, scheduled for day case surgery were studied. Children were randomly assigned to one of 3 groups (n=20), control (C) group received 10ml saline, (D1) group received dexmedetomidine 0.3 µg/kg and (D2) group received dexmedetomidine 0.5 µg/kg. Anesthesia was induced and maintained with sevoflurane inhalational anesthetic. Dexmedetomidine doses was diluted in 10ml saline and administered over 10min after induction of anesthesia. Heart rate (HR) and mean arterial blood pressure (MAP) were recorded after induction of anesthesia, after drug administration and then every 10min during the procedure and in the postanesthesia care unit (PACU) until discharge. Time to eye opening and to achieve full alderte score were recorded. Incidence and severity of EA was evaluated using Aono's and pediatric Anesthesia Emergence Delirium (PAED) scores respectively.

Results: MAP and HR decreased significantly in D2 group compared to its base line and to the control group till PACU admission (p<0.05). Hemodynamic values were comparable between D1 and C groups throughout the study period (p>0.05). The incidence of severe EA was significantly lower in group D1 and D2 groups (30% and 20% respectively) than that in the control group (60%) (p<0.05). The PAED scales on PACU admission were significantly lower in D1 and D2 groups compared to C group (p<0.05). The time to eye opening and to achieve full alderle score were significantly longer in D2 group than C group (p<0.05).

Conclusion: Dexmedetomidine at a dose of 0.3 and 0.5 µg/kg reduced the incidence and severity of emergence agitation after sevoflurane anesthesia in the pre-school children. The lower dose of 0.3 µg/kg was associated with better recovery profile and more hemodynamic stability than the dose of 0.5 µg/kg.

Key Words: Dexmedetomidine – Emergence agitation – Sevoflurane.

Introduction

EMERGENCE agitation (EA) is a commonly seen complication in children undergoing general anesthesia, with an estimated incidence of 18-80% [1]. Although EA is short lived and self-limiting yet it can result in injury to the patient, it requires greater post-operative nursing care and leads to dissatisfaction and anxiety to the parents.

The etiology of EA remains undefined. Risk factors such as preschool age, some surgical procedures, postoperative pain and the use of sevoflurane anesthesia are suggested to provoke EA [2,3].

As sevoflurane with its popular anesthetic characteristics has largely replaced halothane in pediatric clinical practice, the number of articles related to EA has thus increased. Studies comparing sevoflurane with halothane maintenance of anesthesia found higher incidence of agitation in children anesthetized with sevoflurane than those with halothane [4-6]. This renewed the interest of pediatric anesthesiologists in finding different techniques in order to provide efficient and high-quality care for both patients and their parents.

Multiple pharmacological interventions have been used to prevent EA as benzodiazepines, opioid analgesics and alpha 2 adrenoceptor agonist [7-9]. Dexmedetomidine (DEX), a highly selective alpha 2 adrenoceptor agonist (receptor selectivity alpha 2/alpha1=1620/1) that has sedative and analgesic effects as well as limited clinical effects on ventilatory functions and has thus been successfully used in prevention of EA in pediatric patients [10-13]. However, stimulation of alpha 2 adrenoceptor...
Effect of Dexmedetomidine on Emergence Agitation

can result in hemodynamic changes as hypotension, bradycardia and even sinus arrest depending on dose, rapid loading and co-morbidities in the patient [14,15].

Different doses, regimen and routes of administration of dexmedetomidine were studied to investigate its effect on postoperative agitation [16-19], however the optimum protocol for its use without adverse effect is not yet well established.

The present study was designed as double blind, randomized, clinical study to compare the efficacy and safety of two different doses of 0.3 and 0.5 µg/kg dexmedetomidine on EA after sevoflurane based anesthesia in children undergoing day case surgery. The incidence and severity of EA, time to eye opening, perioperative hemodynamic and respiratory effects were studied.

Material and Methods

After Ethics Committee approval and obtaining written informed parental consent, 60 pediatric patients ASA physical status I or II, aged 3-6 years old, scheduled for day case surgery were studied. The study was conducted at Abu El-Rish Hospital, Cairo University from April 2009 to December 2010.

Children with severe chest infection, heart disease, mental or neurologic disease were all excluded from the study.

