"COMPARISON OF TWO DIFFERENT DOSES OF DEXMEDETOMIDINE IN ATTENUATION OF SEVOFLURANE ASSOCIATED EMERGENCE AGITATION IN PAEDIATRIC PATIENTS, POSTED FOR LOWER ABDOMINAL SURGERIES –A HOSPITAL BASED, RANDOMIZED CONTROLLED TRIAL".

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With reference to the above, we wish to inform you that your proposed research project titled **"COMPARISON OF TWO DIFFERENT DOSES OF DEXMEDETOMIDINE IN ATTENUATION OF SEVOFLURANCE ASSOCIATED EMERGENCE AGITATION IN PAEDIATRIC PATIENTS, POSTED FOR LOWER ABDOMINAL SURGERIES.- A HOSPITAL BASED, RANDOMIZED CONTROLLED TRIAL",** is ethical and justifiable.

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LIST OF ABBREVIATIONS

ASA	–	American society of Anaesthesiologists
HR	–	Heart rate (bpm)
SBP	–	Systolic Blood Pressure (mm Hg)
DBP	–	Diastolic Blood Pressure (mm Hg)
SpO_2	–	Saturation of peripheral oxygen (%)
MAP	–	Mean arterial pressure
EA	-	Emergence agitation
ED	-	Emergence delirium
PAED	-	Paediatric Anaesthesia Emergence Delirium
VAS	–	Visual Analog Score
ICU	–	Intensive Care Unit
PACU	–	Post Anaesthesia Care Unit
ECG	–	Electrocardiogram
I.V	–	Intravenous
mcg	–	Micrograms
min.	–	Minutes
kg	–	Kilograms
mm Hg	–	Millimetres of mercury
ICP	-	Intracranial pressure
TIVA	-	Total intravenous anaesthesia
DEX	-	Dexmedetomidine
ng	-	Nanogram
mL	-	Millilitre
h	-	hour
PICU	-	Paediatric intensive care unit
L	-	Litre
mg	-	milligrams
PLMA	-	ProSeal Laryngeal mask airway

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ABSTRACT

TITLE: "COMPARISON OF TWO DIFFERENT DOSES OF DEXMEDETOMIDINE IN ATTENUATION OF SEVOFLURANE ASSOCIATED EMERGENCE AGITATION IN PAEDIATRIC PATIENTS, POSTED FOR LOWER ABDOMINAL SURGERIES –A HOSPITAL BASED, RANDOMIZED CONTROLLED TRIAL".

BACKGROUND:

Emergence Agitation (EA) is a very common immediate postoperative complication encountered in paediatric population undergoing anaesthesia with Sevoflurane. Owing to its low blood solubility and rapid recovery, Sevoflurane is the best inhalational agent for use in paediatric age group. But its disadvantage of causing agitation is distressing to the anaesthesiologists. Dexmedetomidine is found to reduce the incidence of EA. Fewer studies have been done comparing the effects of particularly two doses (0.3 mcg/kg and 0.5 mcg/kg) of Dexmedetomidine. Hence this study was designed to compare the effectiveness of these two doses in reducing EA in children.

AIM:

To assess the effectiveness of two different doses of Dexmedetomidine 0.3 mcg/kg IV and 0.5 mcg/kg IV, given intraoperatively in reducing postop EA along with the changes in hemodynamic parameters after drug administration.

MATERIALS AND METHODS:

The study is a prospective double-blinded randomised study done on 60 children, belonging to ASA I and II, aged 2-10 years undergoing lower abdominal surgeries under sevoflurane anaesthesia. After obtaining informed consent, patients in Group D 0.3 (n = 30) received 0.3 µg/kg and patients in group D 0.5 (n = 30) received

0.5 µg/kg infusion of dexmedetomidine, 10 mins prior to end of surgery. Postoperatively, in the PACU, charting of PAED score, Watcha scale, Cravero scale and haemodynamic parameters was done at 10 mins, 20 mins, 30 mins, 40 mins, 50 mins and 60 mins post operatively.

