Original Research

A COMPARATIVE STUDY OF DEXMEDETOMIDINE AS BOLUS OR LOW-DOSE INFUSION FOR PREVENTION OF EMERGENCE AGITATION AFTER SEVOFLURANE IN PAEDIATRIC ANAESTHESIA

Sardar Bhagat Singh[1], Manmohan Jindal[2], Namrata Jain[3], Devendra Singh[4*], Neelima Tandon[5]

[1]Postgraduate Resident, Department of Anaesthesiology, Gajra Raja Medical College & J.A. Group of Hospitals, Gwalior, Madhya Pradesh, India.
[2]Professor, Department of Anaesthesiology, Gajra Raja Medical College & J.A. Group of Hospitals, Gwalior, Madhya Pradesh, India.
[3]Associate Professor, Department of Anaesthesiology, Gajra Raja Medical College & J.A. Group of Hospitals Gwalior, Madhya Pradesh, India.
[4*]Postgraduate Resident, Department of Anaesthesiology, Gajra Raja Medical College & J.A. Group of Hospitals, Gwalior, Madhya Pradesh, India.
[5]Professor, Department of Anaesthesiology, Gajra Raja Medical College & J.A. Group of Hospitals, Gwalior, Madhya Pradesh, India.
Corresponding Author: Devendra Singh
Postgraduate Resident, Department of Anaesthesiology, Gajra Raja Medical College & J.A. Group of Hospitals, Gwalior, Madhya Pradesh, India.
Email: drbhagatsinghmd@gmail.com

ABSTRACT

Background: Emergence agitation (EA) is a common and distressing complication following sevoflurane anaesthesia in paediatric patients, characterized by non-purposeful restlessness, crying, and disorientation. Dexmedetomidine, a highly selective α2-agonist, is frequently utilized to prevent EA. However, the optimal administration method, rapid intravenous bolus versus continuous intraoperative infusion, remains debated regarding the balance of efficacy and hemodynamic stability.

Aims and Objectives: To systematically compare the clinical effectiveness, postoperative recovery profile, and perioperative hemodynamic stability of a rapid bolus versus a continuous low-dose infusion of dexmedetomidine (0.3 µg/kg) in preventing emergence agitation in children undergoing elective abdominal and genitourinary surgeries under sevoflurane anaesthesia.

Materials and Methods: This prospective, randomized, double-blind, comparative study enrolled 80 paediatric patients (ASA physical status I–II, aged 2–12 years) randomly allocated into two equal groups (n=40). Group B received IV dexmedetomidine 0.3 µg/kg as a bolus over 10 minutes; Group I received a continuous infusion at 0.3 µg/kg/hour. Intraoperative Heart Rate and MAP were continuously monitored. Postoperative emergence agitation and pain were assessed using the PAED scale and Observational Pain Scores (OPS) in the PACU.

Results: Both techniques provided comparable hemodynamic stability. A transient, statistically significant decrease in MAP ($p=0.015$) was noted at 10 minutes post dexmedetomidine administration in group B, accompanied by a mild, non significant reduction in heart rate ($p=0.184$); all values remained within 20% of pre-induction baselines without requiring intervention. Group B demonstrated superior early analgesia (OPS on PACU arrival: 1.8 ± 1.2 vs 4.6 ± 1.8; $p<0.001$). Extubation times (6.2 ± 1.1 vs 6.5 ± 1.4 min; $p=0.284$) and emergence times (8.1 ± 1.5 vs 8.4 ± 1.8 min; $p=0.418$) were highly comparable.

Conclusion: A 0.3 µg/kg bolus of dexmedetomidine is hemodynamically safe and clinically superior to an equivalent continuous infusion. It provides good early postoperative analgesia and effectively mitigates sevoflurane-induced emergence agitation without extending recovery or extubation times.

Keywords: Dexmedetomidine, Emergence Agitation, Sevoflurane, Paediatric Anaesthesia, Bolus Vs Infusion, PAED Scale.

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INTRODUCTION

Undergoing general anaesthesia and surgical intervention is a profoundly distressing experience for paediatric patients. The perioperative period often precipitates abnormal postoperative behaviors, most notably emergence delirium (ED) or

emergence agitation (EA).[1,2] Inhaled induction of anaesthesia is a fundamental and commonly utilized method by paediatric anaesthesiologists, especially in situations where establishing intravenous access prior to induction is challenging or traumatic for the child.[1] Currently, sevoflurane is widely considered the volatile anaesthetic of choice for paediatric inhalational induction due to its low blood-gas partition coefficient, non-pungent odor, and minimal airway irritability, facilitating both rapid induction and rapid washout.[1,3]

However, this rapid washout and subsequent rapid awakening are frequently associated with a high incidence of emergence agitation.[4] EA is characterized by a spectrum of negative behaviors, including non-purposeful restlessness, thrashing, crying, moaning, disorientation and incoherence.[5] The incidence of EA following sevoflurane anaesthesia in paediatric populations is reported to range from 10% to 80%, typically manifesting within the first 30 to 45 minutes of recovery in the Post-anaesthesia Care Unit (PACU).[1] While generally self-limiting, severe agitation can lead to physical injury, surgical site disruption, increased nursing requirements, and immense parental anxiety. Furthermore, maladaptive behavioral changes can persist for days or weeks postoperatively, with multiple factors implicated including pain, anxiety, and patient characteristics.[6,7]