Children were randomly assigned by computer generated list of numbers to one of 3 groups, control (C) group received 10ml saline, (D1) group received dexmedetomidine 0.3 µg/kg and (D2) group received dexmedetomidine 0.5 µg/kg (n=20 for each). Dexmedetomidine (Precedex, Abbott Laboratories, North Chicago, IL) was diluted in 10ml of 0.9% NaCl. An assistant anesthesiologist not involved in data collection prepared the study syringe for each patient.

Upon arrival to the operating room standard monitors including electrocardiogram, non-invasive blood pressure and pulse oximeter were attached (Infinity SC 8000, Drager medical system, Avenue, Danvers, MA, USA) and baseline readings were recorded (T0).

Anesthesia was induced in all groups with 5-8% sevoflurane (Sevorane, Abbott Laboratories S.A, Abbott Park, IL, USA) in oxygen through facemask and a peripheral intravenous line (22G)

was inserted. After obtaining sufficient depth of anesthesia, the airway was secured with laryngeal mask airway (LMA). Anesthesia was maintained using sevoflurane inhalational anesthetic, spontaneous ventilation was allowed and capnogram was attached to obtain continuous measurement of ETCO2. After placement of LMA, the study drug or placebo were administered as IV bolus over 10min by anesthetist who was blind to the group assignment.

In all groups 10mg/kg paracetamol suppository was given after induction of anesthesia and the surgeon infiltrated the wound with 0.25% bupivacaine at the end of surgery.

Sevoflurane concentration was adjusted by the attending anesthetist to control the mean arterial blood pressure (MAP) and heart rate (HR) ±20% of baseline. Intra-operative HR and MAP were recorded after induction of anesthesia (T1), after drug administration (T2) and then every 10min until the end of the procedure (mean intraoperative value) (T3). End tidal sevoflurane concentration was recorded at 10 minutes interval. If any patient developed SpO2 <95% or ETCO2 >50mmHg, assisted bag ventilation was started.

At the end of surgery sevoflurane was discontinued and LMA was removed then the child was transferred to the postoperative anesthetic care unit (PACU). The time from the discontinuation of sevoflurane till eye opening on command (Time to eye opening) was recorded.

Parents were allowed to stay with their children in the PACU. During PACU stay MAP, HR and SpO2 were continuously monitored. MAP and HR were recorded upon arrival to the PACU, at 10, 20min postoperatively and on PACU discharge (T4-T7). If oxygen saturation fell below 95%, oxygen facemask was given to the child.

Modified Aldrete score (0-10 point scale) [20] was used to monitor sedation on PACU admission and at 5min interval. Time to achieve full Aldrete (≥9) was recorded. Children's behavior was evaluated on PACU admission using Aono's scale [21] (1=calm; 2=not calm but could be easily calmed; 3=not easily calmed, moderately agitated; 4=combative or disoriented). Agitation score of 3 or 4 was considered as an agitation episode. The severity of EA was evaluated using Pediatric Anesthesia Emergence Delirium (PAED) scale (Table 1) [22] which provides a score from (0-20) upon arrival to PACU, at10 and 20min postoperatively then on PACU discharge (T4-T7). Post-operative pain was
assessed at the same time intervals using Objective Pain Score (OPS) [23]. Each criterion scored from (0-2) to give a total score of (0-10). If OPS ≥4, 1-2mg/kg diclofenac suppository was administered. Midazolam 0.1mg/kg intravenously was given to treat agitation without pain.

Children were discharged from PACU after satisfying discharge criteria of being calm, fully awake, minimum pain, stable vital signs and oxygen saturation >95% on room air. Discharge time which was defined as the time from PACU admission until the child fulfilled the discharge criteria was recorded.

Statistical analysis:

Statistical analysis was performed using SPSS-win statistical package version 15 (SPSS Inc., Chicago, IL). Data was presented as mean (SD), median (range) or number (%) as appropriate. Comparison between the two groups was performed using unpaired student's t-test. Scores was compared between the two group using Mann-Whitney test. Intragroup comparison relative to baseline was performed using repeated measure analysis (ANOVA) with post hoc Dunnet's test if ANOVA results were significant. Categorical variables were compared using Chi-square test. A p-value <0.05 was considered significant.

Results

There were no difference in the patients characteristics, duration of surgery among the three groups (Table 2).

