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Original Research Article

Effect of Perioperative Dexmedetomidine Infusion on Incidence of Emergence Delirium in Pediatric Patients Undergoing Elective ENT Surgery: A Double-Blind Randomized Study

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Abstract

Background: Emergence delirium (ED) is a frequent and distressing complication following sevoflurane-based general anesthesia in pediatric patients undergoing ear, nose, and throat (ENT) surgery. Dexmedetomidine, a selective alpha-2 adrenergic agonist, has been proposed as a pharmacological strategy for reducing ED incidence; however, optimal dosing protocols and comprehensive hemodynamic safety data in the pediatric ENT population remain insufficiently characterized.

Methods: In this prospective, double-blind, randomized controlled trial, 120 children (ASA I–II) were randomly allocated to receive either dexmedetomidine (0.5 µg/kg loading dose followed by 0.2 µg/kg/h maintenance infusion; Group D, n = 60) or equivalent volume normal saline (Group S, n = 60). The primary outcome was ED incidence assessed using the Pediatric Anesthesia Emergence Delirium (PAED) scale. Secondary outcomes included ED severity scores, time to extubation, postoperative pain scores (FLACC scale), rescue analgesic requirements, and hemodynamic parameters.

Results: The incidence of ED was significantly lower in Group D compared to Group S (18.3% vs. 46.7%; $p < 0.001$). The mean peak PAED score was 8.42 ± 3.71 in Group D versus 13.86 ± 4.29 in Group S ($p < 0.001$). FLACC pain scores at 30 minutes postoperatively were significantly lower in Group D (2.18 ± 1.34 vs. 4.52 ± 1.87; $p < 0.001$). Time to extubation was modestly prolonged in Group D (9.73 ± 2.46 vs. 7.85 ± 2.12 min; $p = 0.003$). No clinically significant bradycardia or hypotension episodes requiring intervention were observed.

Conclusion: Perioperative dexmedetomidine infusion significantly reduces the incidence and severity of emergence delirium and postoperative pain in pediatric ENT surgery patients with an acceptable hemodynamic safety profile and minimal prolongation of extubation time.

Keywords: Dexmedetomidine; Emergence delirium; Pediatric anesthesia; Sevoflurane; ENT surgery; PAED scale.

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Introduction

Emergence delirium (ED) is a transient but clinically significant behavioral disturbance occurring during the early recovery phase from general anesthesia, characterized by inconsolable crying, disorientation, thrashing, and non-purposeful agitation [1].

The reported incidence of ED in pediatric populations ranges from 10% to 80%, with higher rates consistently observed following sevoflurane anesthesia and ENT procedures [2]. The phenomenon is particularly prevalent in preschool-aged children (2–6 years) and following surgical procedures involving the airway, such as adenotonsillectomy, myringotomy, and endoscopic sinus surgery [3]. The clinical consequences of ED extend beyond transient distress. Agitated children are at risk of self-injury, surgical site disruption, intravenous catheter dislodgement, and increased caregiver anxiety, all of which contribute to delayed post-anesthesia care unit (PACU) discharge and increased nursing workload [4]. Furthermore, emerging evidence suggests that severe ED episodes may be associated with maladaptive postoperative behavioral changes persisting for weeks after surgery [5].

Sevoflurane, despite its favorable pharmacokinetic profile for pediatric induction and maintenance, is recognized as an independent risk factor for ED,

possibly due to its rapid elimination generating a state of neural excitation during awakening [6]. This has prompted extensive investigation into pharmacological prevention strategies, including propofol, fentanyl, ketamine, midazolam, and alpha-2 adrenergic agonists [7].

Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist with sedative, anxiolytic, and analgesic properties, has attracted considerable interest as an ED-preventing agent [8]. Its mechanism of action involves modulation of noradrenergic transmission in the locus coeruleus, producing a state of cooperative sedation that mimics natural sleep, without significant respiratory depression [9]. Several randomized trials have demonstrated the efficacy of single-dose intravenous dexmedetomidine in reducing ED incidence in pediatric surgical populations [10]. However, intranasal and single bolus intravenous regimens may produce inconsistent plasma levels during the critical emergence period, particularly in longer ENT procedures [11].

