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ORIGINAL RESEARCH ARTICLE

Sedation Profile and Safety of Intravenous Dexmedetomidine for Procedural Sedation During MRI Brain in Paediatric Patients: A Prospective Observational Study

Thengujam Ashapriya Devi[1*] | Rupali N. Gorgile[2] | Pawal Avinash Tukaram[3]

Abstract

Background: Procedural sedation for MRI brain in children demands an agent ensuring complete immobility, respiratory preservation, and rapid recovery. Dexmedetomidine, a selective α-2 adrenoceptor agonist producing natural sleep-like sedation, represents a compelling candidate for this indication.

Objectives: To assess the sedation profile and safety of intravenous dexmedetomidine (loading dose 1 mcg/kg over 10 minutes + maintenance 0.7–1.0 mcg/kg/hr) for procedural sedation during MRI brain in paediatric patients aged 1–10 years.

Methods: This prospective observational study enrolled 52 paediatric patients (ASA Grade I–II). Primary outcomes included time to onset of adequate sedation (Ramsay Sedation Score ≥ 4), recovery time, MRI image quality score, and requirement for propofol rescue. Serial haemodynamic parameters and adverse events were recorded at 10-minute intervals. Statistical analysis employed Friedman test, Wilcoxon signed-rank test, and Kruskal-Wallis test; $p < 0.05$ was considered significant.

Results: Mean age was 5.83 $\pm$ 2.17 years. Sedation onset was 6.59 $\pm$ 0.99 minutes with recovery at 11.25 $\pm$ 1.61 minutes. Optimal MRI quality score 1 was achieved in 47 patients (90.4%); 5 patients (9.6%) required propofol rescue and achieved MRI quality score 2. Procedural success rate was 100% with zero repeat scans. Heart rate declined significantly from 111.58 $\pm$ 9.00 to 94.92 $\pm$ 8.18 bpm at 20 minutes (Friedman $\chi^2 = 312.0$, $p < 0.001$). SpO_2 remained clinically stable throughout the procedure, with no desaturation events. Adverse events were limited to 2 cases (3.8%) of self-limiting bradycardia.

Conclusion: Intravenous dexmedetomidine provides effective, safe, and rapidly reversible sedation for paediatric MRI brain with a 100% procedural success rate and no observed respiratory adverse events.

Key words: Dexmedetomidine, paediatric sedation, MRI brain, Ramsay Sedation Score, procedural sedation, respiratory preservation

1 | INTRODUCTION

Magnetic resonance imaging (MRI) has emerged as the imaging modality of choice for a wide spectrum of paediatric neurological conditions, offering superior soft-tissue contrast resolution without ionising radiation. However, the unique challenges of the MRI environment — including prolonged scan durations, confined bore space, acoustic noise exceeding 100 decibels, and the absolute requirement for patient immobility — render voluntary cooperation largely unachievable in young children. Consequently, procedural sedation or general anaesthesia is required in the majority of children under 6 years of age and in a significant proportion of older children with neurode-

[1]Senior Resident, Department of Anaesthesiology, Government Medical College Miraj, Maharashtra, India..

[2]Associate Professor, Department of Anaesthesiology, Chhatrapati Sambhaji Maharaj Government Medical College Satara, Maharashtra, India..

[3]Senior Resident, Department of Anaesthesiology, Government Medical College Miraj, Maharashtra, India..

Address correspondence to: Thengujam Ashapriya Devi, Senior Resident, Department of Anaesthesiology, Government Medical College Miraj, Maharashtra, India., *Email: apthengujam@gmail.com*

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velopmental disorders, anxiety, or cognitive impairment (1, 2).

The ideal paediatric sedative for MRI must fulfil several stringent criteria: rapid and predictable onset, adequate depth of sedation to ensure complete immobility, preservation of spontaneous ventilation and protective airway reflexes, haemodynamic stability, and prompt, clear-headed recovery without residual sedation. Historically, agents such as chloral hydrate, oral midazolam, intramuscular ketamine, and inhaled sevoflurane have been employed for this purpose. However, each carries recognised limitations — chloral hydrate is associated with prolonged and unpredictable sedation duration with regulatory restrictions in several countries; midazolam frequently produces inadequate sedation depth for MRI; ketamine causes excessive secretions, emergence delirium, and is contraindicated in patients with raised intracranial pressure; and inhalational anaesthesia necessitates the presence of a dedicated anaesthesiologist with MRI-compatible equipment (3).

Dexmedetomidine, a highly selective α-2 adrenoceptor agonist with a selectivity ratio of 1620:1 (α-2:α-1), has attracted increasing interest as a procedural sedative in the paediatric population over the past two decades. Its mechanism of action — stimulation of α-2A adrenoceptors in the locus coeruleus producing a natural non-REM sleep-like sedation — is mechanistically distinct from all other sedative classes and uniquely preserves respiratory drive, airway tone, and protective laryngeal reflexes. These properties make dexmedetomidine particularly attractive for procedural sedation in non-operating-room environments, where continuous anaesthesiologist presence and intubation capability may be limited.

Published studies have reported dexmedetomidine procedural success rates of 94–100% for paediatric MRI when used as a sole agent at higher doses, or in combination with low-dose midazolam or propofol (4). However, the optimal dosing regimen, particularly the loading dose and maintenance infusion rate that balances sedation adequacy against haemodynamic side effects in a resource-limited tertiary care setting, remains an area of active investigation. The present study was therefore designed to assess the sedation profile and safety of a standardised intravenous dexmedetomidine protocol (loading dose 1 mcg/kg + maintenance 0.7–1.0 mcg/kg/hr) for MRI brain sedation in paediatric patients aged 1–10 years in a single-centre observational setting.

2 | MATERIALS AND METHODS

Study Design and Setting

This was a prospective observational study conducted in the Department of Anaesthesiology at a tertiary care teaching hospital. The study was carried out over a defined period following approval from the Institutional Ethics Committee (IEC) and written informed consent obtained from the parents or legal guardians of all participating children. The study was conducted in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines.

Study Population

Inclusion Criteria

Paediatric patients aged 1–10 years, of either sex, classified as ASA physical status Grade I or II, scheduled for elective MRI brain under procedural sedation, were enrolled in this study.

Exclusion Criteria

Patients were excluded if they had known hypersensitivity to dexmedetomidine, significant cardiac conduction abnormalities (second- or third-degree atrioventricular block, sick sinus syndrome), haemodynamic instability, severe hepatic impairment, active respiratory infection, obesity (body mass index > 95th percentile for age), or if informed consent was refused or withdrawn.