To mitigate this distress, various pharmacological agents have been prophylactically administered, including propofol,[8] midazolam,[2,9] ketamine,[6] and α-2 agonists such as clonidine.[10] However, these medications often introduce secondary complications such as prolonged sedation, delayed awakening, respiratory depression, and an increased incidence of postoperative nausea and vomiting (PONV).[11] Recently, there has been a significant shift toward the use of α2-adrenergic agonists, particularly dexmedetomidine, in paediatric anaesthesia. Dexmedetomidine is a highly selective central α2-agonist that provides a unique triad of anxiolysis, sedation, and analgesia without causing significant respiratory depression, reflecting its favorable safety profile.[12]

While the efficacy of dexmedetomidine in preventing EA is well-established, the optimal intraoperative dosing regimen, specifically the choice between a rapid intravenous bolus versus a continuous low-dose infusion, remains a subject of clinical research. Doses of 0.5 µg/kg and above have been shown to effectively reduce post-sevoflurane agitation but are frequently associated with undesirable central sympatholytic side effects, including significant reductions in heart rate (bradycardia), blood pressure (hypotension), and delayed extubation times, raising concerns regarding overall recovery dynamics.[13,3] Therefore, this prospective, randomized, double-blind study was designed to evaluate and compare the efficacy, hemodynamic safety, and postoperative recovery profile of a conservatively dosed (0.3 µg/kg) dexmedetomidine regimen, administered either as a bolus or a continuous infusion, for the prevention of EA in paediatric patients undergoing elective abdominal and genitourinary surgeries under sevoflurane anaesthesia.

MATERIALS AND METHODS

This prospective, randomized, double-blind, comparative clinical study was conducted in the Department of Anaesthesiology at the Superspeciality Block, Gajra Raja Medical College and JAH Group of Hospitals, Gwalior. The study protocol was formally reviewed and approved by the Institutional Ethics Committee prior to the commencement of patient enrollment. Detailed written informed consent was systematically obtained from the parents or legal guardians of all participating children after a thorough explanation of the study's purpose, potential risks, and benefits.

Study Population

A total of 80 paediatric patients were enrolled to achieve the calculated sample size (n=40 per group) required for adequate statistical power. The inclusion criteria comprised children aged between 2 and 12 years, belonging to the American Society of anaesthesiologists (ASA) Physical Status I and II, who were scheduled for elective abdominal and genitourinary surgical procedures under general anaesthesia. To ensure pharmacological uniformity and minimize pharmacokinetic variability, only patients presenting with an actual body weight strictly within ± 20% of their ideal body weight were included.

Patients were excluded from the study if their parents/guardians refused consent, if they belonged to ASA physical status III or IV, or if they presented with contraindications to general anaesthesia. Furthermore, children with a documented history of cardiac disease, developmental delay, psychological disorders, epilepsy, neurological deficits, known allergy to dexmedetomidine, malignancy, severe gastrointestinal bleeding, or significant organ dysfunction were excluded.

Randomization and Blinding

Following enrollment, patients were randomly allocated into two equal groups using a computer-generated randomization sequence:

Group B (Bolus Group, n=40): Patients received intravenous dexmedetomidine at 0.3 µg/kg as a slow, controlled bolus over 10 minutes following successful orotracheal intubation.

Group I (Infusion Group, n=40): Patients received intravenous dexmedetomidine at 0.3 µg/kg/hour as a continuous infusion throughout surgery, initiated

immediately following successful orotracheal intubation.

To ensure scientific validity, the study maintained a strict double-blind design. The dexmedetomidine solutions were prepared in identical, unlabeled syringes by an independent anaesthesiologist not involved in intraoperative management or postoperative data collection. Both the attending anaesthesiologist and the independent observer recording PACU data were entirely blinded to group allocation.

anaesthetic Management and Study Protocol

All participating patients were maintained strictly nil per oral (NPO) preoperatively per institutional paediatric fasting guidelines. Upon arrival, a secure peripheral venous access was established and IV Ringer's Lactate initiated at 10 ml/kg/hour. Premedication consisted of IV Glycopyrrolate (0.005 mg/kg) and IV Midazolam (0.05 mg/kg).

Standard ASA physiological monitors were attached including continuous ECG, pulse oximetry (SpO_2), and non-invasive arterial blood pressure (NIBP). General anaesthesia was induced via inhalational technique utilizing 8% inspired Sevoflurane in 100% oxygen via a paediatric facemask. Following loss of consciousness, IV Fentanyl (1 µg/kg) and Atracurium (0.5 mg/kg) were administered. Maintenance was achieved with 60% Nitrous Oxide / 40% Oxygen supplemented with 2%–3% end-tidal Sevoflurane. End-tidal CO_2 was maintained between 35 - 45 mmHg. The study drug was administered immediately following airway securement per group allocation.

