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DEXMEDETOMIDINE FOR SEDATION IN CHILDREN – A NARRATIVE REVIEW OF CLINICAL EVIDENCE, SAFETY CONSIDERATIONS, AND EMERGING THERAPEUTIC ROLES

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ABSTRACT

Dexmedetomidine is a highly selective alfa2-adrenergic agonist increasingly used in pediatric anesthesia and intensive care because of its unique sedative profile and minimal respiratory depressive effects. This narrative summarizes current evidence regarding the pharmacology, pharmacokinetics, clinical efficacy, safety profile and emerging applications of dexmedetomidine in pediatric patients.

Current literature supports the use of dexmedetomidine in multiple clinical settings, including procedural sedation, perioperative management, and pediatric intensive care. The drug has demonstrated efficacy in reducing opioid requirements, preventing emergence agitation and delirium, and providing effective sedation while preserving spontaneous respiration. However, its use remains associated with dose-dependent cardiovascular adverse effects, particularly bradycardia and hypotension, requiring individualized dosing and careful monitoring, especially in neonates and infants due to age-related pharmacokinetic variability.

Emerging evidence suggests potential neuroprotective and organ-protective properties, although robust pediatric clinical data remains limited. Novel applications and alternative administration routes continue to expand the potential role of dexmedetomidine in pediatric practice.

Overall, dexmedetomidine represents a versatile and valuable sedative agent in pediatric medicine, though further high-quality studies are needed to optimize dosing strategies and better define long-term safety outcomes.

KEYWORDS

Dexmedetomidine, Children, Pediatric, PICU, Sedation, Anesthesia

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1. Introduction

Sedation is widely used in hospitalized children across various settings, including the pediatric intensive care unit (PICU), diagnostic procedures, and the perioperative period (1) (2) (3). Adequate sedation reduces anxiety, facilitates mechanical ventilation, and enables safe performance of imaging and invasive procedures (4) (3). However, achieving optimal sedation remains challenging due to heterogeneity of pediatric populations, from neonates to adolescents, each with distinct pharmacokinetic and pharmacodynamic profiles (5).

Traditional sedatives, including benzodiazepines and opioids, have significant limitations such as risk of respiratory depression, tolerance, paradoxical reactions, and potential association with delirium and withdrawal with prolonged use (6) (7) (1) (8).

Dexmedetomidine, a highly selective ⍺2-adrenergic agonist, has emerged as a promising alternative in pediatric patients (2) (9) (3). It provides sedation resembling natural sleep, allowing easy arousal while preserving respiratory function (10). Its pharmacological profile includes sedative, anxiolytic, analgesic, sympatholytic, and opioid-sparing properties (1) (3) (10).

In the European Union, all pediatric applications are largely off-label (2). Despite this, over 50% of European pediatric anesthesiologists prescribe dexmedetomidine for various indications (11). Globally, regulatory approvals differ, and pediatric use is often limited to specific indications or considered off-label.

Clinical applications include PICU sedation, procedural sedation for diagnostic imaging, perioperative use, and prevention of emergence agitation (12) (3) (2). Novel applications, such as management of anticholinergic toxidrome, a have also been reported (13).

Despite growing evidence, questions remain regarding optimal dosing, long-term safety, comparative effectiveness, and emerging indications (14) (12) (4). This narrative review summarizes current evidence on the efficacy, safety, and clinical applications of dexmedetomidine in pediatric patients, focusing on intensive care, procedural sedation, and perioperative settings.

2. Methodology

2.1. Study Design and Literature Search

This narrative review summarizes current evidence on dexmedetomidine use in pediatric patients, with a focus on efficacy, safety, and emerging indications. A literature search was conducted in PubMed using combinations of the following keywords: “dexmedetomidine”, “children”, “pediatric”, “sedation”, “intensive care”, “PICU”, and “anesthesia”. The search primarily included articles published between 2018 and 2026, with earlier studies incorporated when considered relevant. Reference lists of selected articles were also screened to identify additional publications.

2.2. Inclusion and Exclusion Criteria

Studies addressing dexmedetomidine use in pediatric patients for sedation, analgesia, anesthesia, or intensive care were included. Both original studies and review articles were considered. One study, although primarily focused on adults, was included due to the presence of a dedicated pediatric subgroup analysis. Non-English language publications were excluded. Studies were selected based on relevance to pediatric clinical use of dexmedetomidine and contribution to the predefined thematic scope.

