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ORIGINAL ARTICLE

Emergence Agitation: Mechanism, Risk Factors, Assessment and Management

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ABSTRACT
The effects of emergence agitation (EA), also known as emergence delirium, can be clinically substantial. EA's mechanism is yet unknown. Age, male sex, the type of surgery, an emergency procedure, the use of inhalational anesthetics with low blood-gas partition coefficients, a lengthy procedure, anticholinergics, premedication with benzodiazepines, postoperative pain, and the presence of invasive devices are among the proposed risk factors for EA. There are numerous tools available for evaluating EA. However, there are no established best practices for clinical research. Preoperative instruction for surgery, parent-present induction, localized analgesia, multimodal analgesia, propofol, Nefopam, N-methyl-D-aspartate receptor antagonists, and opioid agonists, and total intravenous anesthesia may all aid in preventing EA. However, it might be challenging to recognize high-risk patients and implement preventative strategies in varied therapeutic settings. The techniques of studies and the patients evaluated affect the risk variables and outcomes of preventative interventions. In this review, we cover significant findings from EA research as well as prospective research directions.

Key words: Emergence agitation, Pediatric Anesthesia, Emergence Delirium, Dexmedetomidine

INTRODUCTION

Emergent agitation (EA), which includes agitation, disorientation, excitation, and unintentional movement, is a sign of early recovery from general anesthesia. Age, the assessment method utilized, definitions, anesthetic practices, the type of operation, and the timing of the EA assessment during recovery all affect the frequency of EA, which ranges from about 0.25% to 90.5%. The effects of EA on the clinical setting are also diverse. Since it often resolves on its own and is short-lived, its clinical effects are frequently seen as being modest**(1)**. Nevertheless, it might result in clinically significant side effects, harm from getting out of bed, harm to the patient or their medical personnel, surgery-related bleeding, inadvertent removal of intravenous catheters or drains, unintended extubation, and respiratory depression **(2)**.

Emergence delirium (ED), a state of acute confusion that can occur after anesthetic recovery, can include disorientation, hallucinations, restlessness, and uncontrolled, hyperactive physical behavior in patients. ED symptoms can be hypoactive, mixed, or hyperactive indications resembling agitation, which makes it not entirely equal to EA. But in many research, the words EA and ED have been used interchangeably. Additionally, the same evaluation instruments (such as the Riker Sedation-Agitation Scale **(3)** Both of these states have been measured using the Richmond Agitation-Sedation Scale (4). Postoperative delirium should be distinguished from EA and ED (5).

ED is a component of postoperative delirium; it denotes the condition's early onset in the operating room or upon entry into the postanesthesia care unit (PACU) right after the anesthetic period. Strong indicators of postoperative delirium in the PACU include EA and ED. Postoperative delirium is linked to a longer hospital stay, greater morbidity (for example, pulmonary problems), death, and the requirement for institutionalizing patients. As in earlier research (6).

Mechanism of emergence agitation:

It is unclear what specific pathophysiological mechanism causes EA after general anesthesia. High levels of worry before surgery, unusual surroundings, being away from parents, and interactions with unknown medical personnel are some of the proposed reasons of EA in children. These could lead to an increase in sympathetic

tone and a prolongation of the aroused state during the anesthetic recovery period **(7)**.

The incidence of EA in children has grown with the advent of volatile medications with low blood solubility, such as sevoflurane and desflurane. This has been explained as being caused by different rates at which inhalational anesthetics are cleared from the central nervous system by sevoflurane and desflurane; while auditory and motor functions recover first, cognitive function recovers later, resulting in EA. Additionally, it has been postulated that sevoflurane anesthesia, This results in clinically undetectable sevoflurane-induced epileptogenic activity, elevated lactate and glucose levels in the parietal cortex, and EA **(8,9)**.

Proposed risk factors for emergence agitation:

definitions, study strategies (such as prospective observational studies, prospective randomized controlled experiments, or retrospective studies), and so on. Various risk factors for EA in children have been suggested. Preschool age (2–5 years), lack of prior hospitalization, surgery, or high number of treatments, patient pre-existing behavior, psychological immaturity, preoperative anxiety, parental anxiety, and lack of premedication (with midazolam) are all possible risk factors for EA in children **(10)**.