Observations and Postoperative Assessment

Intraoperative HR, MAP, and SpO_2 were recorded at predefined intervals: before induction, after intubation, at skin incision, 10 and 30 minutes post-dexmedetomidine, at skin closure, and at discontinuation of the inhalational agent. Intraoperative hemodynamic deviations exceeding 20% from baseline were managed with targeted rescue therapies. Tachycardia (a >20% increase from baseline) was managed with rescue IV fentanyl. Bradycardia (a >20% decrease from baseline) was treated with IV atropine. Hypertension (a >20% increase from baseline) was managed with rescue IV fentanyl. Hypotension (a >20% decrease from baseline) was treated with IV fluid boluses. Following surgery, Sevoflurane and Nitrous Oxide were discontinued and residual neuromuscular blockade was reversed with IV Neostigmine and Glycopyrrolate combination (0.08 mg/kg). Patients were extubated after satisfying standard clinical criteria. Postoperatively, patients were observed in the PACU for 60 minutes. Emergence agitation was assessed using the paediatric anaesthesia Emergence Delirium (PAED) scale and pain using Observational Pain Scores (OPS) upon PACU arrival and at 15, 30, and 60 minutes. Rescue analgesia (IV Paracetamol 10 mg/kg) was administered for OPS ≥ 4 or severe restlessness. Time to Aldrete Discharge Score ≥ 9 was also documented.

RESULTS

A total of 80 paediatric patients successfully completed the study protocol without any dropouts, yielding complete datasets for both Group B (n=40) and Group I (n=40). Demographic profiles were highly comparable between the two groups, with no significant differences in age, body weight, ASA physical status distribution, or total duration of surgery (all $p > 0.05$), ensuring a well-matched cohort.

Table 1: Summary of Demographics and Recovery Characteristics

Parameter	Group B (N=40)	Group I (N=40)	P-Value
Age (years)	6.2 ± 2.1	6.4 ± 2.3	0.672
Weight (kg)	18.5 ± 4.2	19.1 ± 4.5	0.541
Extubation Time (min)	6.2 ± 1.1	6.5 ± 1.4	0.284
Emergence Time (min)	8.1 ± 1.5	8.4 ± 1.8	0.418
Time to Aldrete ≥ 9 (min)	12.4 ± 2.1	12.6 ± 2.4	0.687
OPS on PACU Arrival	1.8 ± 1.2	4.6 ± 1.8	< 0.001*

* Highly significant | Values expressed as Mean ± SD

Intraoperative Hemodynamic Profile

The comparative analysis of intraoperative hemodynamics revealed that both administration techniques provided exceptional stability, safely navigating the recognized sympatholytic risks associated with central α2-agonists. Baseline MAP and Heart Rate prior to induction were comparable. Following intubation, both groups exhibited the anticipated physiological sympathetic stress response with no significant intergroup divergence.

A notable pharmacokinetic divergence manifested 10 minutes following the administration of the study drug. A transient, statistically significant decrease in MAP was recorded in the bolus group (Group B: 71.5 ± 4.8 mmHg) compared to the continuous infusion group (Group I: 74.2 ± 4.9 mmHg) ($p = 0.015$). A concordant sharp decline in heart rate was

also observed (Group B: 100.1 ± 6.2 beats/min vs. Group I: 102.0 ± 7.1 beats/min; p = 0.184).

Despite the sharper and more rapid initial sympatholytic effect observed with the bolus technique, the hemodynamic parameters (MAP and HR) for all patients in both groups remained safely within the predefined threshold of ± 20% of their respective pre-induction baseline values. No patient in either cohort met the clinical criteria for severe bradycardia or profound hypotension; consequently, no patient required pharmacological rescue interventions. By the time of skin closure and discontinuation of the volatile agent, both MAP and HR had returned to baseline and remained stable and comparable throughout the entire 60-minute PACU observation period.

Table 2: Comparison of Heart Rate (beats/min)

Time Interval	Group B	Group I	P-Value
Before induction	105.4 ± 8.2	106.1 ± 7.9	0.697
After intubation	114.5 ± 9.1	115.8 ± 8.8	0.519
At skin incision	110.2 ± 8.5	112.0 ± 8.2	0.334
10 mins post-dexmedetomidine	100.1 ± 6.2	102.0 ± 7.1	0.184
30 mins post-dexmedetomidine	97.0 ± 6.7	100.1 ± 6.9	0.052
At skin closure	104.5 ± 7.5	106.8 ± 7.8	0.181
Discontinuation of agent	108.2 ± 8.0	110.5 ± 8.4	0.213
On arrival to PACU	101.5 ± 6.5	102.8 ± 6.9	0.384
Post-op 15 min	100.8 ± 6.2	101.5 ± 6.5	0.622
Post-op 30 min	102.5 ± 6.0	103.2 ± 6.1	0.605
Post-op 60 min	104.8 ± 5.8	105.5 ± 5.9	0.592