Sample Size

A total of 52 patients were enrolled based on a priori sample size calculation using the expected procedural success rate of dexmedetomidine sedation (90%) derived from published literature, with a margin of error of 5% at a 95% confidence level (two-tailed).

Sedation Protocol

All patients were kept nil-by-mouth as per standard fasting guidelines (6 hours for solids and 2 hours for clear fluids). On arrival at the MRI suite, intravenous access was secured under topical anaesthetic cream (EMLA). A portable pulse oximeter was placed for continuous monitoring of peripheral oxygen

saturation(SpO_2) and pulse rate (PR).Premedication was administered with Inj. ondansetron and Inj. glycopyrrolate according to institutional protocol. All patients were allowed to breathe spontaneously through a face mask with oxygen at a flow rate of 2-3L/min throughout the sedation period as part of the routine procedural protocol.

Sedation was initiated with a loading dose of intravenous dexmedetomidine at 1 mcg/kg, infused over 10 minutes. This was followed immediately by a maintenance infusion of 0.7–1.0 mcg/kg/hr, titrated to effect. The MRI scan was commenced once adequate sedation (RSS $\geq$ 4) was achieved. If adequate sedation was not achieved or maintained, rescue sedation with intravenous propofol 1 mg/kg bolus was administered at the discretion of the attending anaesthesiologist. Dexmedetomidine infusion was discontinued 10 minutes before the anticipated completion of the procedure.

Outcome Measures

The primary outcomes were time to onset of adequate sedation (defined as RSS $\geq$ 4), recovery time (time from infusion cessation to RSS $\leq$ 2), MRI image quality (scored 1–3: 1 = no motion artefact; 2 = minor motion, diagnostically acceptable; 3 = repeat scan required), and requirement for propofol rescue sedation.

The secondary outcomes included serial haemodynamic parameters (heart rate, SpO_2) recorded at baseline and at 10-minute intervals up to 90 minutes, Ramsay Sedation Score at each time point, and incidence of adverse events (bradycardia, desaturation, respiratory depression, laryngospasm, nausea, or vomiting).

Statistical Analysis

Data were expressed as mean ± standard deviation (SD) for continuous variables and as frequency with percentage for categorical variables. Serial haemodynamic and sedation score data were analysed using the Friedman test for overall repeated-measures comparison. Pairwise comparisons between each post-dose time point and baseline were performed using the Wilcoxon signed-rank test with Bonferroni correction. Categorical outcomes were compared using Fisher's exact test. Group differences in propofol rescue rates across age groups were assessed using the Kruskal-Wallis test. Correlation between continuous variables was evaluated using Pearson's correlation coefficient. All analyses were performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). A two-tailed p-value of < 0.05 was considered statistically significant.

3 | RESULTS

3.1 | Demographic and Baseline Characteristics

A total of 52 paediatric patients were enrolled in this prospective observational study over the study period. The demographic and baseline characteristics are detailed in Table 1 .

The cohort comprised 33 males (63.5%) and 19 females (36.5%), with a mean age of 5.83 ± 2.17 years (range: 1.5–10 years) and a mean body weight of 15.12 ± 4.71 kg (range: 6.5–25 kg). Based on the American Society of Anesthesiologists (ASA) physical status classification, 13 patients (25.0%) were ASA Grade I and 39 (75.0%) were ASA Grade II. Age-wise, 29 patients (55.8%) belonged to the school-age group (6–10 years), 20 (38.5%) to the preschool group (3–5 years), and 3 (5.8%) to the toddler group (1–2 years) (Table 1). All patients received a uniform loading dose of dexmedetomidine 1 mcg/kg intravenously over 10 minutes, followed by a maintenance infusion of 0.7–1.0 mcg/kg/hr (median 1.0 mcg/kg/hr).

3.2 | Clinical Indications for MRI Brain

The distribution of clinical indications for which MRI brain was requested is presented in Table 2. Convulsion disorder was the most frequent indication (n = 16; 30.8%), followed by acute febrile illness (n = 13; 25.0%) and developmental delay (n = 9; 17.3%). Together, these three conditions accounted for 73.1% of all cases. Meningitis constituted 7.7% (n = 4), while headache and cerebral palsy each accounted for 3.8% (n = 2 each). Rare indications — including encephalitis, hydrocephalus, peripheral neuropathy, head trauma, and microcephaly — each occurred in one patient (1.9%) Table 2 .

Table 1. Demographic and Baseline Characteristics of the Study Population (n = 52)

Variable	Value	Percentage / Range
Total patients enrolled	52	—
Male	33	63.5%
Female	19	36.5%
Mean age (years) ± SD	5.83 ± 2.17	Range: 1.5–10 years
Mean body weight (kg) ± SD	15.12 ± 4.71	Range: 6.5–25.0 kg
ASA Physical Status Grade I	13	25.0%
ASA Physical Status Grade II	39	75.0%
Toddler age group (1–2 years)	3	5.8%
Preschool age group (3–5 years)	20	38.5%
School-age group (6–10 years)	29	55.8%
Loading dose of dexmedetomidine	1 mcg/kg (uniform)	—
Maintenance dose range	0.7–1.0 mcg/kg/hr	Median: 1.0 mcg/kg/hr

Table 2. Distribution of Clinical Indications for MRI Brain (n = 52)

Clinical Indication	n	%
Convulsion disorder	16	30.8
Acute febrile illness	13	25.0
Developmental delay	9	17.3
Meningitis	4	7.7
Headache	2	3.8
Cerebral palsy	2	3.8
Encephalitis	1	1.9
Hydrocephalus	1	1.9
Peripheral neuropathy	1	1.9
Head trauma	1	1.9
Microcephaly	1	1.9
Total	**52**	**100.0**

3.3 | Primary Outcome Measures

The primary outcome measures — including time to onset of adequate sedation, recovery time, MRI scan duration, requirement for supplementary propofol, and MRI image quality — are summarised in Table 3 .

The mean time to onset of adequate sedation (defined as Ramsay Sedation Score ≥ 4) was 6.59 ± 0.99 minutes (range: 4.20–8.33 min). The mean recovery time following cessation of the infusion was 11.25 ± 1.61 minutes (range: 7.60–15.17 min). The mean duration of MRI scanning was 58.28 ± 7.25 minutes (range: 48.17–84.50 min). Supplementary propofol rescue was required in only 5 of 52 patients (9.6%).