2.3. Included studies

A total of 29 articles were included in this review:

- 9 systematic reviews and meta-analyses,
- 7 prospective clinical trials, randomized and non-randomized,
- 5 observational studies, prospective and retrospective, including surveys,
- 6 narrative reviews,
- 2 case reports

2.4. Data Synthesis

Due to heterogeneity among studies, a formal meta-analysis was not performed. A qualitative synthesis was conducted, with data organized thematically into the following domains: pharmacology, pharmacokinetics, dosing, clinical efficacy, comparative effectiveness, safety profile, withdrawal syndrome, and emerging indications. The methodological quality of included studies, particularly systematic reviews and randomized controlled studies, was considered during interpretation of findings.

Results

3. Pharmacology, Mechanism of Action, and Pharmacokinetics in Pediatric Patients

3.1. Mechanism of Action

Dexmedetomidine belongs to the class of ɑ2-adrenergic receptor agonist and demonstrates high receptor selectivity, with ɑ2: ɑ1 binding ratio of approximately 1600:1 – substantially higher than that of clonidine (200:1) (1). The preferential ɑ2-receptor affinity underlies its distinctive clinical effects, encompassing sedation, anxiolysis, analgesia, sympathetic modulation, and reduction of opioid requirements (3) (1).

The sedative properties of dexmedetomidine are primarily mediated by its action ɑ2-adrenoreceptors within the locus coeruleus, a key brainstem structure involved in regulating alertness and sleep-wake cycles (10). Activation of these receptors inhibits noradrenergic neurotransmission, thereby engaging physiological sleep-promoting circuits (10) (3). The resulting sedation is described as “arousable” or “cooperative”, as patients can be readily awakened and remain responsive to verbal commands, yet return to a calm state when unstimulated (10) (1). This pattern closely mimics physiological sleep and distinguishes it from GABAergic sedatives such as benzodiazepines and propofol (3).

3.2. Pharmacological Effects

Sedative and Analgesic Effects

The sedative effects of dexmedetomidine are dose-dependent. While higher doses can achieve profound sedation approaching deep sedation or anesthesia, the accompanying hemodynamic changes may restrict its use in patients with cardiovascular instability. Notably, cognitive function and memory appear to remain relatively intact at sedative doses (3).

Dexmedetomidine exerts analgesic effects through actions at multiple levels of the nervous system, including the spinal cord, supraspinal centers, and peripheral sites (3). As a standalone analgesic, however, its efficacy is modest, with evidence of a ceiling effect at higher concentrations (3). The primary analgesic value of dexmedetomidine lies in its role as an adjuvant, consistently demonstrating the ability to reduce opioid consumption in clinical studies (3) (1). In pediatric practice, this opioid-sparing capacity is particularly advantageous, potentially mitigating risks associated with prolonged opioid exposure, including respiratory compromise, tolerance development, and withdrawal phenomena (8).

Cardiovascular Effects

The hemodynamic profile of dexmedetomidine reflects a balance between centrally-mediated sympathetic inhibition and peripheral vascular effect (10). Bolus administration characteristically produces initial hypertension through peripheral ⍺2-receptor stimulation of vascular smooth muscle, which subsequently gives way to hypotension as central ⍺2-receptor-mediated sympatholysis becomes dominant (10) (3). Bradycardia occurs in a dose-related fashion, driven primarily by reduced sympathetic output with additional contributions from baroreceptor activation and parasympathetic enhancement (3) (2).

Respiratory Effects

In contrast to conventional sedatives and anesthetic agents, dexmedetomidine exerts minimal effects on respiratory drive, even when administered at elevated doses. This respiratory-sparing characteristic permits continued infusion during extubation and reduces the likelihood of ventilatory complications(10) (3). The maintenance of spontaneous breathing and intact airway protective mechanisms renders dexmedetomidine especially suitable for pediatric applications (2) (9).

Other Effects

Dexmedetomidine has been associated with lower delirium rates compared to alternative sedatives, and may confer protective effects against its development, which is particularly relevant in critically ill pediatric patients (6) (3) (7). The drug also demonstrated antisialagogue activity and potential neuroprotective properties, including attenuation of intracranial pressure and dose-related reductions in both cerebral perfusion and cerebral metabolic demand (3).