Values expressed as Mean ± SD | $p < 0.05$ considered significant

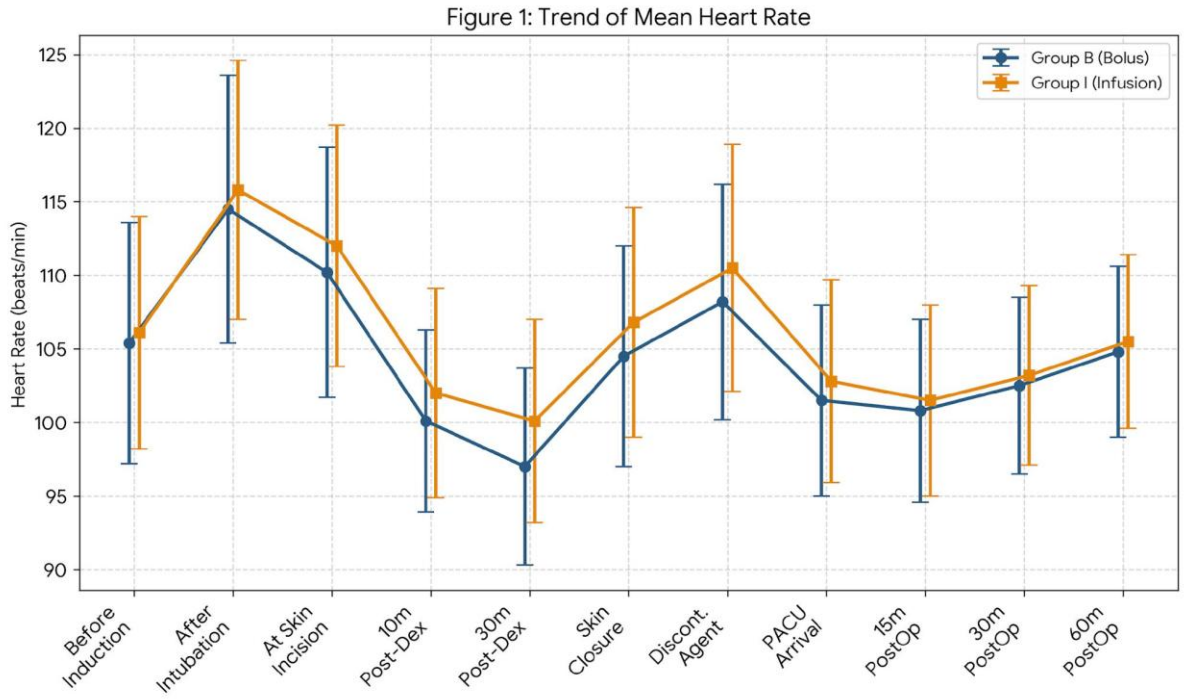


Figure 1: Heart Rate Trends across Time Intervals for Group B and Group I

Table 3: Comparison of Mean Arterial Pressure (mmHg)

Time Interval	Group B	Group I	P-Value
Before induction	76.5 ± 5.2	75.8 ± 5.5	0.558
After intubation	85.2 ± 6.1	86.1 ± 5.9	0.485
At skin incision	82.1 ± 5.5	83.0 ± 5.2	0.450
10 mins post-dexmedetomidine	71.5 ± 4.8	74.2 ± 4.9	0.015*
30 mins post-dexmedetomidine	69.8 ± 4.5	72.1 ± 4.6	0.094
At skin closure	75.0 ± 4.9	76.2 ± 5.1	0.280

Discontinuation of agent	78.2 ± 5.1	79.5 ± 5.4	0.270
On arrival to PACU	75.5 ± 4.8	76.8 ± 4.9	0.235
Post-op 15 min	74.8 ± 4.7	75.5 ± 4.5	0.501
Post-op 30 min	75.5 ± 4.9	76.2 ± 4.8	0.521
Post-op 60 min	76.8 ± 5.0	77.5 ± 5.2	0.538

* Statistically significant ($p < 0.05$) | Values expressed as Mean ± SD

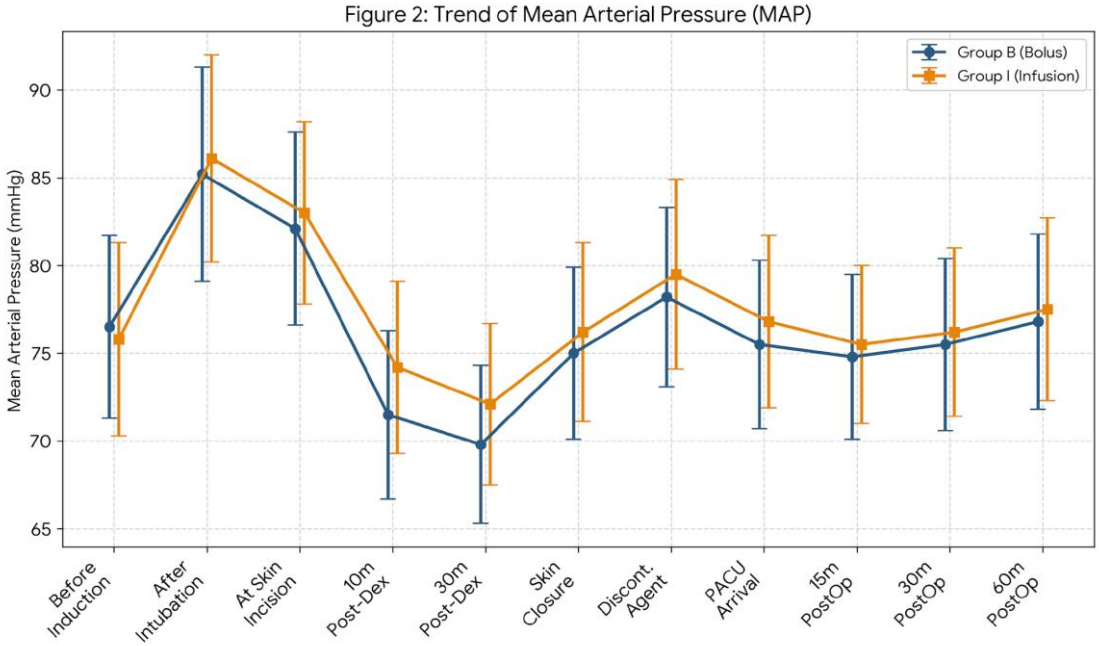


Figure 2: Mean Arterial Pressure trends across time intervals for Group B and Group I