The data was analysed using statistical software R version 4.4.1. and Microsoft Excel. Chi square test, Shapiro Wilk test, sample t-test, Mann Whitney U test and Friedman test were used wherever appropriate.

RESULT:

The reduction in incidence and severity of EA was comparable in both Group D 0.3 and D0.5 (p value >0.05) with statistically significant reduction in agitation scores i.e., PAED score, Watcha scale and Cravero scale, in both groups (p value <0.001). Variability in hemodynamic parameters, i.e. MAP,HR and RR, were statistically not significant in both groups (p>0.005) or within each group.

CONCLUSION:

This study concludes that both 0.3 mcg/kg and 0.5 mcg/kg of intravenous dexmedetomidine are comparable, hemodynamically stable and effective in reducing emergence agitation in paediatric patients undergoing lower abdominal surgeries under sevoflurane anaesthesia.

Keywords: lower abdominal surgeries, emergence agitation, caudal anaesthesia, paediatric, dexmedetomidine, sevoflurane

INTRODUCTION

Anaesthetic management of paediatric patients is uniquely challenging. Perioperative care of paediatric patients can be a real concern. Approach to the paediatric patient must consider heightened preoperative anxiety, of both the patient and parents, and its postoperative behavioural and pain effects which requires diligent handling.

Paediatric anaesthesia presents with various perioperative challenges such as airway challenges(bronchospasm,laryngospasm), pain management, thermoregulation concerns, fluid and electrolyte management and delayed recovery. One such commonly encountered challenge in pediatric anesthesia is that of 'emergence delirium (ED)', also referred to as 'emergence agitation (EA)'.

Emergence agitation is a complication that appears in the immediate post operative period that occurs in paediatric patients on emergence from general anaesthesia. EA is a state of altered consciousness where generally the patients are disoriented with hyperactive behavioural symptoms & hypersensitivity in the early post-operative period [2,3]. It usually presents in the form of agitated behaviour & non-purposeful movement which includes "kicking, pulling & flailing", as well as inability to maintain or establish eye contact and lack of self-awareness. The occurrence of ED in the contemporary paediatric anaesthesia practice is not totally clear. Previous studies have reported an incidence ranging from as low as two percent going up till eighty percent in certain other studies [4,5]. ED can put patients, their families, and healthcare providers to various physical, emotional, and financial challenges. Although the exact causes of ED are not fully understood, factors such as pre-school children, exposure to low-solubility volatile anaesthetics, pre-surgery anxiety, and specific types of surgeries can increase the risk. While many instances of

ED resolve naturally without intervention and self-limiting, medication may be necessary in some cases.

The present contemporary practice of pre-emptive administration of analgesics, sedatives ('dexmedetomidine') [6], and other drugs/agents have contributed to a reduction in the occurrence of ED. The frequency of ED is influenced by various factors, such as the child's age, the 'type of anaesthetic agent' administered, the 'nature of the surgery or procedure', and the diagnostic criteria used for evaluation. It commonly develops shortly after regaining consciousness from general anaesthesia, but in some cases, it may be delayed for up to 45 minutes.

The exact causes of ED remain unclear, maybe multifactorial. One suggested theory is that ED arises due to the rapid redistribution of the anaesthetic. However, the link between a quick recovery from general anaesthesia and the increased risk of ED has been inconsistently observed. Inhaled anaesthetics, particularly sevoflurane, have been shown to stimulate 'locus ceruleus neurons' in animal studies involving mice, an area associated with adrenergic activation. This effect could also possibly have a role to play in the occurrence of ED[7].

Variety of risk factors have been found to be related with 'ED', with the condition most frequently occurring in preschool-aged children, typically between 2 and 6 years old [8]. Studies have yielded conflicting results, with some indicating a higher risk in male patients, while others report no significant gender differences. Additionally, specific procedures, like those involving the ear, nose, throat, or eyes, may increase the likelihood of ED [5,9,10,11]. Anaesthetic agents: The use of newer, less soluble volatile anaesthetics like 'sevoflurane' and 'desflurane' has been linked to a increased incidence of 'ED'. The anaesthetics most commonly associated with ED, in order of likelihood, are sevoflurane > desflurane > isoflurane > propofol > halothane.