The following are possible risk factors for EA in children: Sexual orientation, obesity (30 kg/m2), the quantity of times an intubation attempt was made, the kind of surgery, the emergency procedure, the type of anesthesia used (inhalation anesthesia), the length of the procedure or the duration of the anesthesia. Review the most common risk factors and other important issues covered in the literature **Table 1 (11)**.

1. Age

EA is more common in children than in adults. There was an inverse connection between age and the prevalence of EA in a study of children aged 2 to 12. In a prospective cohort study, children aged 3 to 10 years were found to have an elevated risk of preoperative anxiety. Children with preoperative anxiety experienced more EA after surgery than children without it **(12)**.

2. Sex

It is unknown how sex affects children's EA. However, sex was not connected to the prevalence of EA in a prospective observational study of young infants. However, numerous studies have found a connection between EA and male sex. Men are more likely to develop EA due to weaker pain tolerance and a significant link between postoperative pain and sex. Male sex is another risk factor for catheter-related bladder discomfort, which is characterized by frequent, urgent urination. Vaing urgency is a separate risk factor for EA **(13)**.

3. Surgery

In a prospective cohort research involving 521 kids aged 3 to 7 years, it was discovered that ophthalmological and otorhinolaryngological operations were linked to EA. Otorhinolaryngological procedures, in particular, were separate EA risk factors. Similarly, strabismus surgery and tonsillectomy have been recognized as risk factors for EA in early newborns**(14)**.

Depending on whether elective or emergency surgery was done, varying results have been recorded. In a study from 2006, **Lepouse et al. (15)** discovered that the prevalence of EA was unaffected by emergency surgery. A 2019 investigation, **Ramroop et al. (16)** discovered that when compared to elective surgery, Emergency medical intervention raised the risk of postoperative EA. EA is more common among people receiving emergency surgery, according to the investigators, may have been caused by heightened anxiety and untreated physiological disturbances.

Duration of surgery/anesthesia

The duration of the anesthesia is influenced by the length of the procedure. Studies that suggest longer anesthesia or surgical durations are risk factors for EA should be taken cautiously because just one of these variables (i.e., anesthesia time or surgical duration) may have been studied and assessed in the specific study. A research that considered these parameters found that patients with EA needed much more time during surgery and anesthesia than patients without EA**(17)**.

4. Inhalational anesthetics

Sevoflurane and desflurane are examples of inhalational anesthetics with low blood-gas solubility, are more frequently associated with EA than halothane, isoflurane, desflurane, and sevoflurane. Desflurane and sevoflurane both caused EA in pediatric patients, although desflurane did so more frequently **(18-20)**.

5. Neuromuscular blocking agents and reversal agents

Anticholinergic drugs like atropine and scopolamine are well recognized to increase the risk of EA. For general anesthesia, cholinesterase inhibitors like pyridostigmine and neostigmine, anticholinergics like glycopyrrolate and atropine, and sugammadex are frequently used as neuromuscular blocking and reversal drugs. Meanwhile, just a few randomized controlled trials have been conducted to assess the impact of

neuromuscular blocking or reversing medications on EA **(21)**.

In a prospective randomized controlled research, rocuronium-sugammadex was found to be more effective than succinylcholine at reducing the frequency, severity, and duration of EA in patients undergoing nasal bone fracture closure reduction. According to the authors, higher lactate and potassium concentrations, insufficient neuromuscular blockade during surgery, elevated intraocular pressure, and histamine release as a result of succinylcholine administration may have been more harmful to EA than rocuronium-sugammadex. Studies show that cholinesterase inhibitors and sugamadex have various effects on EA(22).

6. Pain

Despite the fact that EA has been reported in the absence of pain-free therapy and may occur regardless of pain intensity, pain is a substantial risk factor for EA in children. The findings suggest that EA and postoperative pain are different clinical occurrences; however, distinguishing between EA and behavioral changes caused by postoperative pain can be difficult. A score of less than five points on a numerical rating scale used to gauge postoperative pain was found to increase the likelihood of EA. However, EA might make the pain after surgery worse. Consequently, effective perioperative pain management may affect the onset of EA **(12)**.