The concept of perioperative continuous infusion—comprising a loading dose at induction followed by maintenance infusion throughout surgery—offers theoretically more stable drug concentrations during the emergence window. While this approach has been explored in adult populations, data specific to pediatric ENT surgery remain limited [12]. Moreover, concerns regarding dexmedetomidine-induced bradycardia and hypotension in pediatric patients necessitate careful hemodynamic characterization in this vulnerable population [13]. Additionally, the interplay between dexmedetomidine's analgesic properties and postoperative pain—a known contributor to ED—requires further elucidation in the ENT surgical context, where mucosal inflammation and referred otalgia are common [14].

Materials and Methods

Study Design and Setting: This prospective, double-blind, randomized, placebo-controlled trial was conducted at the Department of Anesthesiology.

Sample Size Calculation: Based on previous literature reporting ED incidence of approximately 45% with sevoflurane alone and an anticipated reduction to 18% with dexmedetomidine, with $\alpha = 0.05$ and power = 80%, the minimum required sample size was calculated as 52 per group. To account for potential dropouts, 60 patients per group (total N = 120) were enrolled.

Participants: Children aged 2–8 years, classified as American Society of Anesthesiologists (ASA) physical status I or II, scheduled for elective ENT surgery (adenoidectomy, tonsillectomy, adenotonsillectomy, or myringotomy with tympanostomy tube insertion) under general anesthesia were eligible for inclusion.

Exclusion Criteria Included: Known allergy or contraindication to dexmedetomidine; history of developmental delay, neurological disorders, or psychiatric illness; congenital heart disease or heart block of any degree; baseline heart rate below the 5th percentile for age; concurrent treatment with alpha-2 agonists, beta-blockers, or digoxin; anticipated difficult airway; and parental refusal.

Randomization and Blinding: Patients were randomly allocated to one of two groups using a computer-generated randomization sequence with block sizes of four, concealed in sequentially numbered opaque sealed envelopes. Group D (Dexmedetomidine group, n = 60) received dexmedetomidine 0.5 µg/kg as an intravenous loading dose over 10 minutes after induction, followed by a continuous infusion of 0.2 µg/kg/h maintained until the commencement of skin closure or the final surgical step. Group S (Saline group, n = 60) received equivalent volumes of 0.9% normal saline delivered identically in timing and appearance. Study solutions were prepared by a pharmacist not involved in patient care, in identical 50-mL syringes, ensuring that the anesthesiologists, surgeons, PACU nurses, and outcome assessors were blinded to group allocation throughout the study.

Anesthetic Protocol: All children received standard preoperative fasting instructions. No premedication was administered. In the operating room, standard ASA monitoring was applied, including electrocardiography, pulse oximetry, non-invasive blood pressure, capnography, and temperature monitoring. Anesthesia was induced with sevoflurane 8% in 100% oxygen via face mask.

Following intravenous cannulation, fentanyl 1 µg/kg and atracurium 0.5 mg/kg were administered to facilitate endotracheal intubation. Anesthesia was maintained with sevoflurane (end-tidal concentration 2.0–2.5%) in a mixture of oxygen and air (FiO_2 0.5). The study infusion was commenced immediately after induction. Intraoperative analgesia was supplemented with paracetamol 15 mg/kg intravenously. At surgical completion, sevoflurane was discontinued, the study infusion was stopped, and neuromuscular blockade was reversed with neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg. Extubation was performed upon return of protective airway reflexes and spontaneous ventilation meeting standard criteria.

Outcome Measures

Primary outcome: Incidence of emergence delirium, defined as a PAED scale score ≥ 10,

assessed at 5-minute intervals for 30 minutes after arrival in the PACU. The peak PAED score recorded during this period was used for analysis.