Regarding MRI image quality, 47 patients (90.4%) achieved a quality score of 1 (no patient motion; optimal image quality) with dexmedetomidine alone. The remaining 5 patients (9.6%) who required propofol rescue each achieved a quality score of 2 (minor motion artefact, but diagnostically acceptable). No patient required a repeat MRI scan (score 3), representing a 100% procedural success rate Table 3 and Figure 1 .

3.4 | Haemodynamic Monitoring — Heart Rate

Serial heart rate (HR) measurements were obtained at baseline and at 10-minute intervals up to 90 minutes following the loading dose. The mean HR at each time point with corresponding standard deviations and percentage change from baseline are presented in Table 4 , and the temporal trend is depicted in Figure 2 .

At baseline, the mean HR was 111.58 ± 9.00 bpm. Following initiation of dexmedetomidine, a statis-

Table 3. Primary Outcome Measures of Dexmedetomidine Sedation (n = 52)

Outcome Measure	Mean ± SD / n (%)	Range
Time to onset of sedation (min)	6.59 ± 0.99	4.20–8.33
Recovery time (min)	11.25 ± 1.61	7.60–15.17
MRI scan duration (min)	58.28 ± 7.25	48.17–84.50
Propofol rescue required	5 / 52 (9.6%)	—
MRI Quality Score 1 (no motion)	47 / 52 (90.4%)	—
MRI Quality Score 2 (minor motion)	5 / 52 (9.6%)	—
MRI Quality Score 3 (repeat scan)	0 / 52 (0.0%)	—
Overall procedural success rate	100%	—

Figure 1. MRI Image Quality Score by Sedation Adequacy (n=52)
Procedural success rate = 100% | 0 repeat scans required

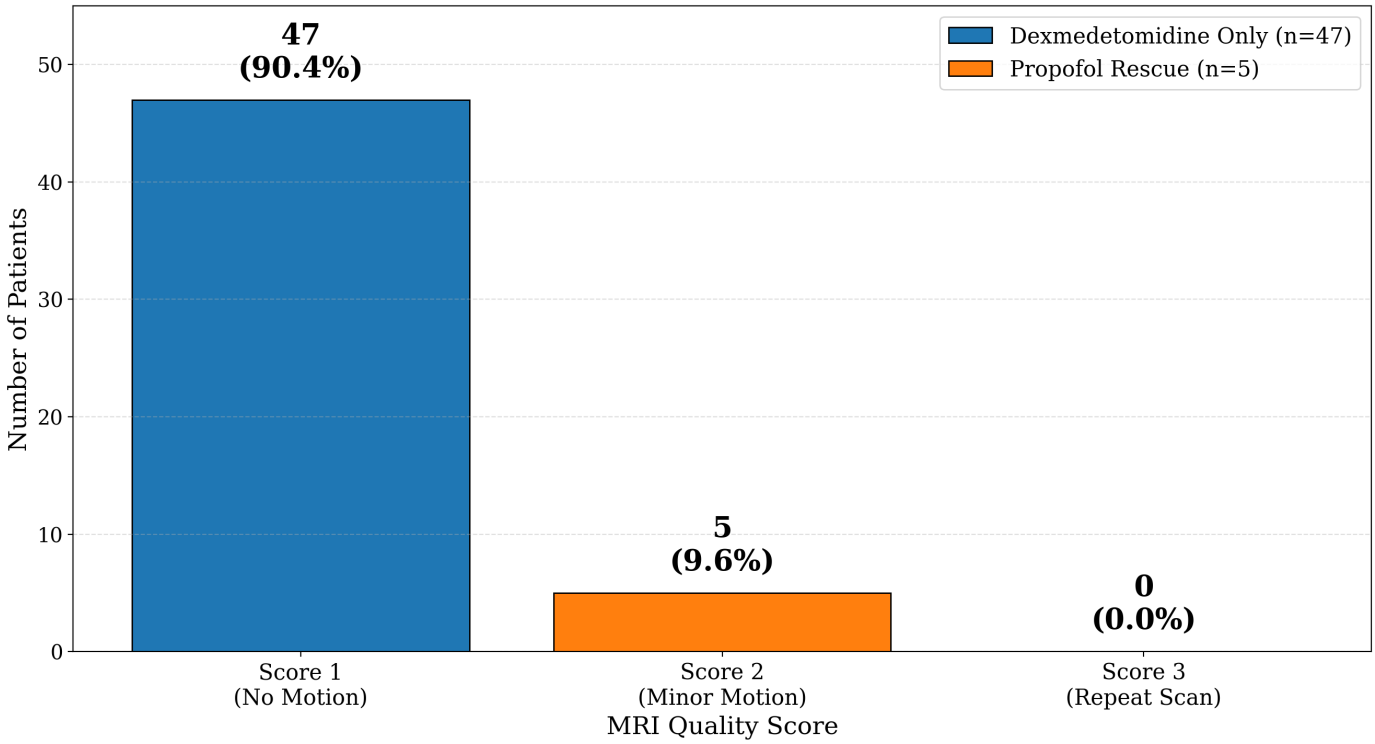

Fig. 1: MRI image quality score distribution by sedation adequacy (n = 52). Blue bar = dexmedetomidine-only group (n = 47); orange bar = propofol-rescue group (n = 5). Score 1 = no motion artefact (optimal); Score 2 = minor motion (diagnostically acceptable); Score 3 = repeat scan required. All 52 scans were completed successfully without repeat.

tically significant and progressive reduction in HR was observed across all time points (Friedman χ^2 = 312.0, $p < 0.001$). The HR reached its nadir at 20 minutes, recording a mean of 94.92 ± 8.18 bpm — a reduction of 14.9% from baseline. Thereafter, a gradual recovery trend was observed, with the mean HR reaching 100.42 ± 6.62 bpm at 90 minutes. All pairwise Wilcoxon signed-rank comparisons between baseline and each subsequent time point were statistically significant ($p < 0.001$ for all), supporting the consistent sympatholytic effect of dexmedetomidine Table 4 and Figure 2 .

Importantly, the HR reduction remained within clinically acceptable limits in 50 of 52 patients (96.2%). Only 2 patients (3.8%) developed transient bradycardia, which was self-limiting and required no pharmacological intervention (see Section 8).