7. Presence of invasive devices

The presence of intrusive devices (such as urine catheters, nasogastric tubes, chest tubes, and tracheal tubes) during emergence is a well-known risk factor for EA. Patients who have it during emergence may feel humiliated, distressed, uncomfortable, and painful. By using more opioids and benzodiazepines in the PACU, it may also make delirium worse. **(23)**.

Prediction of emergence agitation

Preventive care is advised above curative treatments in this circumstance because EA can have significant negative effects for patients and increase the burden of patient care. The EA risk scale was recently developed and validated for children receiving sevoflurane anesthesia by **Hino et al. (14)** in a single-center investigation. Pediatric Anesthesia, Age Its four domains are behavior score, surgical procedure, and anesthetic duration. The EA risk scale worked admirably in terms of prediction. As a result, the EA risk scale can be used to predict and prevent EA in pediatric patients after sevoflurane anesthesia. Patients undergoing anesthesia with medicines other than sevoflurane, on the other hand, are not permitted to use the EA risk scale. More research is needed to demonstrate external validity at other hospitals. It would also be helpful for the effective prevention of EA to discover a biomarker that might indicate the presence of EA based on the analysis of preoperative blood samples. Patients with EA had considerably higher plasma levels of the brain-derived neurotrophic factor (BDNF) measured after blood sample at skin closure in elderly patients having gastrointestinal surgery **(24,25)**.

Assessment Tools for Emergence Agitation (11)

For the assessment of agitation and sedation, a variety of scoring systems or instruments are available, however they are mostly intended for use in psychiatric or acute care settings. To compare the qualities and characteristics of the various instruments employed, an overview of the scoring methods that are now accessible is given (Table 2) **(26)**.

Although other scales and variations have been offered as instruments for measuring EA in children, the Pediatric Anesthesia Emergence Delirium (PAED) scale was developed in 2004 and is the most commonly used scale in pediatric EA studies. It offers a score between 0 and 20, and it is said to be valid for evaluating children's EA. However, the PAED scale has limitations, such as poor inter-rater reliability and intrinsic subjectivity in judging each behavior item. Furthermore, the criterion for detecting the presence of EA is controversial. According to Bong and Ng (27), a PAED score of 10 or less is the suitable cutoff for EA (17). According to Bajwa et al. (28), a PAED score greater than 12 demonstrated greater sensitivity and specificity in the evaluation of EA PAED. A PAED score of less than 16 was used as an indicator of EA in another investigation without a clear justification **(11)**.

The evaluation site (e.g., operating room vs. PACU), assessment technique (e.g., RASS), and definition of EA (e.g., RASS $\geq +1$ vs. $\geq +2$ vs. $\geq +3$) all had an impact on the reported incidence of EA. The reported incidence of EA in the operating room was higher than in the PACU when patients were waking up from general anesthesia (e.g., 3.7% vs. 1.3% and 54.3% vs. 28.6%, respectively) (29). **Jee et al. (7)** and Ham et al. (31) accepted RASS $\geq +2$ as an indicator of EA, and the majority of other groups defined RASS +1 as an indicator of EA, While **Fields et al. (30)** utilized RASS +3 as an indicator of EA.

Pediatric Anesthesia Emergence Delirium Scale

The latter scale, known as the Pediatric Anesthesia Emergence Delirium Scale (PAEDS), has been suggested for use in identifying ED in children and adolescents. However, the PAEDS diagnostic accuracy criteria have varied substantially between investigations, with sensitivity ranging from 64% to 100% and specificity ranging from 80% to 98% **table 3 (32)**.

Richmond Agitation Sedation Scale :

A person's level of agitation or sedation can be measured using the Richmond Agitation-Sedation Scale (RASS), a medical scale. It was developed via the efforts of several professionals, including medical professionals, nurses, and pharmacists**(34)**.

All hospitalized patients can utilize the RASS to rate their level of alertness or agitation. To avoid over- and under-sedation, it is usually applied to patients who are mechanically ventilated. The Confusion Assessment Method in the ICU (CAM-ICU), a technique to identify delirium in patients of intensive care units, begins with obtaining a RASS score. One of the various sedative scales used in medicine is the RASS. The Ramsay scale, the Sedation-Agitation-Scale, and the Comfort scale for pediatric patients are further scales **Table 4 (35)**.