Secondary outcomes:

- Severity of ED (peak PAED score as a continuous variable)
- Time to extubation (minutes from sevoflurane discontinuation to extubation)
- Postoperative pain assessed using the Face, Legs, Activity, Cry, Consolability (FLACC) scale at 0, 15, and 30 minutes post-PACU arrival
- Rescue analgesic requirement (intravenous fentanyl 0.5 µg/kg administered for FLACC ≥ 4)
- Hemodynamic parameters (heart rate and mean arterial pressure) recorded at baseline, post-induction, post-loading dose, at 15-minute intraoperative intervals, and at 5-minute intervals in PACU for 30 minutes
- Adverse events: clinically significant bradycardia (heart rate < 60 bpm or > 20% decrease from baseline requiring atropine), hypotension (mean arterial pressure < 20% from baseline requiring fluid bolus or vasopressor), laryngospasm, postoperative nausea and vomiting (PONV), and oxygen desaturation (SpO_2 < 94%)

Statistical Analysis: Data were analyzed using IBM SPSS version 25.0. Normality was assessed using the Shapiro-Wilk test. Continuous variables were expressed as mean ± standard deviation (SD) and compared using independent samples t-test or Mann-Whitney U test as appropriate.

Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test. Repeated-measures ANOVA was used for hemodynamic parameter comparisons over time. A two-sided p-value < 0.05 was considered statistically significant.

Results

Patient Flow and Demographics: Of the 132 patients screened, 120 met eligibility criteria and were randomized (60 per group). Three patients in Group S were excluded post-randomization (two due to surgical plan change and one due to protocol deviation), leaving 60 in Group D and 57 in Group S for final analysis.

However, for consistency with the intention-to-treat principle, all 120 randomized patients were included in the primary analysis with last observation carried forward for the three excluded patients. The two groups were comparable in demographic and surgical characteristics (Table 1).

Table 1: Demographic and Surgical Characteristics of the Study Groups

Variable	Group D (n = 60)	Group S (n = 60)	p-value
Age (years), mean ± SD	4.82 ± 1.63	4.97 ± 1.71	0.623
Sex (Male/Female), n	34/26	37/23	0.582
Weight (kg), mean ± SD	17.45 ± 3.92	18.02 ± 4.18	0.439
ASA I/II, n	48/12	45/15	0.507
Type of surgery, n (%)			0.871
- Adenotonsillectomy	28 (46.7)	26 (43.3)	
- Tonsillectomy	14 (23.3)	16 (26.7)	
- Adenoidectomy	10 (16.7)	12 (20.0)	
- Myringotomy with tubes	8 (13.3)	6 (10.0)	
Duration of surgery (min), mean ± SD	38.64 ± 12.53	36.92 ± 11.87	0.439
Duration of anesthesia (min), mean ± SD	52.18 ± 13.67	50.43 ± 12.94	0.473

Primary and Secondary Outcomes: The incidence of ED (PAED ≥ 10) was significantly lower in Group D (18.3%) compared to Group S (46.7%; p < 0.001). The peak PAED score, FLACC pain scores, extubation time, and rescue analgesic requirements are presented in Table 2.

Table 2: Primary and Secondary Outcome Measures

Outcome	Group D (n = 60)	Group S (n = 60)	p-value
Incidence of ED (PAED ≥ 10), n (%)	11 (18.3)	28 (46.7)	<0.001*
Peak PAED score, mean ± SD	8.42 ± 3.71	13.86 ± 4.29	<0.001*
Time to extubation (min), mean ± SD	9.73 ± 2.46	7.85 ± 2.12	0.003*
FLACC score at 0 min, mean ± SD	2.85 ± 1.52	5.14 ± 2.03	<0.001*
FLACC score at 15 min, mean ± SD	2.43 ± 1.28	4.78 ± 1.92	<0.001*
FLACC score at 30 min, mean ± SD	2.18 ± 1.34	4.52 ± 1.87	<0.001*
Rescue fentanyl required, n (%)	9 (15.0)	27 (45.0)	<0.001*
PONV incidence, n (%)	5 (8.3)	8 (13.3)	0.384
PACU stay duration (min), mean ± SD	28.63 ± 7.24	34.17 ± 9.81	0.001*

***p < 0.05**

Hemodynamic Parameters: Hemodynamic data demonstrated that Group D exhibited lower mean heart rates and mean arterial pressures compared to Group S at most intraoperative and postoperative time points; however, these differences remained within clinically acceptable ranges (Table 3).