Table 4. Serial Heart Rate Values at Each Time Point — Mean ± SD and Statistical Significance (n = 52)

Time Point	Mean HR (bpm)	SD (±)	% Change from Baseline	p-value vs Baseline†
Baseline	111.58	9.00	—	—
10 min	96.33	8.66	−13.7%	< 0.001
20 min	94.92	8.18	−14.9% ▼	< 0.001
30 min	95.38	8.11	−14.5%	< 0.001
40 min	96.23	8.07	−13.7%	< 0.001
50 min	97.04	7.75	−13.0%	< 0.001
60 min	97.69	7.58	−12.4%	< 0.001
70 min	98.75	7.48	−11.5%	< 0.001
80 min	99.62	6.33	−10.7%	< 0.001
90 min	100.42	6.62	−10.0%	< 0.001

†Wilcoxon signed-rank test (two-tailed). ▼ Nadir value. Friedman χ^2 = 312.0, p < 0.001 for overall repeated-measures comparison.

Figure 2. Mean Heart Rate (±SD) During IV Dexmedetomidine Sedation
Friedman χ² = 312.0, p < 0.001 | All time points vs baseline: Wilcoxon p < 0.001

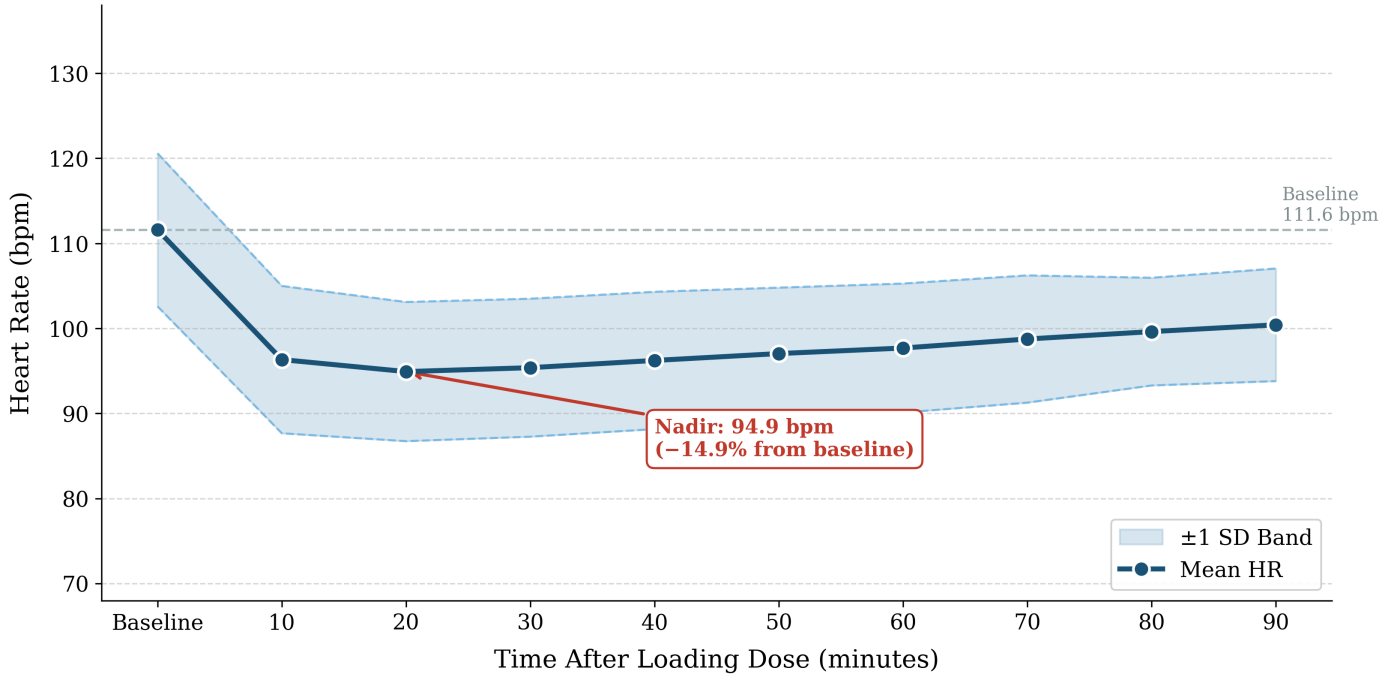

Fig. 2: Mean heart rate (±SD) trend from baseline to 90 minutes following IV dexmedetomidine. The shaded region represents the ±1 SD band; dashed lines are ±1 SD borders. HR nadir occurred at 20 minutes (94.92 ± 8.18 bpm; −14.9% from baseline). Dashed horizontal grey line = baseline reference (111.6 bpm). Friedman χ^2 = 312.0, p < 0.001. All post-dose time points differ significantly from baseline (Wilcoxon p < 0.001).

3.5 | Peripheral Oxygen Saturation (SpO_2) Monitoring

Peripheral oxygen saturation (SpO_2) was monitored continuously by pulse oximetry and recorded at baseline and at 10-minute intervals throughout sedation. The recorded values are presented in Table 5 , and the temporal profile is illustrated in Figure 3 .

SpO_2 remained clinically stable throughout the procedure. No patient experienced clinically significant oxygen desaturation (defined as SpO_2 < 92%) at any point during the study. No patient required airway repositioning, assisted ventilation, or any rescue airway intervention. These findings support the respiratory-sparing pharmacological profile of dexmedetomidine.

Table 5. Peripheral Oxygen Saturation (SpO_2) at Each Time Point — Mean ± SD (n = 52)

Time Point	Mean SpO_2 (%)	SD (±)	Min (%)	Max (%)	Clinical Event
Baseline	98.50	0.70	97.0	100.0	None
10 min	99.15	0.52	98.0	100.0	None
20 min	99.87	0.34	99.0	100.0	None
30 min	99.79	0.41	99.0	100.0	None
40 min	99.83	0.38	99.0	100.0	None
50 min	99.83	0.38	99.0	100.0	None
60 min	99.48	0.54	98.0	100.0	None
70 min	98.96	0.19	98.5	99.5	None
80 min	98.69	0.50	97.5	99.5	None
90 min	98.67	0.51	97.5	100.0	None

SpO_2 remained clinically stable throughout the monitoring period. No patient recorded SpO_2 < 92% (clinical desaturation threshold) at any time point.