Development of a Modified Version of the Agitation Severity Scale (MASS) (36)

The scale was revised by a group of psychiatrists, a PhD biostatistician, and an experimental psychologist. The original Agitation Severity Scale assessed agitation in five areas: verbal actions, nonverbal face expressions, purposeful and nonpurposeful movement behaviors, and interpersonal behaviors. There were 4 to 6 distinct behaviors mentioned for each of these five domains, making a total of 25 things. Scores ranging from 0 to 75 on Likert scales were used to rank these behaviors **(36)**.

Prevention and treatment of EA

Prevention of EA

Here is a summary of EA prevention techniques, broken down into pharmaceutical and non-pharmacological approaches (Table 5). Studies comparing the preventive effects of various medications or agents on EA should be interpreted with caution, as the same medications may not have the same effects depending on dosage, administration strategy (e.g., continuous infusion versus single bolus), time of administration, or individuals (e.g., children) **(11)**.

Treatment

1. **Pharmacological methods:**

Choice of anesthesia methods

Balanced anesthesia, inhalational anesthesia, and total intravenous anesthesia (TIVA) are a few different anesthetic techniques. In a randomized controlled study, TIVA with propofol and remifentanil reduced EA following strabismus surgery in children aged 2 to 6 years as compared to volatile induction and maintenance of anesthesia with sevoflurane.. However, there have been inconsistent findings regarding the impact of inhalation vs balanced anesthetic on EA in pediatric patients **(37)**. It is likely that this was caused by several surgical procedures (such as adenotonsillectomy, fiberoptic bronchoscopy, and balanced anesthetic regimens like sevoflurane-remifentanil or sevoflurane-fentanyl), and EA assessment tools. Additionally, even with the same regimen, the amounts of medications delivered can affect how balanced anesthesia affects EA. Therefore, additional research is required to establish the ideal dosages for efficient EA prevention **(38,39)**.

Propofol

The ideal medication for both preventing and treating EA in pediatric patients is propofol. Propofol shown a preventive Depending on the timing of the medication, a meta-analysis of pediatric patients found an effect against EA.. Following the induction of anesthesia, a 2 mg/kg intravenous bolus of propofol was given, however this did not lessen EA following desflurane anesthesia. Contrarily, adding a bolus of propofol at the end of surgery or giving it continuously throughout anesthesia maintenance had a protective effect against EA in children receiving general anesthetic. The fast pharmacokinetics of propofol can primarily be blamed for these effects **(40)**.

In pediatric patients, continuous infusion of propofol alone during maintenance of anesthesia reduced the incidence of EA in patients having closure reduction of nasal bone fracture compared to sevoflurane anesthesia. Therefore, additional investigation into this aspect of propofol use is required **(41)**.

Opioids

The prophylactic injection of -opioid agonists such fentanyl, sufentanil, alfentanil, or remifentanil was observed to minimize the incidence of EA following sevoflurane anesthesia in a meta-analysis of 19 randomized controlled studies with 1528 children **(42)**.

Ketamine

Ketamine is a noncompetitive sedative, amnestic, and analgesic antagonist of the N-methyl-D-aspartate (NMDA) receptor. After sevoflurane anesthesia, ketamine (0.25 mg/kg and 0.5 mg/kg)

was given to children 10 minutes before the completion of the procedure to help avoid EA without delaying recovery. Ketamine did not significantly differ in the incidence of EA between 0.25 mg/kg and 0.5 mg/kg, however the patients' pain levels dropped as the ketamine dose increased. Injecting 0.5 mg/kg of ketamine 20 minutes prior to the conclusion of surgery into individuals enduring sevoflurane anesthesia helped to prevent EA following rhinoplasty, however the anesthesia time was extended because of the delayed recovery **(12)**.

Magnesium sulfate

As a noncompetitive NMDA receptor antagonist, magnesium sulfate has sedative, neuroprotective, and analgesic-saving actions in the brain. (54, 55). Magnesium sulfate (30 mg/kg) given 10 minutes prior to the conclusion of surgery in a research involving children (3–16 years old) did not lessen EA following sevoflurane anesthesia. Contrarily, in young children (4–7 years old) undergoing the same surgery (adenotonsillectomy) under sevoflurane anesthesia, a 30 mg/kg bolus with a continuous infusion of 10 mg/kg/h (from the commencement of the procedure to the time the surgery was ended) reduced the incidence and severity of EA. (56). The authors proposed that the neuroprotective and anticonvulsant properties of magnesium sulfate may have reduced the incidence of EA **(57)**.