Table (1): Pediatric anesthesia emergence delirium (PAED) scale [22]

<table>
<thead>
<tr>
<th>Score</th>
<th>C group n = 20</th>
<th>D1 group n = 20</th>
<th>D2 group n = 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>The child makes eye contact with the caregiver</td>
<td>4= Not at all</td>
<td>3= Just a little</td>
<td>3= Just a little</td>
</tr>
<tr>
<td>The child's actions are purposeful</td>
<td>2= Quite a bit</td>
<td>1= Very much</td>
<td>1= Very much</td>
</tr>
<tr>
<td>The child is aware of his/her</td>
<td>0= Extremely</td>
<td>0= Extremaly</td>
<td>0= Not at all</td>
</tr>
<tr>
<td>The child is restless</td>
<td>0= Not at all</td>
<td>1= Just a little</td>
<td>1= Just a little</td>
</tr>
<tr>
<td>The child is inconsolable</td>
<td>2= Quite a bit</td>
<td>2= Quite a bit</td>
<td>2= Quite a bit</td>
</tr>
<tr>
<td></td>
<td>3= Very much</td>
<td>3= Very much</td>
<td>3= Very much</td>
</tr>
<tr>
<td></td>
<td>4= Extreme</td>
<td>4= Extreme</td>
<td>4= Extreme</td>
</tr>
</tbody>
</table>

The highest value of PAED scale is 20.

The three studied groups were comparable as regards the baseline values of MAP and HR. After dexmedetomidine injection in D2 group, MAP and HR decreased significantly compared to its base line and to the control group and returned to its base line at 10min postoperatively (p<0.05). Comparable values were reported in D1 and C groups throughout the study period (Tables 3,4). In group D2 three children out of 20 experienced intraoperative desaturation (SPO₂ <95%) versus no patient in D1 and C groups (p>0.05).

Mean Endtidal sevoflurane showed no significant difference between D1 and D2 groups. However it was significantly lower in D1 and D2 groups (2.3±0.6 and 2.2±0.3 respectively) when compared to the control group (2.8±0.5) (p<0.05).

The time to eye opening and to achieve full Aldrete score (≥9) in the PACU were significantly longer in D2 group compared to C group (p<0.05). Comparable Times were recorded in D1 and C groups. Time to PACU discharge as defined by the protocol was comparable in all groups (Table 5).

The incidence of EA (Aono’s scale ≥3) was significantly lower in group D1 and D2 groups than that in the control group (p<0.05) (Table 5). The PAED scales on PACU admission were significantly lower in D1 and D2 groups compared to C group (p<0.05) (Table 6). There was no statistical significant difference in EA incidence and PAED scales between D1 and D2 groups (p>0.05) (Tables 5,6).

OPS scores were comparable among the three groups at all time interval during PACU stay (p>0.05). (Table 7).

Table (2): Demographic data and duration of surgery.

<table>
<thead>
<tr>
<th>Score</th>
<th>C group n = 20</th>
<th>D1 group n = 20</th>
<th>D2 group n = 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>4.5±1.9</td>
<td>4.7±1.8</td>
<td>4.3±2.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>19.1±4.1</td>
<td>21.3±4.2</td>
<td>21.4±3.9</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>17/3</td>
<td>18/2</td>
<td>16/4</td>
</tr>
<tr>
<td>ASA (I/II)</td>
<td>18/2</td>
<td>17/3</td>
<td>18/2</td>
</tr>
<tr>
<td>Duration of surgery (min.)</td>
<td>41.2±13.1</td>
<td>43.6±11.4</td>
<td>39.7±13.6</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD or ratio. C group = Control group. D1 group = 0.3μg/kg dexmedetomidine. D2 group = 0.5μg/kg dexmedetomidine. ASA = American Society of Anesthesia.
Table (3): Mean arterial blood pressure measures (mean ±SD).

<table>
<thead>
<tr>
<th></th>
<th>C group</th>
<th>D1 group</th>
<th>D2 group</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 20</td>
<td>n = 20</td>
<td>n = 20</td>
<td></td>
</tr>
<tr>
<td>T0</td>
<td>81.61±7.1</td>
<td>80.61±6.8</td>
<td>81.32±7.4</td>
</tr>
<tr>
<td>T1</td>
<td>79.64±6.7</td>
<td>78.43±7.2</td>
<td>79.21±7.8</td>
</tr>
<tr>
<td>T2</td>
<td>80.52±6.6</td>
<td>77.27±5.8</td>
<td>75.14±5.2</td>
</tr>
<tr>
<td>T3</td>
<td>79.73±5.3</td>
<td>78.41±7.1</td>
<td>74.81±5.4†</td>
</tr>
<tr>
<td>T4</td>
<td>82.11±5.8</td>
<td>80.52±7.3</td>
<td>76.36±6.5†</td>
</tr>
<tr>
<td>T5</td>
<td>81.63±7.1</td>
<td>79.73±6.9</td>
<td>79.32±6.2</td>
</tr>
<tr>
<td>T6</td>
<td>80.62±7.4</td>
<td>81.43±6.9</td>
<td>80.41±6.8</td>
</tr>
<tr>
<td>T7</td>
<td>81.23±5.9</td>
<td>80.41±6.1</td>
<td>82.62±5.1</td>
</tr>
</tbody>
</table>