Figure 3. Mean SpO_2 (± SD) During IV Dexmedetomidine Sedation
Clinically stable oxygen saturation | Routine supplemental oxygen | No desaturation events

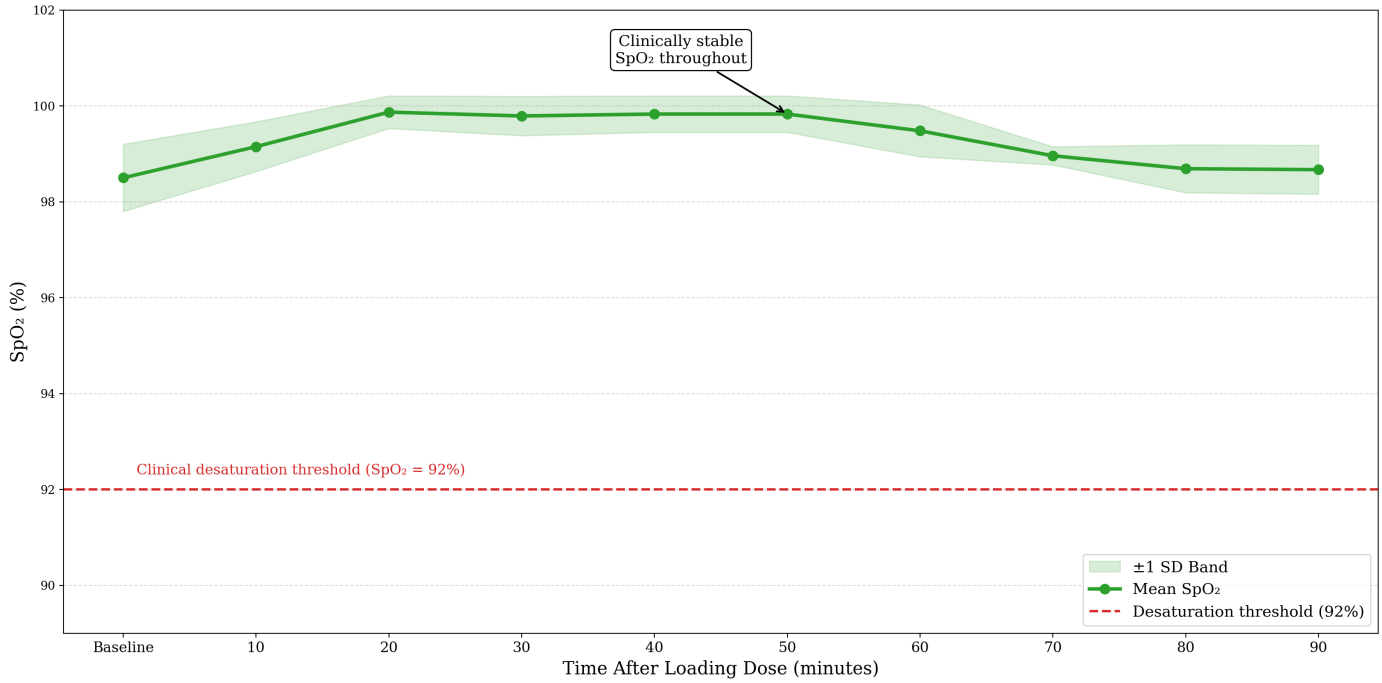

Fig. 3: Mean peripheral oxygen saturation (SpO_2 ± SD) from baseline to 90 minutes during IV dexmedetomidine sedation. Shaded region = ±1 SD band; red dashed horizontal line = clinical desaturation threshold (SpO_2 = 92%). SpO_2 remained clinically stable throughout, and no desaturation events were recorded.

3.6 | Sedation Depth — Ramsay Sedation Score (RSS

Sedation depth was assessed using the six-point Ramsay Sedation Scale (RSS) at baseline and at 10-minute intervals throughout the procedure. The RSS trajectory is presented in Table 6 and illustrated in Figure 4 .

At baseline, patients were awake and alert with a mean RSS of 1.85 (corresponding to RSS 2: cooperative, orientated, and tranquil). Following the dexmedetomidine loading dose, the RSS increased rapidly to a mean of 4.98 at 10 minutes, reaching its maximum mean of 5.0 at 20 minutes, indicating deep procedural sedation (asleep with sluggish response to stimuli) in virtually all patients. This level of deep sedation (RSS ≥ 4) was consistently maintained from 10 minutes through 50 minutes, encompassing the full MRI acquisition window for all 52 patients Table 6 and Figure 4 .

The RSS declined progressively following infusion cessation: mean RSS was 4.0 at 60 minutes and 3.0 at 70 minutes. By 80 minutes, patients had returned to a near-baseline level of RSS 1.98, with a further return to RSS 1.90 at 90 minutes, supporting smooth and predictable recovery without residual sedation. The mean recovery time to RSS ≤ 2 was 11.25 ± 1.61 minutes.

Table 6. Ramsay Sedation Score (RSS) Trajectory During IV Dexmedetomidine Sedation (n = 52)

Time Point	Mean RSS	RSS Level Description	Sedation Phase
Baseline	1.85	Awake: alert or anxious (RSS 1–2)	Pre-sedation
10 min	4.98	Asleep: brisk response to stimuli	Deep sedation (onset)
20 min	5.00	Asleep: sluggish response — PEAK	Deep sedation
30 min	4.98	Asleep: sluggish response	Deep sedation
40 min	4.98	Asleep: sluggish response	Deep sedation
50 min	4.90	Asleep: brisk–sluggish response	Deep sedation (waning)
60 min	4.00	Asleep: brisk response to stimuli	Deep sedation (lower)
70 min	3.00	Drowsy: responds to commands	Moderate sedation
80 min	1.98	Cooperative, orientated	Recovery (near baseline)
90 min	1.90	Alert, cooperative	Full recovery

3.7 | Requirement for Supplementary Propofol (Rescue Sedation

Supplementary propofol was required in 5 of 52 patients (9.6%) to achieve and maintain adequate sedation for MRI completion. The distribution of rescue requirement by age group is presented in Table 7 and illustrated in Figure 5 .

The requirement for propofol rescue demonstrated a clinically meaningful inverse relationship with patient age (Kruskal-Wallis, p = 0.07). Among toddlers (1–2 years, n = 3), 1 patient (33.3%) required rescue sedation; in the preschool group (3–5 years, n = 20), 3 patients (15.0%) required supplementation; and in the school-age group (6–10 years, n = 29), only 1 patient (3.4%) required propofol (Table 7 and Figure 5). All patients who received supplementary propofol subsequently achieved MRI completion with a quality score of 2 (minor motion, diagnostically acceptable), and no repeat scan was required in any case.