3.3. Pharmacokinetics Across Pediatric Age Groups

Dexmedetomidine pharmacokinetics in children diverge markedly from adult parameters and exhibit substantial variation across the developmental spectrum, from neonatal period through adolescence (5) (2) (1). Recognition of these maturational differences is. Fundamental to establishing appropriate dosing regimens and ensuring safe use in pediatric population.

The drug exhibits high plasma protein binding (approximately 94%) and is eliminated predominantly through hepatic biotransformation, involving both glucoronidation and CYP2A6-dependent hydroxylation pathways (1) (5). Developmental maturation of hepatic enzyme systems significantly influences drug elimination in pediatric patients. In neonates and young infants, incomplete hepatic enzyme development results in diminished clearance capacity and extended elimination half-life relative to older children and adults (5) (9) (1).

Dexmedetomidine clearance is reduced in neonates due to hepatic enzyme immaturity, increases during infancy, peaking at approximately 3 years of age, and subsequently decreases toward adult values in older children (5) (15). Volume of distribution similarly decreases with age, from approximately 2.5L/kg in infants to 1.8 L/kg in school-age children (15). These developmental pharmacokinetic changes support age-stratified dosing strategies, with reduced doses generally indicated to neonates to prevent drug accumulation and prolonged clinical effects (9).

The PROSDEX investigation, a prospective multicenter trial, demonstrated effective sedation in mechanically ventilated pediatric ICU patients across multiple pediatric age groups (16). Complementary data from a Japanese phase 3 study confirmed both the safety and efficacy of dexmedetomidine across pediatric age groups with pharmacokinetic findings consistent with earlier published data (15).

4. Dosing in Pediatric Patients

Dexmedetomidine dosing varies by age, indication, and administration route (9) (5).

Intravenous administration is most commonly used and typically involves a bolus of 0.5-1.0 μg/kg, sometimes up to 2.0 μg/kg in older children and adolescents, administered over approximately 10 minutes, followed by 0.2-1.0 μg/kg/h maintenance infusion (2) (5) (9). The loading dose is frequently omitted in critically ill children to minimize transient hemodynamic perturbations (10) (17). Available data suggest starting at lower doses in infants and young children, with subsequent titration based on clinical response and tolerability (5).

Intranasal dexmedetomidine is commonly used as premedication at doses of 1.0-4.0 μg/kg and has an onset of action of approximately 25-40 minutes (18) (19) (20). Caudal administration at a dose of 1.0 μg/kg is used as an adjuvant to local anesthetics (21).

5. Clinical Efficacy

5.1. Sedation in the Pediatric Intensive Care Unit (PICU)

Dexmedetomidine has been evaluated in multiple studies in the PICU (16) (4). The multicenter PROSDEX study confirmed effective sedation in critically ill children (16). Meta-analytic data indicate that dexmedetomidine reduces the duration of mechanical ventilation by approximately 3.5 hours compared to other sedatives, particularly fentanyl, in postoperative patients (4).

5.2. Procedural Sedation

Dexmedetomidine is effective for sedation in children for diagnostic imaging such as MRI, as well as invasive procedures including bronchoscopy and central venous catheterization (3). Randomized data comparing intranasal premedication agents have shown that ketamine provides faster sedation onset, while dexmedetomidine achieves more effective sedation at 40-50 minutes post-administration (22). Intranasal administration is particularly advantageous for non-invasive procedures, eliminating intravenous access requirements or serving as premedication before more complex procedures (9) (18).

5.3. Perioperative Use

Dexmedetomidine has multiple perioperative roles: premedication, intraoperative adjunct, and postoperative sedation (10). As premedication, intranasal dexmedetomidine effectively reduces preoperative anxiety and facilitates parent-child separation process (23) (6).

Intraoperatively, dexmedetomidine reduces anesthetic and opioid requirements (10). As a caudal adjuvant, dexmedetomidine significantly prolongs postoperative analgesia compared with local anesthetic alone, without increasing adverse effects (21).

5.4. Prevention of Emergence Agitation and Delirium

Emergence agitation and delirium frequently complicate pediatric recovery from general anesthesia, especially following sevoflurane (3) (24). Multiple studies and meta-analyses have confirmed dexmedetomidine effectively reduces the incidence of both emergence agitation (10-26% vs 37-60% with placebo) and emergence delirium compared to midazolam and placebo without clinically significant differences in time of extubation or discharge from the recovery room (6) (10) (25) (24) (26). This protective effect likely stems from dexmedetomidine's unique sedation profile, which resembles natural sleep and enables smoother anesthetic emergence (10).