Tramadol

Atypical centrally acting opioid tramadol inhibits NMDA receptors as well as M1 and M3 muscarinic and nicotinic acetylcholine and nicotinic acetylcholine receptors. In a retrospective cohort study, it was discovered that giving patients having nasal surgery a single intravenous dosage of tramadol (2 mg/kg) at the beginning of the procedure lowers the likelihood that they will experience EA following sevoflurane anesthesia. The authors hypothesized that tramadol's analgesic, antitussive, and antishivering properties, along with its capacity to lessen voiding urgency, may have contributed to the avoidance of EA. When compared to 1 g/kg of dexmedetomidine given in the same way, 2 mg/kg of tramadol intravenously infused after tracheal intubation for 10 minutes showed a similar protective effect against EA in adenotonsillectomy procedures **(43)**.

Nefopam

Nefopam is a non-narcotic analgesic with a central action. Nefopam possesses anticonvulsant, depressive, anti-shivering, and opioid-sparing actions because it regulates glutaminergic transmission by blocking postsynaptic NMDA receptors and serotonin and noradrenaline reuptake. Patients undergoing nose surgery discovered that a 20 mg nefopam infusion given for 20 minutes just after the induction of anesthesia was beneficial in lowering the frequency and severity of EA during desflurane anesthesia **(7)**.

α2-adrenoreceptor agonists

Adrenoreceptor agonists having sympatholytic, analgesic, and sedative properties include 2-clonidine and dexmedetomidine. In a double-blind trial, it was discovered that administering 2 g/kg of clonidine intravenously after inducing anesthesia significantly decreased the incidence and severity of sevoflurane-induced EA in young male volunteers. Furthermore, premedication with clonidine was discovered to be superior to premedication with midazolam for lowering EA in a meta-analysis that focused on clonidine as a premedication agent in children **(44)**.

Dexmedetomidine is a very selective 2-adrenoreceptor agonist with a 7–8-times higher affinity for the 2-adrenoreceptor than clonidine. Dexmedetomidine was reported to have a lower incidence of EA compared to a placebo in a meta-analysis examining its effects on EA following sevoflurane anesthesia in children, but it was also linked to a longer recovery time. Compared to ketamine, propofol, clonidine, midazolam, fentanyl, and sufentanil, dexmedetomidine was the medicine that prevented EA the best in a network meta-analysis evaluating the effects of anesthetic adjuvants for sevoflurane anesthesia in children**(45)**.

Benzodiazepines

Midazolam in particular is frequently used as a premedication to induce amnesia, drowsiness, and anxiolysis in children. The effects of preoperative midazolam treatment on EA in pediatric patients varied. In particular, prophylactic midazolam treatment in children receiving sevoflurane, desflurane, or combined anesthesia had no protective benefit against EA, according to a meta-analysis published in 2010. Another meta-analysis, however, found that preemptive midazolam delivery decreased sevoflurane-induced EA (published in 2013) **(46)**.

It's interesting to observe that midazolam administration during surgery decreased EA in both kids, in contrast to the effects of benzodiazepine premedication. Following an intravenous dosage of midazolam (0.03 mg/kg) given just before the procedure was ended, children having sevoflurane anesthesia for strabismus surgery showed decreased EA. Furthermore, in patients undergoing nasal surgery

under sevoflurane anesthesia, midazolam infusion from 15 minutes before anesthesia induction through the end of the procedure demonstrated an EA reduction effect comparable to that of dexmedetomidine infusion **(47)**.

Regional analgesic techniques

Numerous research have been done to determine whether proper pain management with localized blocking can decrease the incidence and/or severity of EA while lowering the harmful effects of systemic analgesics. Postoperative pain is a significant risk factor for EA. Preoperative caudal block was reported to lower the incidence of EA compared to intraoperative intravenous fentanyl in a prospective randomized double-blind trial of children aged 2 to 6 undergoing inguinal hernia surgery under sevoflurane anesthesia (4.5% vs. 59%, respectively) **(48)**.