Table 7. Propofol Rescue Requirement by Age Group and Patient Details (n = 52)

Age Group	Total n	Rescue n (%)	MRI Quality (Post-Rescue)	Outcome
Toddler (1–2 years)	3	1 (33.3%)	Score 2	Scan completed; no repeat
Preschool (3–5 years)	20	3 (15.0%)	Score 2	Scan completed; no repeat
School-age (6–10 years)	29	1 (3.4%)	Score 2	Scan completed; no repeat
Overall	**52**	**5 (9.6%)**	**Score 2 (all)**	**100% success**

Kruskal-Wallis test for age-group effect: p = 0.07. Trend toward inverse relationship between age and rescue requirement.

3.8 | Adverse Events

The adverse event profile during dexmedetomidine sedation is summarised in Table 8. The overall incidence of adverse events was low (3.8%), with no serious adverse events recorded in any patient.

Only 2 patients (3.8%) experienced a clinically noted adverse event — both cases of transient bradycardia (heart rate < 80 bpm), occurring in cases 9 and 19. Case 9 was a 7-year-old female (15 kg, ASA II) and Case 19 was a 4.5-year-old female (13 kg, ASA II). In both patients, the bradycardia was transient, self-

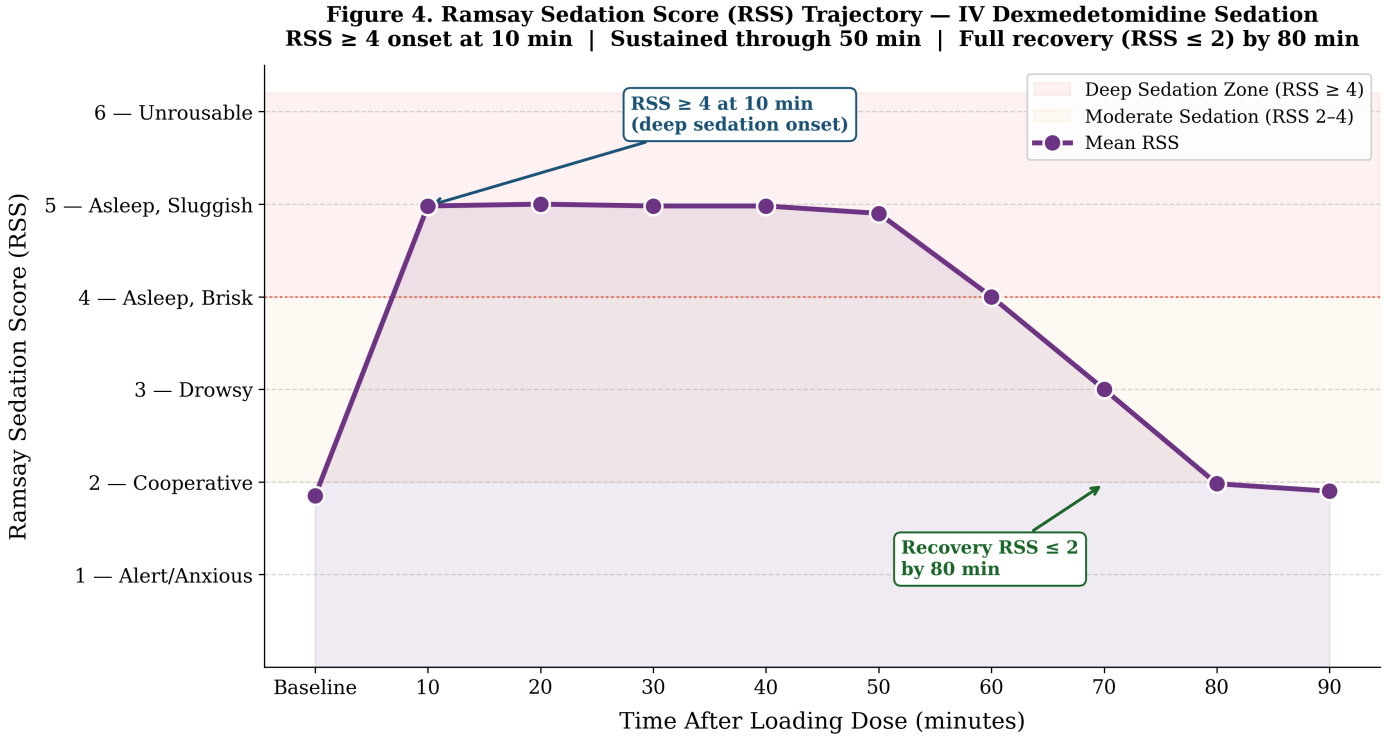

Fig. 4: Mean Ramsay Sedation Score (RSS) trajectory during IV dexmedetomidine sedation. Red shaded zone = deep sedation target (RSS $\geq$ 4). Yellow zone = moderate sedation (RSS 2–4). RSS $\geq$ 4 was achieved within 10 minutes in all patients and maintained through 50 minutes. Recovery to baseline RSS ($\leq$ 2) occurred by 80 minutes following infusion cessation.