C group = Control group.
D1 group = 0.3 µg/kg dexmedetomidine.
D2 group = 0.5 µg/kg dexmedetomidine.
T0 = Base line.
T1 = After induction.
T2 = After dexmedetomidine or placebo administration.
T3 = Mean intraoperative value.
T4 = On postanaesthesia care unit (PACU) admission.
T5 = After 1 hmin in PACU.
T6 = After 20 min in PACU.
T7 = On PACU discharge.
† = p<0.05 compared to the C group.
‡ = p<0.05 compared to baseline.

Table (4): Heart rate values (mean ± SD).

<table>
<thead>
<tr>
<th></th>
<th>C group</th>
<th>D1 group</th>
<th>D2 group</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 20</td>
<td>n = 20</td>
<td>n = 20</td>
<td></td>
</tr>
<tr>
<td>T0</td>
<td>121.32±8.7</td>
<td>123.13±9.6</td>
<td>121.91±8.9</td>
</tr>
<tr>
<td>T1</td>
<td>117.91±8.2</td>
<td>119.41±9.2</td>
<td>117.62±9.8</td>
</tr>
<tr>
<td>T2</td>
<td>117.51±9.1</td>
<td>117.92±8.5</td>
<td>110.46±9.2†</td>
</tr>
<tr>
<td>T3</td>
<td>118.21±7.1</td>
<td>116.96±10.2</td>
<td>109.35±8.7†</td>
</tr>
<tr>
<td>T4</td>
<td>122.42±7.6</td>
<td>119.35±8.2</td>
<td>112.61±9.1†</td>
</tr>
<tr>
<td>T5</td>
<td>119.63±6.2</td>
<td>117.42±9.1</td>
<td>116.42±9.6</td>
</tr>
<tr>
<td>T6</td>
<td>120.37±7.1</td>
<td>119.61±8.4</td>
<td>118.39±7.3</td>
</tr>
<tr>
<td>T7</td>
<td>120.91±7.3</td>
<td>121.43±8.1</td>
<td>120.16±9.4</td>
</tr>
</tbody>
</table>

C group = Control group.
D1 group = 0.3 µg/kg dexmedetomidine.
D2 group = 0.5 µg/kg dexmedetomidine.
T0 = Base line.
T1 = After induction.
T2 = After dexmedetomidine or placebo administration.
T3 = Mean intraoperative value.
T4 = On postanaesthesia care unit (PACU) admission.
T5 = After 1 hmin in PACU.
T6 = After 20 min in PACU.
T7 = On PACU discharge.
† = p<0.05 compared to the C group.
‡ = p<0.05 compared to baseline.

Table (5): Recovery times and incidence of emergence agitation [mean ± SD or number (%)].

<table>
<thead>
<tr>
<th></th>
<th>C group</th>
<th>D1 group</th>
<th>D2 group</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 20</td>
<td>n = 20</td>
<td>n = 20</td>
<td></td>
</tr>
<tr>
<td>Time to eye opening (min.)</td>
<td>8.4±1.3</td>
<td>9.2±2.1</td>
<td>11.7±1.9*</td>
</tr>
<tr>
<td>Time to full Aldrete (min.)</td>
<td>11.1±1.7</td>
<td>11.7±2.1</td>
<td>14.3±2.7*</td>
</tr>
<tr>
<td>Time to discharge from PACU (min.)</td>
<td>39.2±7.2</td>
<td>41.3±6.1</td>
<td>43.7±7.6</td>
</tr>
<tr>
<td>Incidence of agitation [n (%)]</td>
<td>(12) 60% (6) 30%* (4) 20%*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

C group = Control group.
D1 group = 0.3 µg/kg dexmedetomidine.
D2 group = 0.5 µg/kg dexmedetomidine.
PACU = Postanaesthesia care unit.
* = p<0.05 compared to C group.