Peripheral nerve blocking also decreased the frequency or severity of EA in pediatric patients. in youngsters having lateral or anterior thigh orthopedic surgery. **Wang et al. (49)** EA was thought to have diminished as a result of less intraoperative sevoflurane use and a reduction in discomfort from the infraorbital nerve block that was performed prior to surgery. A randomized controlled study employing different sevoflurane dosages, however, was unable to show a significant drop in EA in young patients **(50).** More research is required to determine how localized analgesia reduces EA.

Multimodal analgesia

Examples of multimodal analgesia include ketamine, magnesium, tramadol, nefopam, 2-adrenoreceptor agonists (such as clonidine or dexmedetomidine), nonsteroidal anti-inflammatory medications (such as ketorolac), dexamethasone, and localized analgesia. However, the impact of multimodal analgesic regimens on EA has only been studied in a small number of trials. In a prospective, randomized, double-blind research, it was discovered that low-dose intravenous ketamine (0.15 mg/kg) and dexmedetomidine (0.3 g/kg) reduced the occurrence and severity of EA in young patients having adenotonsillectomy under sevoflurane anesthesia when compared to volume-matched saline**(51)**.

Non-pharmacological methods:

Preoperative anxiety and recuperation in unfamiliar settings are risk factors for EA in pediatric patients. In children aged 1-3 years receiving sevoflurane anesthesia, parental presence during the induction of anesthesia was demonstrated to improve the efficacy of oral midazolam on EA; parental presence during the patient's arrival in the PACU is also anticipated to help minimize EA in pediatric patients. It has also been discovered that family-centered behavioral preparation for surgery, which includes preoperative instruction and training for kids and their parents, lowers the incidence of EA in pediatric patients between the ages of 2 and 10 years. This was found to be more effective than oral midazolam administration (0.5 mg/kg) at 30 minutes prior to surgery**(52)**.

Table 1. Possible Risk Factors for Emergence Agitation (11)

Risk factor	Children
Patient related	Preschool age (2–5 years) No previous surgery Hospitalization or high number of previous interventions Poor adaptability Attention-deficit hyperactivity disorder Patient preexisting behavior Psychological immaturity Preoperative anxiety Parental anxiety Patient and parent interaction with healthcare providers
Anesthesia related	Lack of premedication (with midazolam) Paradoxical reaction to midazolam stated in child's medical history Use of inhalational anesthetics with low blood–gas partition coefficients (e.g., sevoflurane and desflurane) Excessively rapid awakening (in a hostile environment) Pain
Surgery related	Type of surgery

Table 2. Currently available sedation scores (26)

Applicable to ED	*Sedation*	*Agitation*	*Features*	*Name of score*	*Acronym*
Yes	Yes	Yes	7-point scale (+3 to −3) 2 descriptors: responsiveness and speech	Sedation assessment tool	***SAT***
Yes	Yes	Yes	7-point score (1–7) Only 1 descriptor only	Behavioural activity rating scale	***BARS***
Yes	Yes	Yes	9-point score (−4 to +4) 4 descriptors: responsiveness, speech, facial expression	Altered mental status score	***AMSS***
Yes	Yes limited	Yes	6-point scale (1–6 'combative' to 'deep sleep') 1 descriptor only Only 2 levels of sedation	Sedation scale	***RSS***
Yes	No	Yes	5-point score(1–5) of agitation 1 descriptor only, no sedation scale	Combativeness scale	***CS***
Yes	No	Yes	6-point score (0–5) 1 descriptor only, no sedation scale	Acute arousal scale	***AAS***
No – ICU	Yes	Yes	10-point score (+4 to −5) 2 descriptors 'Term' and 'description'	Richmond agitation sedation scale	***RASS***
Yes	No	Yes	4-point scale of aggression 1 descriptor only, no sedation scale	Overt aggressive scale	***OAS***
Yes	No	Yes	6-point agitation scale. 1 descriptor only, no sedation scale	Agitation scale	***AS***
No – psychiatry	No	Yes	7-point score (1–7) 24 descriptors	Brief psychiatric rating scale	***BPRS***
No – ICU	No	Yes	6-point scale (1–5) 47 descriptors with 12 sub-categories	Overt agitation severity scale	***OASS***
Yes	Yes	No	4-point scale (1; deep sleep to 5; alert) 4 descriptors 'Responsiveness Speech Expression Eyes'	Observers assessment of alertness/sedation	***OAA/S***
No – ICU	Yes	Yes	2-point scale (absent or present) 4 descriptors with sub-categories	Confusion assessment method	***CAM-ICU***
No – psychiatric	Yes	No	7-point scale (0; absent–6 extreme) Four 45 min clinical interviews.	Positive and negative syndrome scale	***PANSS***
No – ICU	Yes	No	6-point scale of sedation(1−6) 1 descriptor only	Ramsay assessment scale	***RAS***
No – neuro ICU	No	Yes	4-point scale (1–4 absent, slight, moderate, extreme) 14 criteria 3 underlying subscales	Agitated behavioural scale	***ABS***
No –	Yes	No	5-point scale alertness scale	Alertness scale	***AS***