6. Comparative Effectiveness with Other Sedatives

6.1. Dexmedetomidine vs Midazolam

Meta-analyses indicate that dexmedetomidine significantly lowers emergence agitation rates compared to midazolam (6). In mechanically ventilated PICU patients, higher-dose dexmedetomidine infusion was associated with greater sedation efficacy than midazolam, as reflected by reduced supplemental opioid use and improved sedation adequacy (3). Dexmedetomidine-treated patients exhibited lower heart rates without significant differences in blood pressure (3).

For procedural sedation, intranasal dexmedetomidine achieves higher success rates for diagnostic imaging compared to midazolam. Midazolam offers faster onset, while dexmedetomidine provides longer-lasting sedation with smoother recovery, with an overall favorable safety profile (27).

6.2. Dexmedetomidine vs Propofol

Dexmedetomidine and propofol both provide effective procedural sedation in children, though their clinical profiles differ. Propofol acts more rapidly with quicker recovery, whereas dexmedetomidine offers enhanced respiratory preservation (3) (10). Regarding emergence delirium, pooled data from multiple trials indicate that dexmedetomidine is associated with greater protective benefit than propofol (25). These findings were corroborated by a randomized study in pediatric cleft palate surgery, which demonstrated reduced emergence delirium rates with dexmedetomidine-based sedation (26).

In patients with contraindications to propofol (such as egg allergy), dexmedetomidine combined with midazolam provides a viable alternative for total intravenous anesthesia (28).

6.3. Dexmedetomidine vs Opioids

Meta-analytic data show dexmedetomidine reduces duration of mechanical ventilation compared to fentanyl in postoperative PICU patients (4). Dexmedetomidine's ability to reduce cumulative opioid exposure may decrease risks of tolerance, dependence, and withdrawal (8) (1).

However, dexmedetomidine provides insufficient standalone analgesia for painful procedures and functions best as an adjuvant to opioids rather than a replacement (10) (2).

6.4. Dexmedetomidine s Ketamine

Ketamine and dexmedetomidine exhibit distinct hemodynamic and temporal profiles. Ketamine provides faster sedation onset (effective within 10-30 minutes), while dexmedetomidine achieves more effective sedation at 40-50 minutes post-administration. Dexmedetomidine demonstrates greater reduction in heart rate and blood pressure compared to ketamine, though these changes remain clinically manageable (22).

7. Safety Profile, Adverse Effects, and Withdrawal Syndrome

7.1. Common Adverse Effects

Cardiovascular effects, predominantly bradycardia and hypotension, represent the most frequently encountered adverse events associated with dexmedetomidine (4) (10). Meta-analytic data demonstrate substantially elevated risk of both bradycardia (OR 6.14; 95% CI 2.20-17.12) and hypotension (OR 8.14; 95% CI 1.37-48.31) relative to alternative sedatives in mechanically ventilated pediatric patients (4). These hemodynamic changes exhibit dose-dependency and predominantly manifest in temporal proximity to bolus administration (10) (2).

The PROSDEX study demonstrated that cardiovascular events, although common, were typically self-limiting and easily reversed by dose reduction without requiring cardiopulmonary resuscitation or causing organ damage (16).

7.2. Serious Adverse Events

Severe bradycardia, occasionally progressing to asystole, has been reported in isolated cases in adult population (10) (2). Predisposing factors include pre-existing conduction system abnormalities, concomitant use of negative chronotropic agents, and hypovolemia (10). Meticulous patient evaluation and individualized dosing are essential for safe use (10).

7.3. Risk Minimization Strategies

Adverse event frequency can be reduced by omitting or slowing the loading infusion, allowing sufficient time between dose adjustments, and exercising caution in patients with cardiac disease or prematurity (10). The PROSDEX study confirmed that loading dose administration and higher infusion rates independently increased the odds of hemodynamic changes (16).

7.4. Withdrawal Syndrome

Extended dexmedetomidine administration may lead to withdrawal syndrome following discontinuation, manifesting as psychomotor agitation, increased anxiety, tachycardia and sympathetic rebound (8). Contributing factors include elevated cumulative exposure, prolonged infusion periods, and concurrent sedative-analgesic therapy (8).