Table 3: Hemodynamic Parameters at Key Time Points

Time Point	Heart Rate (bpm)			Mean Arterial Pressure (mmHg)		
	Group D	Group S	p-value	Group D	Group S	p-value
Baseline	112.34 ± 14.28	110.87 ± 13.65	0.564	72.43 ± 7.16	73.12 ± 6.89	0.587
Post-loading dose	98.56 ± 12.43	108.24 ± 13.18	<0.001*	67.82 ± 6.53	71.48 ± 7.02	0.004*
15min intraoperative	95.72 ± 11.87	105.63 ± 12.94	<0.001*	65.94 ± 6.27	69.76 ± 6.84	0.002*
30min intraoperative	94.18 ± 10.92	104.37 ± 12.56	<0.001*	64.87 ± 5.98	68.92 ± 6.71	0.001*
PACU arrival	102.45 ± 13.16	118.73 ± 15.42	<0.001*	68.56 ± 6.43	74.28 ± 7.53	<0.001*
PACU 15 min	104.82 ± 12.73	114.56 ± 14.38	<0.001*	69.74 ± 6.18	72.63 ± 7.12	0.018*
PACU 30 min	106.94 ± 11.48	112.28 ± 13.27	0.021*	70.32 ± 5.94	71.87 ± 6.58	0.172

***p < 0.05**

No patient in either group required atropine for clinically significant bradycardia. One patient in Group D experienced transient heart rate decrease to 68 bpm, which resolved spontaneously without intervention. No episodes of hypotension requiring fluid bolus or vasopressor were recorded. Oxygen desaturation events were comparable (Group D: 2 [3.3%] vs. Group S: 3 [5.0%]; p = 1.000). No cases of laryngospasm occurred in either group.

Discussion

The present double-blind randomized trial demonstrates that perioperative dexmedetomidine infusion (0.5 µg/kg loading followed by 0.2 µg/kg/h maintenance) significantly reduces the incidence of emergence delirium from 46.7% to 18.3% in children undergoing elective ENT surgery under sevoflurane anesthesia, with concurrent reductions in ED severity, postoperative pain, rescue analgesic consumption, and PACU stay duration. The ED incidence of 46.7% observed in our control group is consistent with the range reported in the literature for sevoflurane-based anesthesia in pediatric ENT populations. Vlajkovic and Sindjelic reported ED rates of 40–55% in children receiving sevoflurane for adenotonsillectomy, attributing the high incidence to the combination of rapid sevoflurane washout and airway-related nociceptive stimulation during emergence [15]. The significant reduction achieved with dexmedetomidine in our study aligns with the meta-analytic findings of Dahmani et al., who reported a pooled relative risk reduction of approximately 60% with alpha-2 agonists compared to placebo for ED prevention [16].

The mechanistic basis for dexmedetomidine's efficacy in preventing ED is likely multifactorial. Its action on alpha-2 adrenoceptors in the locus coeruleus produces a state of sedation that closely mimics natural non-REM sleep, thereby facilitating a smoother transition from anesthesia to wakefulness [17]. Additionally, its analgesic properties—mediated through spinal cord dorsal horn alpha-2 receptors and supraspinal mechanisms—may attenuate the contribution of postoperative pain to agitation during emergence [18]. This dual mechanism is particularly relevant in ENT surgery, where the distinction between pain-related agitation and true ED is often clinically challenging [19].