Applicable to ED	*Sedation*	*Agitation*	*Features*	*Name of score*	*Acronym*
anaesthesia			8 visual and 12 auditory stimuli − 6 sounds and 6 words		
Yes – neurological	Yes	No	4-point scale of responsiveness 1 descriptor only	Alert, verbal, painful, unresponsive	***AVPU***
Yes	Yes	No	15-point score 3 descriptors: (eyes 1–4; motor 1–6; verbal 1–5)	Glasgow Coma Scale	***GCS***
No – ICU	Yes	No	20-point score 7 descriptors: (eyes 0–4; motor 0–4; brainstem 0–4; respiration 0–4)	Full outline of unresponsive score coma scale	***FOUR***
No	Yes	No	4 levels 1 descriptor only	Alert confused drowsy unresponsive	***ACDU***
Yes	No	Yes	5 levels only 1 descriptor: relaxed to coma	Grading of CNS stimulation	***CNS***
No	Yes	Yes	6 levels 1 complex descriptor	Motor activity assessment scale	***MAAS***

Table 3. Pediatric Anesthesia Emergence Delirium (PAED) scales (33).

Point	Description of items	Not at all	Just a little	Quite a bit	Very much	extremely
1	The child makes eye contact with the caregiver	4	3	2	1	0
2	The child's actions are purposeful	4	3	2	1	0
3	The child is aware of his/her surroundings	4	3	2	1	0
4	The child is restless	0	1	2	3	4
5	The child is inconsolable	0	1	2	3	4

Table 4. The Richmond Agitation–Sedation Scale (RASS) (34).

Description	Term	Score
Overtly combative or violent; immediate danger to staff	Combative	**+4**
Pulls on or removes tube(s) or catheter(s) or has aggressive behavior toward staff	Very agitated	**+3**
Frequent nonpurposeful movement or patient–ventilator dyssynchrony	Agitated	**+2**
Anxious or apprehensive but movements not aggressive or vigorous	Restless	**+1**
Spontaneously pays attention to caregiver	Alert and calm	**0**
Not fully alert, but has sustained (more than 10 seconds) awakening, with eye contact, to voice	Drowsy	**-1**

Description	Term	Score
Briefly (less than 10 seconds) awakens with eye contact to voice	Light sedation	**-2**
Any movement (but no eye contact) to voice	Moderate sedation	**-3**
No response to voice, but any movement to physical stimulation	Deep sedation	**-4**
No response to voice or physical stimulation	Unarousable	**-5**

Table 5. Strategies to Prevent Emergence Agitation (11)

Pharmacological methods
Total intravenous anesthesia
Propofol
Opioids
Ketamine
Magnesium sulfate
Tramadol
Nefopam
Dexmedetomidine
Regional analgesia
Multimodal analgesia
Avoidance of premedication with benzodiazepine
Non-pharmacological methods
Informing the patient of predictable pain or discomfort prior to anesthesia
Removing indwelling invasive devices as early as possible
Parental presence during induction of anesthesia and recovery (in pediatric patients)
Family-centered behavioral preparation for surgery

CONCLUSIONS

We may draw the conclusion that magnesium sulfate bolus and infusion are safe and effective in lowering the incidence of emerging agitation in children after adenotonsillectomy. Dexmedetomidine had better analgesic effects than magnesium sulfate did at reducing the frequency and intensity of postoperative emergence agitation (EA).

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