Preventive measures include gradual dose reduction rather than sudden discontinuation, minimizing treatment duration where clinically appropriate, and maintaining the minimum effective dosage (8). Notably, the PROSDEX study reported no rebound signs after discontinuation of dexmedetomidine, suggesting that gradual weaning may protect against withdrawal, although further studies are needed (16).

8. Emerging Indications and Future Directions

8.1. Neuroprotection and Organ Protection

Preclinical evidence suggests dexmedetomidine may confer neuroprotective effects through anti-inflammatory, antioxidative, and antiapoptotic mechanisms (7) (10). Unlike GABAergic sedatives and NDMA antagonists, which have raised concerns regarding neurotoxicity and adverse effects on cerebral development in animal studies, dexmedetomidine has been associated with neuroprotective properties (7) (8). Additionally, dexmedetomidine may exert protective effects on the heart and kidneys, potentially mitigating ischemia-reperfusion injury (7). However, robust pediatric-specific clinical evidence remains limited.

A systematic review and meta-analysis evaluating dexmedetomidine in neonates undergoing therapeutic hypothermia for hypoxic-ischemic encephalopathy suggests a potential neuroprotective role of dexmedetomidine in this population, with favorable sedation efficacy and hemodynamic stability (29).

8.2 Alternative Routes of Administration

Beyond intravenous and intranasal administration, buccal and oral routes have been explored for premedication, though oral bioavailability is low (~16%) (2). Non-invasive administration may expand dexmedetomidine's utility in outpatient settings.

8.3. Novel Clinical Applications

Case reports have suggested expanded potential applications od dexmedetomidine use in pediatric practice beyond conventional indications.

Dexmedetomidine has been successfully employed as the primary hypnotic component of total intravenous anesthesia (TIVA) in a 10-year-old child with a family history of malignant hyperthermia and parental refusal of propofol due to child's egg allergy, combined with remifentanil. Anesthesia was maintained with dexmedetomidine at a dose of 0.8-1.0 mg/kg/h and remifentanil at 0.2-0.5 μg/kg/min. The approach provided adequate anesthetic depth, stable hemodynamics, and prompt emergence, illustrating dexmedetomidine's potential as a TIVA component when both volatile anesthetics and propofol are contraindicated (28).

Additionally, dexmedetomidine has been reported as a potentially useful agent in pediatric anticholinergic toxidrome. In a toddler with accidental antihistamine drug ingestion presenting with tachycardia, hypertension, and respiratory compromise, dexmedetomidine (at a dose of 0.3 μg/kg/h) was initiated to avoid benzodiazepine-related respiratory depression, with subsequent hemodynamic stabilization (13).

In a more severe case involving intentional diphenhydramine overdose in a 14-year-old child with prolonged QTc and agitation refractory to lorazepam, dexmedetomidine (at a dose of 0.3-0.7 μg/kg/h) was associated with resolution of anticholinergic symptoms within 13 hours, avoiding the need for physostigmine (13). The sympatholytic properties of dexmedetomidine may counteract the sympathetic hyperactivity characteristic of anticholinergic crisis, while its minimal respiratory depressant effect offers a safety advantage over benzodiazepines in this setting.

9. Conclusions

Dexmedetomidine's distinctive pharmacological profile has made it an increasingly important agent in pediatric sedation and anesthesia. Across intensive care, procedural, and perioperative settings, it appears to provide effective sedation while preserving respiratory function, representing a key advantage over conventional sedatives.

Current evidence primarily supports its role as an adjunct and in some cases a primary sedative, particularly in opioid-sparing strategies and in the prevention of emergence agitation and delirium. Its use is, however, limited by dose-dependent cardiovascular effects, including bradycardia and hypotension, which necessitate careful titration and individualized dosing.

Pharmacokinetic variability across pediatric age groups further emphasizes the need for age-adjusted dosing, particularly in neonates and infants. Although preclinical and early clinical data suggest potential neuroprotective and organ-protective effects, robust pediatric evidence remains limited.

Overall, dexmedetomidine is a versatile sedative agent with an expanding range of clinical applications in pediatrics. Further high-quality studies are needed to better define optimal dosing strategies and long-term safety outcomes.

Conflicts of Interest: The authors declare no conflict of interest.

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