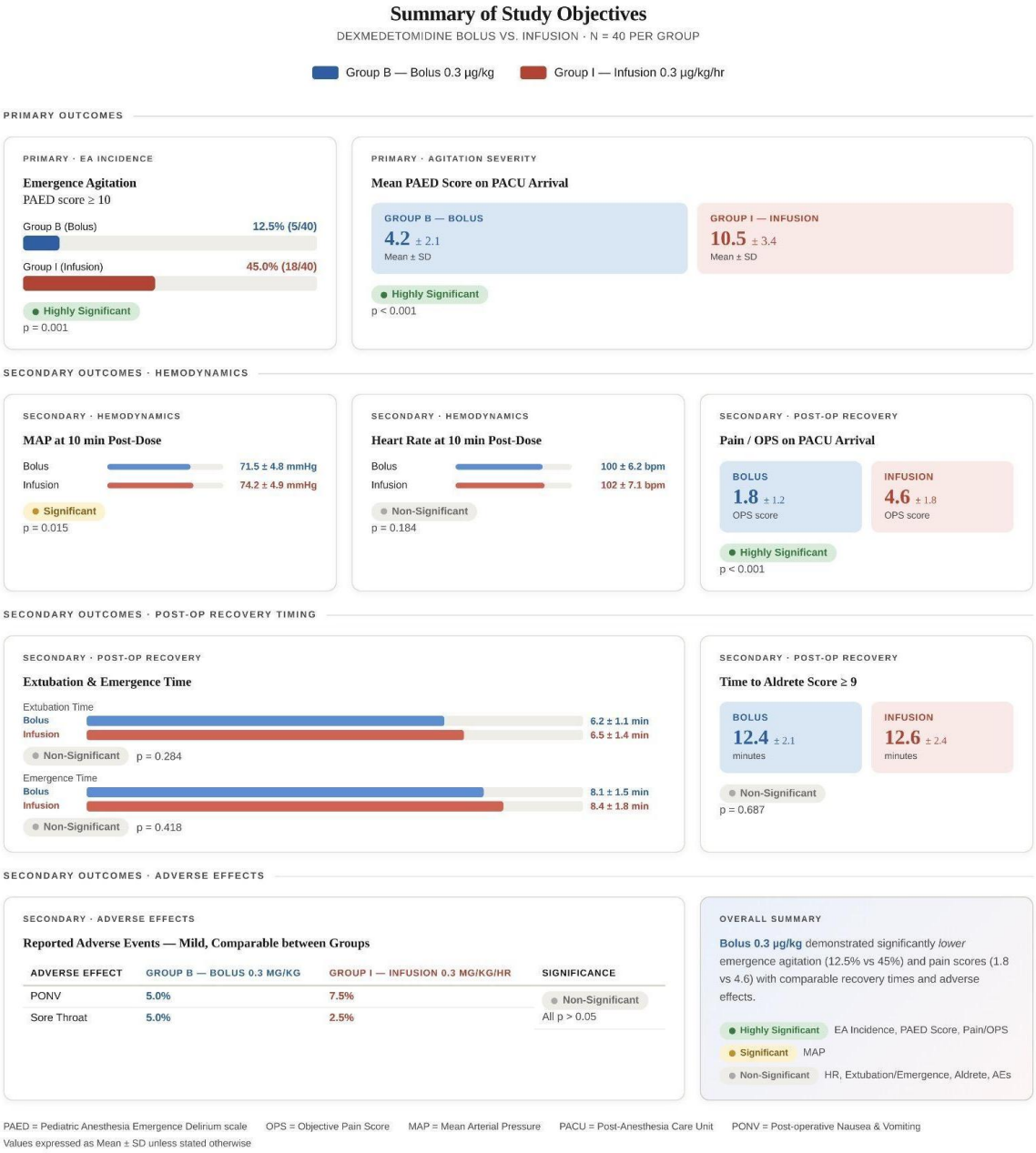
Postoperative Recovery and Agitation Assessment

Table 4: Summary of Study Objectives and Outcomes

Study Objective & Parameter	Group B (Bolus 0.3 µg/kg)	Group I (Infusion 0.3 µg/kg/hr)	Statistical Significance	Clinical Inference
PRIMARY: Emergence Agitation Incidence (PAED score ≥ 10)	12.5% (5/40 patients)	45.0% (18/40 patients)	p = 0.001	Bolus significantly more effective at preventing emergence agitation.
PRIMARY: Agitation Severity (Mean PAED on PACU Arrival)	4.2 ± 2.1	10.5 ± 3.4	p < 0.001	Bolus patients calm; infusion patients crossed clinical agitation threshold.
SECONDARY: Hemodynamics — MAP at 10 mins post-dose	71.5 ± 4.8 mmHg	74.2 ± 4.9 mmHg	p = 0.015*	Transient MAP drop with bolus; values within safe 20% physiological threshold.
SECONDARY: Hemodynamics — Heart Rate at 10 mins post-dose	100 ± 6.2 bpm	102 ± 7.1 bpm	p = 0.184	Slightly stronger sympatholytic effect with bolus; not clinically significant.
SECONDARY: Post-op Pain — OPS on PACU Arrival	1.8 ± 1.2	4.6 ± 1.8	p < 0.001	Superior and more rapid onset of early postoperative analgesia with bolus.