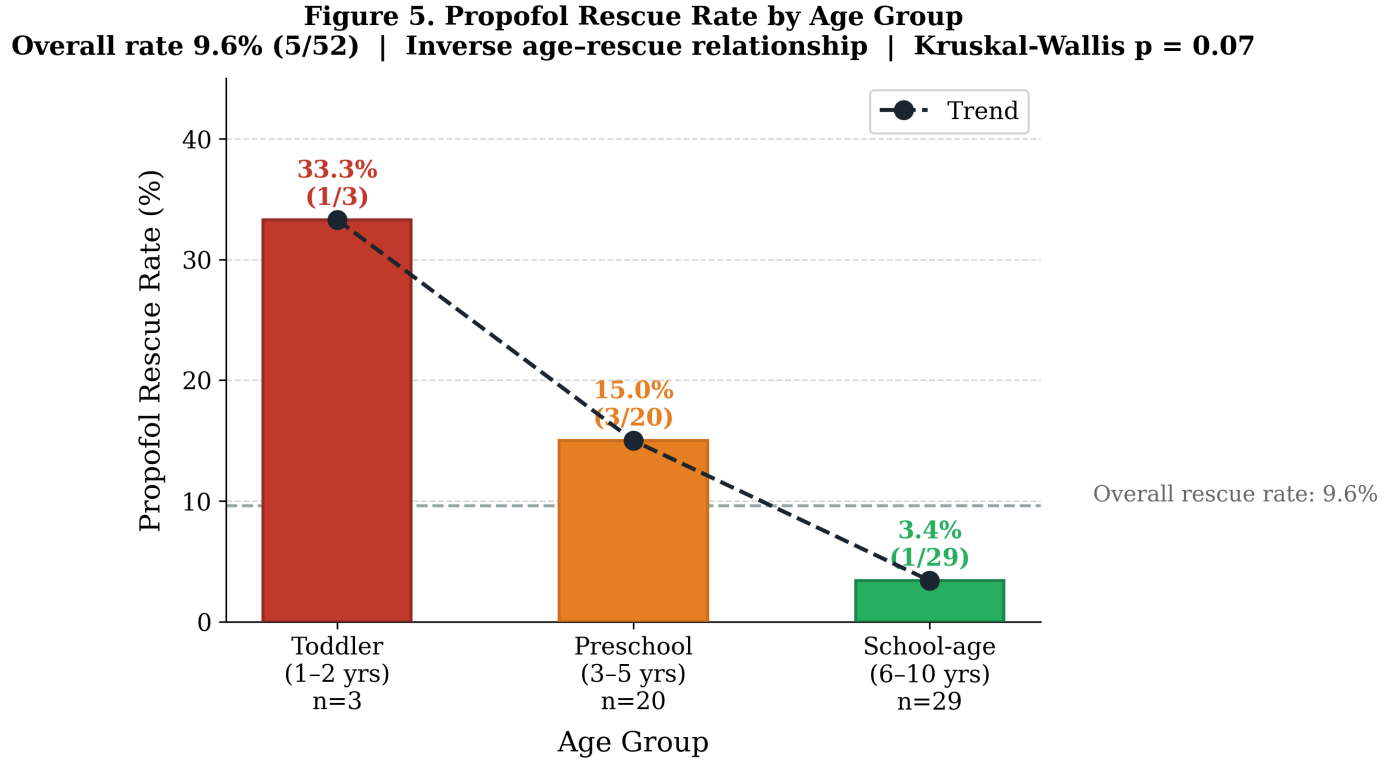

Fig. 5: Propofol rescue rate (%) by age group. Bars show rescue percentage per group; dotted trend line illustrates the inverse age–rescue relationship. Grey dashed horizontal line = overall rescue rate (9.6%). Rescue rate: 33.3% (toddlers), 15.0% (preschool), 3.4% (school-age). Kruskal-Wallis p = 0.07.

limiting, and resolved spontaneously without pharmacological intervention (e.g., atropine or glycopyrrolate). Neither event resulted in haemodynamic instability or premature termination of the scan.

No patient developed respiratory depression, oxygen desaturation (SpO_2 < 92%), laryngospasm, apnoea, nausea, vomiting, hypotension, or any other clinically significant adverse event during the study period Table 8 . The 95% confidence interval for the overall adverse event rate was 0.0%–9.6%.

3.9 | Correlation Analysis

Pearson correlation analysis was performed to examine relationships between key patient and procedural variables. Results are presented in Table 9 .

No statistically significant correlation was identified between patient age and onset of sedation (r = −0.102, p = 0.470), nor between body weight and onset of sedation (r = −0.130, p = 0.357). This indicates that the standardised weight-based dosing protocol (loading 1 mcg/kg + maintenance 1 mcg/kg/hr) produced consistent and predictable pharmacodynamics across the paediatric age and weight spectrum studied. A weak positive trend was observed between MRI scan duration and recovery time (r = +0.214, p = 0.127), suggesting that longer scans may marginally prolong emergence, though this did not reach statistical significance in the current sample (Table 9).

4 | DISCUSSION

Procedural sedation for paediatric MRI remains a significant clinical challenge, demanding an agent that reliably achieves deep, rapidly reversible sedation while preserving respiratory function. The present study evaluated intravenous dexmedetomidine at a loading dose of 1 mcg/kg followed by a maintenance infusion of 0.7–1.0 mcg/kg/hr in 52 children aged 1–10 years. Our findings demonstrate a 100% procedural success rate, rapid onset and recovery, complete respiratory preservation, and an adverse event rate of only 3.8%, establishing dexmedetomidine as a highly effective and safe first-line sedative for this indication.

The overall procedural success rate of 100% in this study, with 90.4% of patients achieving optimal MRI quality score 1 with dexmedetomidine alone, compares favourably with previously published data. Mason et al., in a landmark retrospective study of 544 paediatric patients using a higher dose protocol (2 mcg/kg bolus + 1 mcg/kg/hr infusion), reported a 100% scan completion rate, though 21.5% required additional medications (midazolam or fentanyl) to suppress motion artefacts (5, 6) . In the largest nurse-led dexmedetomidine sedation series to date, Sulton et al. analysed 1,768 consecutive intravenous dexmedetomidine sedation episodes and reported an overall success rate of 98.9% for intravenous dexmedetomidine, with a scan interruption rate of 8.8% — results broadly consistent with our findings. In the current study, only 9.6% of patients required supplementary propofol rescue, and zero patients required a repeat scan, suggesting that our lower-dose standardised weight-based protocol may achieve comparable procedural performance with reduced pharmacological burden (7) , though direct randomised comparison is warranted. In a recent meta-analysis of six studies (n=633) comparing dexmedetomidine with midazolam and chloral hydrate for paediatric MRI/CT sedation, dexmedetomidine demonstrated a significantly higher successful sedation rate compared to midazolam (risk ratio 0.43, 95% CI 0.29–0.64), while showing comparable procedural performance to chloral hydrate (8).

The mean onset of adequate sedation in the present study (6.59 ± 0.99 minutes) is consistent with established pharmacokinetic data and closely parallels prior reports. Heard et al., in one of the early case series of dexmedetomidine for paediatric MRI, reported variable onset times, noting that dexmedetomidine alone was ”more unpredictable than anticipated” at lower doses (0.5–1.5 mcg/kg bolus). Liaudanskytė et al. reported a median onset time of 8 minutes (range 3–13 min) in 87 paediatric MRI sedation cases, with 51% of patients achieving deep sedation before 10 minutes — comparable to the near-universal deep sedation (RSS ≥ 4 in all patients) at 10 minutes in our cohort. The mean

Table 8. Adverse Events During IV Dexmedetomidine Sedation (n = 52)