Table (6): Pediatric Anesthesia Emergence Delirium (PAED) scores in all groups [median (range)].

<table>
<thead>
<tr>
<th></th>
<th>C group</th>
<th>D1 group</th>
<th>D2 group</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 20</td>
<td>n = 20</td>
<td>n = 20</td>
<td></td>
</tr>
<tr>
<td>On PACU admission</td>
<td>14 (5-18)</td>
<td>7 (0-15)*</td>
<td>6 (0-15)*</td>
</tr>
<tr>
<td>After 10 min</td>
<td>5 (0-11)</td>
<td>5 (0-7)</td>
<td>4 (0-7)</td>
</tr>
<tr>
<td>After 20 min</td>
<td>3 (0-5)</td>
<td>2 (0-5)</td>
<td>2 (0-5)</td>
</tr>
<tr>
<td>On PACU discharge</td>
<td>0 (0-4)</td>
<td>0 (0-4)</td>
<td>0 (0-3)</td>
</tr>
</tbody>
</table>

C group = Control group.
D1 group = 0.3 µg/kg dexmedetomidine.
D2 group = 0.5 µg/kg dexmedetomidine.
PACU = Postanaesthesia care unit.
* = p<0.05 compared to control group.

Table (7): Objective pain score (OPS) values in all groups [median (range)].

<table>
<thead>
<tr>
<th></th>
<th>C group</th>
<th>D1 group</th>
<th>D2 group</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 20</td>
<td>n = 20</td>
<td>n = 20</td>
<td></td>
</tr>
<tr>
<td>On PACU admission</td>
<td>5 (1-6)</td>
<td>4 (0-6)</td>
<td>4 (0-5)</td>
</tr>
<tr>
<td>After 10 min</td>
<td>4 (0-6)</td>
<td>3 (0-5)</td>
<td>3 (0-5)</td>
</tr>
<tr>
<td>After 20 min</td>
<td>3 (0-5)</td>
<td>3 (0-5)</td>
<td>3 (0-4)</td>
</tr>
<tr>
<td>On PACU discharge</td>
<td>2 (0-4)</td>
<td>2 (0-4)</td>
<td>2 (0-3)</td>
</tr>
</tbody>
</table>

C group = Control group.
D1 group = 0.3 µg/kg dexmedetomidine.
D2 group = 0.5 µg/kg dexmedetomidine.
PACU = Postanaesthesia care unit.

Discussion

This study showed that post-induction administration of dexmedetomidine 0.3 and 0.5 µg/kg reduced the incidence and severity of emergence agitation after sevoflurane anesthesia in the preschool children. Although both dexmedetomidine doses were effective in decreasing the EA yet the lower dose of 0.3 µg/kg was associated with better recovery profile and more hemodynamic stability than the dose of 0.5 µg/kg.

Sevoflurane, with its hemodynamic stability, minor airway adverse effects and rapid induction and emergence characteristics is considered the agent of choice for induction and maintenance of anesthesia in the pediatric age group [24,25]. Despite the clear usefulness of sevoflurane anesthesia, there is definite evidence of emergence agitation after its use in children [26] which could be attributed to its low blood solubility and rapid emergence [25].

In a previous study, Ibacache et al. [27] compared the effect of 0.1 and 0.3 µg/kg dexmedetomidine on EA and found better control with the 0.3 µg/kg group. Other studies used higher doses of dexmedetomidine and reported that dose as high as 0.5 or 1 µg/kg of dexmedetomidine reduced the incidence of post-sevoflurane EA significantly however they were associated with prolongation of post-anesthesia recovery and hemodynamic instability.
Based on the results of these previous studies, the authors of this work decided to be more cautious and bolus doses of 0.3 and 0.5 µg/kg IV dexametomidine were chosen to evaluate their efficacy on reducing EA with minimal side effects.

Pediatric anesthesia emergence delirium (PAED) scale is the most comprehensive, reliable, valid and currently available tool to measure the severity of EA. However it lacks a validated threshold value to indicate the presence or absence of EA [30]. That is way in the current study the independent Aono’s four point scale was used to assess the incidence of EA and the PAED was used to assess its severity.