SECONDARY: Post-op Recovery — Extubation / Emergence Time	Extubation: 6.2 ± 1.1 min Emergence: 8.1 ± 1.5 min	Extubation: 6.5 ± 1.4 min Emergence: 8.4 ± 1.8 min	p = 0.284 p = 0.418	Administration route does not delay immediate recovery profile in OR.
SECONDARY: Post-op Recovery — Time to Aldrete Score ≥ 9	12.4 ± 2.1 min	12.6 ± 2.4 min	p = 0.687	Both techniques provide comparable PACU discharge readiness.
SECONDARY: Adverse Effects	PONV 5.0% Sore Throat 5.0%	PONV 7.5% Sore Throat 2.5%	All p > 0.05	Highly favorable safety profile. No severe bradycardia, hypotension, or respiratory depression recorded.

* Statistically significant | p < 0.001 considered Highly Significant



As shown in Table 4, the analysis of postoperative recovery metrics demonstrated the clinical efficacy of the 0.3 μg/kg bolus dose. Patients in Group B exhibited significantly superior early postoperative analgesia and a smoother emergence profile compared to their counterparts in Group I. Specifically, the OPS recorded immediately upon arrival in the PACU were markedly lower in the bolus group (1.8 ± 1.2) compared to the infusion group (4.6 ± 1.8), representing a highly significant statistical difference ($p < 0.001$). This early analgesic coverage effectively suppressed the incidence and severity of emergence agitation, as reflected by parallel improvements in the early PAED scores.

Despite the potent central sedative and analgesic effects provided by the dexmedetomidine bolus, it did not compromise the speed of physiological recovery. The precise tracking of recovery timelines confirmed that Extubation Time was small and highly comparable between the two cohorts (Group B: 6.2 ± 1.1 min vs. Group I: 6.5 ± 1.4 min; $p = 0.284$). Similarly, Emergence Time demonstrated no significant delay (Group B: 8.1 ± 1.5 min vs. Group I: 8.4 ± 1.8 min; $p = 0.418$). The total duration from PACU admission until Aldrete Score ≥ 9 was statistically identical (Group B: 12.4 ± 2.1 min vs. Group I: 12.6 ± 2.4 min; $p = 0.687$).

The incidence of postoperative adverse effects, including nausea/vomiting, drowsiness, respiratory depression, and sore throat, was recorded and found to be clinically negligible, with no statistically significant differences observed between the two study groups.

DISCUSSION

Sevoflurane is widely used in paediatric anaesthetic practice due to its rapid onset and offset. Its excitatory washout phase can precipitate non-purposeful restlessness, thrashing and inconsolable crying, collectively termed emergence agitation (EA). The incidence of EA ranges from 10% to 80%, frequently occurring before adequate pain control is established. Cravero et al.[4] demonstrated a significantly higher incidence of EA with sevoflurane compared to halothane, even without surgical stimulation. Aono et al.[45] corroborated this with an age-dependent, agent-specific effect, while Vlajkovic and Sindjelic[41] confirmed that pain is not the sole cause, as EA occurs in pain-free scenarios such as MRI or eye examinations.

Emergence agitation is a dissociative state of consciousness occurring within the first 30–45 minutes of recovery. While self-limiting, it carries significant clinical risks. Several pharmacological interventions have been employed for its management, with dexmedetomidine emerging as a particularly effective option. Acting on presynaptic α2 receptors in the locus coeruleus, it reduces central sympathetic outflow and facilitates inhibitory GABAergic activity. Tang et al.[16], in a meta-analysis of over 3,000 paediatric patients, demonstrated a marked reduction in emergence agitation incidence with dexmedetomidine. Rao et al.[20] and Dahmani et al.[39] further confirmed its superiority over placebo, midazolam, and opioids.

Higher doses, commonly ranging from 0.5 to 1.0 μg/kg in the literature, risk undesirable sympatholytic side effects including prolonged sedation, delayed extubation, bradycardia, and hypotension. To optimize the balance between prophylactic efficacy and hemodynamic safety, this study utilized a reduced dose of 0.3 μg/kg, delivered either as a rapid bolus or a continuous intraoperative infusion in 80 paediatric patients aged 2–12 years undergoing elective lower abdominal and genitourinary surgeries.

Both study groups were well-matched across all baseline demographic parameters, including age, weight, gender, and ASA physical status, ensuring the validity of clinical outcomes and eliminating selection bias. The paediatric age range of 2–12 years are the most susceptible demographic for sevoflurane-induced EA, consistent with approaches taken by Begum et al.[22], Ibrahim et al.[15] and Aono et al.[45] highlighted that emergence agitation under sevoflurane is significantly higher in preschool boys compared to school-age children, underscoring the importance of this demographic focus. The homogeneous distribution of ASA Grade I and II patients between groups aligns with the practices of Guler et al.[13] and Shukry et al.[43], who similarly selected fundamentally healthy patients to accurately assess the isolated physiological effects of α2 agonists.