The significantly lower FLACC scores across all postoperative time points in Group D support the analgesic contribution of dexmedetomidine. Sun et al. demonstrated that even subanesthetic doses of dexmedetomidine produce clinically meaningful reductions in postoperative opioid requirements in pediatric surgical patients [20]. In our study, only 15% of children in Group D required rescue fentanyl compared to 45% in Group S, representing a clinically and statistically significant opioid-sparing effect that carries implications for reducing opioid-related adverse effects such as nausea, vomiting, and respiratory depression. The modest prolongation of extubation time in Group D (approximately 1.9 minutes longer than Group S) is a recognized trade-off of dexmedetomidine sedation.

This finding is consistent with the observations of Kim et al., who reported a mean extubation delay of 1.5–3 minutes with intraoperative dexmedetomidine infusion in pediatric patients [21]. Importantly, this delay did not translate into adverse respiratory events or prolonged PACU stay; indeed, PACU duration was actually shorter in Group D, likely reflecting the reduced need for managing agitated, delirious children.

The hemodynamic profile observed in Group D—characterized by lower heart rates and mean arterial pressures relative to Group S—remained within physiologically acceptable limits throughout the perioperative period. This is concordant with the findings of Mason and Lerman, who emphasized that dexmedetomidine at doses ≤ 1 µg/kg produces predictable, dose-dependent hemodynamic effects that rarely necessitate pharmacological intervention in healthy pediatric patients [22]. The absence of clinically significant bradycardia or hypotension in

our cohort further supports the safety of the 0.5 μg/kg loading plus 0.2 μg/kg/h maintenance regimen for ASA I–II children.

Our use of continuous infusion rather than single bolus dosing represents a potential advantage over many prior studies. Hauber et al. demonstrated that single-dose dexmedetomidine administered at induction may produce subtherapeutic plasma levels by the time of emergence in procedures exceeding 30 minutes, potentially diminishing its ED-preventing efficacy [23]. The continuous infusion protocol employed in our study ensures sustained drug concentrations during the critical emergence window, which may account for the robust effect size observed.

The study's main strengths include its double-blind randomized design, standardized anesthetic protocol, use of validated outcome instruments (PAED and FLACC scales), and comprehensive hemodynamic monitoring.

However, several limitations warrant acknowledgment. First, the single-center design may limit generalizability. Second, the study was powered for the primary outcome of ED incidence rather than for rare adverse events, meaning that uncommon complications may be underdetected.

Third, we did not measure plasma dexmedetomidine concentrations, precluding pharmacokinetic-pharmacodynamic correlation. Fourth, the follow-up was limited to the PACU period, and long-term behavioral outcomes were not assessed. Finally, the inherent difficulty in distinguishing pain-related agitation from true ED in preverbal children remains a methodological challenge, despite the use of separate validated scales for each domain [24].

Future multicenter trials with larger sample sizes, extended follow-up periods encompassing postoperative behavioral assessments, dose-response analyses comparing different infusion rates, and pharmacokinetic sampling would further refine the evidence base for perioperative dexmedetomidine use in pediatric ENT anesthesia.

Conclusion

Perioperative intravenous dexmedetomidine infusion at a loading dose of 0.5 μg/kg followed by continuous maintenance at 0.2 μg/kg/h significantly reduces both the incidence and severity of emergence delirium in children aged 2–8 years undergoing elective ENT surgery under sevoflurane-based general anesthesia. The intervention concurrently provides meaningful postoperative analgesic benefits, evidenced by lower pain scores and reduced rescue opioid requirements, while shortening PACU stay duration.

The hemodynamic effects of this regimen remain within clinically acceptable limits, with no episodes of bradycardia or hypotension requiring therapeutic intervention. The modest prolongation of extubation time is clinically inconsequential and is offset by the substantial benefits in emergence quality and postoperative comfort. These findings support the routine consideration of perioperative dexmedetomidine infusion as a safe and effective pharmacological strategy for emergence delirium prevention in pediatric ENT surgical populations.

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