In the present study, dexametomidine at doses of 0.3 and 0.5 µg/kg similarly reduced the incidence of EA and the PAED scores compared to the control group, however children who received 0.5 µg/kg had significantly prolonged time to eye opening when compared to those in the control group. These results coincided with the results of previous studies [17,19,27]. Guler et al. [17] reported that administration of 0.5 µg/kg dexametomidine at the end of the surgery reduced the incidence of EA with prolongation of the times to extubation and eye opening when compared to placebo. Ibacache et al. [27], who used different doses of dexmedetomidine 0.15 and 0.3 µg/kg in children anesthetized with sevoflurane found that incidence of EA was reduced to 17 and 10% with 0.15 and 0.3 µg/kg respectively compared to 37% in the control group with comparable time to eye opening among the three groups. Also Sato et al. [19], showed that 0.3 µg/kg dexametomidine reduced the incidence of EA after sevoflurane anesthesia without tendency to delay post-operative recovery.

In contrast to our findings, Erdil et al. [31] administered a bolus dose of 0.5 µg/kg dexametomidine and found that times to extubation and eye opening were comparable to the control group. This could be explained by the different anesthetic protocol they used.

Postoperative pain alone doesn't cause agitation. [32,33] but, it is one of the confounding factors for this phenomenon [34,35]. This shows the importance of adequate postoperative pain management to help discriminating screaming due to pain from EA in children.

In the present study, the administration of acetaminophen suppository and infiltration of 25% bupivacaine at the site of surgical incision in all groups as a standard analgesic technique produced satisfactory postoperative pain relief and comparable OPS scores were recorded in the three groups. This finding suggests that the significantly higher PAED score and incidence of EA in the control group is unlikely to be related to pain.

In the present work, dexametomidine administration significantly reduced the endtidal sevoflurane concentration compared to the control group. This could be due to the anesthetic sparing effect of dexametomidine [36].

The hemodynamic effect of dexametomidine depends on the dose, rate and route of administration [37]. Dexametomidine, like other α2 agonists display a biphasic dose dependent response. High bolus doses initially result in a transient increase of blood pressure and reflex decrease in heart rate followed by a decrease in blood pressure. This hemodynamic effect is thought to be a combination of their central sympatholytic and peripheral vasoconstrictive effect [37,38]. Therefore in the current study dexametomidine at a dose of 0.3 and 0.5 µg/kg was administered slowly over 10 min to avoid unwanted hemodynamic effect of dexametomidine.

The present study demonstrated a significant reduction in MAP and HR in D2 group compared to its baseline and C group values. In D1 group administration of 0.3 µg/kg dexametomidine had no significant hemodynamic effect when compared to its baseline and to the control group. Inspite of the significant reduction in hemodynamic parameters in D2 group, it didn’t require medical interference. The more stable hemodynamics reported with the lower dose of 0.3 µg/kg dexametomidine in this study is comparable with the results found by Ibacache et al. [27]. Although Sato et al. [19] reported significant reduction in HR after administration of 0.3 µg/kg up to 30min postoperatively compared to placebo, this reduction had no clinical value and no children required treatment.

In accordance with the current results, Deutsch and Tobias [39] reported that administration of 0.5 µg/kg DEX over 5min resulted in 25% and 10% reduction in intraoperative HR and MAP respectively in children anesthetized with 1 MAC sevoflurane. They explained this pronounced effect on HR by the synergestic chronotropic effect of sevoflurane and dexametomidine. Erdil et al. [26] as well used 0.5 µg/kg DEX over 10 minutes during sevoflurane anesthesia in children and they reported a significant intraoperative reduction in MAP compared to its baseline value.

The respiratory effect of dexametomidine is controversial. Belleville et al. [40] reported an
increase in PaCO\textsubscript{2}, PtCO\textsubscript{2}, and short episodes of irregular breathing and apnea with higher doses of 1-2 µg/kg dexmedetomidine. On the other hand Hall et al. [11] and Venn et al. [41] stated that administration of dexmedetomidine had no respiratory effect. This discrepancy could be due to different doses and methods of administration in each study.

In the current study, dexmedetomidine had no significant respiratory effect compared to the control group. In accordance with this finding, previous researchers found that dexmedetomidine at doses up to 0.5 g/kg had no respiratory effect when administered during spontaneous breathing in children anesthetized with sevoflurane [19,39].

In conclusion, the administration of 0.3 and 0.5 µg/kg dexmedetomidine IV bolus reduced the incidence and severity of EA after sevoflurane anesthesia in preschool children. The lower dose of 0.3 µg/kg was not associated with delayed recovery or hemodynamic instability.

References