Dexmedetomidine's inherent sympatholytic properties predictably modulate mean arterial pressure (MAP) and heart rate (HR), making continuous hemodynamic monitoring a critical safety metric. Baseline MAP and HR values were comparable between the two groups prior to induction. Following intubation, both cohorts exhibited an expected transient sympathetic response with no significant intergroup difference.

At 10 minutes post-administration, the bolus group demonstrated a more pronounced transient fall in MAP compared to the infusion group, reflecting rapid saturation of central α2-receptors from the concentrated bolus delivery. This sympatholytic divergence persisted to the 30-minute mark, after which hemodynamics fully stabilized and remained comparable between both groups throughout the 60-minute PACU observation period. Crucially, MAP in all patients remained safely within 20% of pre-induction baseline values, and no patient required intravenous fluid resuscitation, anticholinergic agents, or vasopressor interventions.

These findings validate the safety of capping the dexmedetomidine dose at 0.3 μg/kg. Begum et al.[22], using a slightly higher 0.4 μg/kg, similarly reported stable hemodynamics with only a transient fall in the bolus group. In contrast, higher doses in the literature consistently demonstrate severe cardiovascular consequences: Patel et al.[38], using a 2 μg/kg loading dose, encountered profound blood pressure and heart rate reductions requiring intervention, while Petroz et al.[42] confirmed a predictable dose- and time-dependent hemodynamic depression at infusion rates up to 6 μg/kg/hr. Hauber et al.[30] further documented a biphasic blood pressure response to rapid higher-dose bolus administration, strongly supporting restriction to 0.3 μg/kg. Conversely, Aksu et al.[40] demonstrated that controlled sympatholysis at moderate doses can be advantageous in blunting the tachycardic response during extubation.

Heart rate trends followed a similar pattern. Following intubation-related sympathetic stimulation, dexmedetomidine produced a smooth, controlled decline in both groups, consistent with suppression of norepinephrine release from the locus coeruleus. The lowest recorded mean heart rates remained safely within 20% of pre-induction baselines. No patient met the criteria for clinical bradycardia, and no anticholinergic interventions were required. This chronotropic stability, whether via bolus or infusion, validates the safety of the 0.3 μg/kg threshold and contrasts sharply with higher-dose protocols where bradycardia requiring atropine has been reported.

Surgical duration was strictly comparable between groups, with a clinically negligible intergroup difference of approximately three minutes. This equivalence eliminates surgical duration as a confounding variable, ensuring that differences in postoperative recovery outcomes can be directly attributed to the pharmacokinetics of the two delivery techniques rather than variations in surgical stress or sevoflurane tissue accumulation. Zhang et al.[24] confirmed that emergence agitation under sevoflurane is primarily an agent-specific and neurophysiological phenomenon rather than a direct function of surgical duration. Similarly, anaesthesia duration was comparable between groups, confirming that neither cohort was subjected to prolonged volatile anaesthetic exposure, consistent with findings from Zhu M et al.[31] and Amorim et al.[28]

A recognized clinical concern with intraoperative dexmedetomidine is its potential to delay awakening and operating room turnover. Our findings demonstrate that a 0.3 μg/kg dose, irrespective of delivery technique, does not compromise the rapid offset profile that makes sevoflurane advantageous. Mean extubation and emergence times were nearly identical between the bolus and infusion groups, with both groups achieving PACU discharge readiness within approximately 12.5 minutes. These results align with Begum et al.[22] and Ibacache et al.[44], who likewise found no clinically meaningful recovery delay at comparable low doses.

In contrast, Zhu M et al.[31], Amorim et al.[28] and Shi et al.[23] reported statistically significant but minor prolongations in recovery kinetics with dexmedetomidine at higher doses. Ramachandran et al.[19] importantly demonstrated that a lower infusion dose of 0.3 μg/kg/h is as effective as 0.5 μg/kg/h without unnecessarily deepening sedation, further supporting the conservative dosing strategy employed in this study.

The primary objective of this study was to quantify EA using the Paediatric Anaesthesia emergence agitation (PAED) scale, with a score of ≥10 as the clinical threshold. The bolus technique demonstrated markedly superior efficacy. The incidence of clinical agitation in the bolus group was substantially lower than in the infusion group, with the bolus cohort maintaining PAED scores well below the agitation threshold upon PACU arrival and throughout the 60-minute observation period. These findings strongly suggest that pre-treatment with a 10-minute bolus achieves rapid saturation of central α2-receptors precisely at the moment of sevoflurane washout, preventing the activation of excitatory pathways. In contrast, the continuous infusion technique may not achieve sufficient peak plasma concentrations at the critical moment of extubation.