Adverse Event	n (%)	95% CI	Details	Management / Outcome
Transient bradycardia	2 (3.8%)	0.0–9.6%	Cases 9 & 19; HR < 80 bpm; transient	Self-limiting; no drug intervention
Oxygen desaturation (SpO_2 <92%)	0 (0.0%)	—	None observed	N/A
Respiratory depression	0 (0.0%)	—	None observed	N/A
Laryngospasm / apnoea	0 (0.0%)	—	None observed	N/A
Nausea / Vomiting	0 (0.0%)	—	None observed	N/A
Hypotension	0 (0.0%)	—	None observed	N/A
Serious adverse event	0 (0.0%)	—	None observed	N/A

Table 9. Pearson Correlation Analysis Between Key Study Variables

Correlation Pair	Pearson r	p-value	Interpretation	Clinical Significance
Age (yrs) vs Onset of sedation (min)	−0.102	0.470	Very weak inverse	Not significant
Body weight (kg) vs Onset (min)	−0.130	0.357	Weak inverse	Not significant
MRI duration (min) vs Recovery time (min)	+ 0.214	0.127	Weak positive trend	Not significant (trend)

NS = Not statistically significant ($p > 0.05$). Two-tailed Pearson correlation coefficient (r).

recovery time of 11.25 ± 1.61 minutes observed in the present study is notably shorter than the discharge times of 92–94 minutes reported by Ahmed et al. and the 90-minute discharge time reported by Heard et al., though these figures represent time-to-discharge-readiness rather than time to RSS ≤ 2, explaining the apparent disparity. Our recovery metric specifically measures the interval to RSS ≤ 2, reflecting a clinically meaningful and reproducible endpoint. (9, 10)

The statistically significant HR reduction observed in this study (nadir −14.9% at 20 minutes; Friedman $\chi^2 = 312.0$, $p < 0.001$) reflects the well-characterised α-2 adrenoceptor-mediated sympatholytic effect of dexmedetomidine. This magnitude of reduction aligns with the published median HR decrease of 15% (interquartile range 9–23%) reported in a pooled analysis of dexmedetomidine procedural sedation studies. Mason et al. reported a 16% incidence of clinically defined bradycardia with their higher-dose protocol (2 mcg/kg bolus), with all patients maintaining concomitant blood pressure and SpO_2 within acceptable limits. Liaudanskytė et al. reported bradycardia in 11.5% of cases using a combination protocol of high-dose dexmedetomidine (3 mcg/kg over 10 min) with midazolam, with 5 of 10 patients requiring atropine. In the present study, bradycardia occurred in only 2 patients (3.8%), both self-resolving without intervention — an incidence consistent with the lower loading dose employed and comparable to the 3.9% rate reported by Ahmed et al. using a 2 mcg/kg protocol in 544 patients. Siddappa et al., studying high-dose dexmedetomidine (2 mcg/kg bolus) in 77 paediatric MRI patients, reported MRI completion in 76 of 77 (99%) patients with spontaneously resolving haemodynamic side effects and no respiratory depression — further supporting the benign clinical course of dexmedetomidine-associated bradycardia (11, 12) .

The most clinically relevant finding of the present study is the absence of clinically significant respiratory adverse events. No patient experienced oxygen desaturation ($SpO_2 < 92\%$), airway obstruction, laryngospasm, or apnoea. SpO_2 remained clinically stable throughout the procedure and no patient required airway rescue or escalation of respiratory support. This is consistent with the mechanistic basis of dexmedetomidine's respiratory sparing: its action at α-2A adrenoceptors in the locus coeruleus produces natural sleep-like sedation without the GABAergic or opioid receptor-mediated respiratory depression associated with propofol, benzodiazepines, and

opioids. Koroglu et al., in a randomised comparison of dexmedetomidine versus propofol for paediatric MRI sedation, demonstrated that propofol was associated with significantly higher rates of respiratory depression and haemodynamic instability compared to dexmedetomidine, despite offering faster onset and recovery. Ahmed et al. similarly reported that in 544 patients receiving high-dose dexmedetomidine, no desaturation, airway obstruction, or apnoea events were observed, with a 38.6% incidence of mild respiratory rate reduction (>20% from baseline) carrying no clinical consequence (13, 14)

The inverse relationship between patient age and propofol rescue rate (toddlers 33.3% vs. school-age children 3.4%; $p = 0.07$) is a novel and clinically important observation. Ahmed et al. also reported that bradycardia occurred predominantly in children aged 1–3 years in their cohort (15) . The higher sedation failure rate in younger children in our study may reflect developmentally higher metabolic rates, greater volume of distribution, immature hepatic glucuronidation pathways, and potentially higher α-2 receptor threshold variability in toddlers. Intranasal dexmedetomidine augmentation as a premedication strategy — which has demonstrated a success rate of 81% versus 52% for standard oral midazolam ($p = 0.017$) in a paediatric MRI cohort — may represent a useful adjunct for this age group in future protocols (16) .

5 | LIMITATIONS

The present study has several limitations. The single-centre, observational design without a comparator arm limits causal inference. The sample size of 52 patients is adequate for descriptive analysis but may be underpowered to detect rare adverse events. The absence of capnography monitoring is a recognised limitation in procedural sedation outside the operating theatre. Future prospective randomised controlled trials comparing dexmedetomidine with other sedative agents in this paediatric age group, with capnography monitoring and longer follow-up, are warranted to consolidate these findings.

6 | CONCLUSION

Intravenous dexmedetomidine at a loading dose of 1 mcg/kg followed by a maintenance infusion of 0.7–1.0 mcg/kg/hr provides effective, safe, and rapidly reversible procedural sedation for MRI brain in paediatric patients aged 1–10 years. The protocol achieved a 100% procedural success rate with optimal MRI image quality in 90.4% of patients, rapid sedation onset (6.59 ± 0.99 min), and prompt recovery (11.25 ± 1.61 min).No clinically significant respiratory adverse events were observed. Adverse events were limited to 3.8% cases of transient self resolving bradycardia that required no pharmacological intervention. Further prospective comparative studies with larger sample sizes are warranted to validate these findings. Dose optimisation in younger children warrants further investigation in prospective randomised controlled trials.

Declarations

Ethics approval and consent to participate

The authors should provide the correct Institutional Ethics Committee approval details for this study, including approval number and date. Written informed consent was obtained from all participants prior to inclusion in the study.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Dr. Thengujam Ashapriya Devi: Conceptualization, manuscript drafting, literature review, data collection and corresponding author responsibilities.

Dr. Rupali N. Gorgile: Supervision, interpretation of findings, statistical support and critical revision of the manuscript.

Dr. Pawal Avinash Tukaram: Study design support, interpretation of data, data analysis and manuscript review.

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