These outcomes closely align with Begum et al.[22], who reported substantially lower EA incidence in the bolus group compared to the infusion group. Ibacache et al.[44] confirmed that a single 0.3 μg/kg intravenous dose significantly reduces EA incidence compared to control, and Zhang et al.[24] calculated the ED95 of dexmedetomidine for EA prevention at exactly 0.30 μg/kg over 10 minutes, directly validating the bolus dosing employed in this study. Ali and Abdellatif[37] further demonstrated that this dose is significantly more effective than propofol for both the incidence and severity of EA. The broader literature universally supports dexmedetomidine's superior prophylactic efficacy: Costi D et al.[33], Tang et al.[16] and Rao et al.[20] each confirmed lesser emergence agitation across large meta-analyses. Pickard et al.[35]'s meta-analysis also confirmed that increasing the dose beyond modest thresholds does not yield additional agitation prophylaxis, reinforcing the use of the lowest effective dose.

Differentiating true emergence agitation from postoperative pain is clinically challenging, as the presentations overlap significantly in pre-verbal and distressed paediatric patients. The Observational Pain Score (OPS) was used to objectively quantify analgesic efficacy. The bolus group demonstrated markedly lower pain scores upon PACU arrival, an

advantage that persisted throughout the 60-minute observation period, though the gap narrowed over time. This superior early analgesic profile reflects the higher peak plasma concentrations achieved by the bolus technique at the conclusion of surgery.

Begum et al.[22], using a structurally identical comparison at 0.4 µg/kg, similarly found that the bolus method yields lower observational pain scores compared to continuous infusion. The broader analgesic potency of dexmedetomidine is validated by Yang et al.[21]'s meta-analysis, which showed a significant reduction in rescue analgesic requirements. Mathur et al.[26] and Golmohammadi et al.[17] both confirmed lower postoperative pain scores in dexmedetomidine groups compared to controls. The opioid-sparing effect documented by Hauber et al.[30] further supports the bolus approach, while Kim et al.[34] demonstrated that intraoperative dexmedetomidine meaningfully reduces the end-tidal sevoflurane concentration required for adequate anaesthesia, likely contributing to a smoother and less painful emergence.

Prolonged PACU stay is a recognized clinical drawback of intraoperative dexmedetomidine, particularly in paediatric ambulatory settings. Recovery was tracked using the modified Aldrete Scoring System, with a score of ≥9 required for safe ward discharge. The time to achieve this threshold was virtually identical between the bolus and infusion groups confirming that the 0.3 µg/kg dose avoids recovery delay regardless of delivery technique. These results are consistent with Begum et al.[22], Ibacache et al.[44] and Shukry et al.[43], who similarly observed no significant discharge delay at comparable doses.

Zhu M et al.[31] and Costi D et al.[33] identified minor statistically significant prolongations in PACU stay with dexmedetomidine, but both concluded that such small delays are clinically irrelevant when weighed against the substantial reduction in emergence agitation. Pickard et al.[35] similarly noted only a minor mean increase in PACU time compared to placebo, deemed clinically non-significant. Notably, Ali and Abdellatif[37] demonstrated that a 0.3 µg/kg bolus of dexmedetomidine resulted in shorter emergence times compared to a propofol bolus, highlighting a favorable recovery profile at this dose.

No clinically significant hypotension, hypertension or severe bradycardia was recorded in either group. Mild tachycardia and postoperative nausea and vomiting (PONV) occurred at low, statistically comparable rates between groups. Respiratory depression, defined as oxygen saturation below 90% on room air, was absent in both cohorts. Postoperative sore throat and drowsiness were self-limiting and equivalent between groups.

The absence of severe complications aligns with the broader literature. Yang et al.[21] and Zhu M et al.[31] confirmed dexmedetomidine's potent antiemetic properties in large paediatric meta-analyses, likely attributable to opioid-sparing effects and reduced sympathetic outflow. Regarding respiratory safety, Petroz et al.[42] demonstrated minimal respiratory depression even at substantially higher infusion doses in children, while Thomas et al. reported no desaturation events. No incidence of bradycardia and hypotension in our cohort contrasts with the significant cardiovascular depression encountered in high-dose protocols, confirming that the 0.3 µg/kg threshold secures the analgesic and anti-delirium benefits of dexmedetomidine while avoiding dose-dependent hemodynamic penalties.

For paediatric lower abdominal and genitourinary surgeries conducted under sevoflurane anaesthesia, administration of 0.3 µg/kg dexmedetomidine as a 10-minute bolus provides a clinically superior, hemodynamically stable, and practically advantageous approach compared to continuous infusion. The bolus technique achieves rapid central α2-receptor saturation at the critical moment of volatile anaesthetic washout, delivering superior emergence agitation prophylaxis and early postoperative analgesia while ensuring no delay in operating room turnover or PACU discharge. This low-dose bolus protocol represents an optimal balance between prophylactic efficacy and cardiovascular safety in routine paediatric anaesthetic practice.

CONCLUSION

Based on the comprehensive findings of this prospective, randomized, double-blind study, we conclude that the administration of a conservative 0.3 µg/kg rapid intravenous bolus of dexmedetomidine is a hemodynamically safe, highly predictable, and clinically superior technique for the prevention of sevoflurane-induced emergence agitation in paediatric patients compared to an equivalent continuous infusion. The bolus technique safely harnesses the potent anxiolytic and analgesic properties of dexmedetomidine to provide exceptional early postoperative comfort and agitation mitigation, without causing pathological cardiovascular depression, prolonging extubation times, or delaying overall physiological recovery.

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