Additionally, preoperative anxiety is a significant risk factor. The risk of 'ED' can be elevated by the presence of 'anxiety' in either /or both parents and the patients. Since anxiety is a known risk factor for 'ED', individuals with typical temperaments or behavioural traits which may be previously existing are more prone and subjected to experience heightened 'preoperative anxiety', increasing their risk. The duration and depth of anaesthesia do not influence the risk of ED. ED can occur equally after short procedures (such as tympanostomy and tube placement) as it can following longer surgeries[5,10].

ED is a clinical diagnosis. The diagnosis of 'ED' is often invalidated by the presence of pain in the post-op period. It is associated with 'neurological symptoms and terms that amount to a discreet postoperative neurological state, characterised by core neurological and behavioural symptoms: motor agitation, confusion, and lack of recognition or appropriate interaction' with their surrounding environment starting from emergence to up to forty-five minutes after the termination of anaesthesia. Various different descriptive scales, such as the 'Cravero and Watcha scales', have been used to quantify the severity. One of the most commonly used tools is the 'Paediatric Anaesthesia Emergence Delirium (PAED) scale', which reports a sensitivity of around sixty four percent and a specificity of eighty six percent [12].

Some of the differential diagnoses could include 'hypercarbia, hypocarbia, pain, hypo or hyperglycaemia, hypotension or increased ICP'.

ED is typically self-limiting and may resolve on its own without the need for pharmacologic treatment. In most cases, ED resolves with supportive care and measures to prevent harm. Whenever possible, parents and guardians, caretakers should participate actively in the decisions involving treatment among others. When pharmacological intervention is required, it often involves administration of

medications such as dexmedetomidine, propofol, ketamine, or opioids like fentanyl [11,13,14].

Among these medications, DEX is a 'potent and highly selective α2-adrenoreceptor agonist' commonly used in paediatric anaesthesia. It is favoured for its 'sedative, analgesic, amnesic, anxiolytic, and sympatholytic properties' with minimal impact on respiratory function. Recently, DEX has gained attention in paediatric clinical settings for its potential to protect organs and maintain neurocognitive function. Numerous clinical studies have demonstrated the effectiveness of DEX in preventing emergence agitation (EA) through various routes of administration and dosage regimens. [15,16].

'Total intravenous anaesthesia (TIVA)' has also been demonstrated to be efficacious in lowering 'emergence delirium (ED)' in comparison to the use of volatile inhalational anaesthetic agents[17] .

Multiple studies have investigated nonpharmacological techniques to reduce the incidence of 'emergence delirium (ED)' by alleviating preoperative anxiety have produced mixed outcomes and benefits. These interventions include presence of parents/caregivers during induction, tours of the operating theatre before surgery, and use of tablets or phone devices for playing animation videos/ activities during inhalational induction of anaesthesia [2,3].

'Emergence delirium (ED)' can lead to distress for patients and their caretakers both physically and emotionally. The impact of 'ED' may result in behavioural alterations that persist even after being discharged, including 'general anxiety, separation anxiety, sleep disturbances, bed-wetting, eating issues, aggression toward authority, and apathy or withdrawal'. Bodily harm may also happen to patients, parents, and healthcare staff. It is important to take necessary precautions to

protect intravenous lines, surgical drains & dressings. The presence of 'ED' can be disturbing for both parents & healthcare professionals, as well as other patients in the 'post-anaesthesia care unit (PACU)'. Additionally, 'ED' can increase healthcare bills due to prolonged PACU stays and the need for interventions such as surgical corrections & pharmacological ailments. Hence, good understanding of Emergence delirium and its management help improve clinical outcome.

OBJECTIVES

Primary Objective:

To compare effects of 0.3 mcg/kg and 0.5mcg/kg of dexmedetomidine in attenuation of emergence agitation in paediatric patient maintained on Sevoflurane, undergoing lower abdominal surgeries.

Secondary Objective:

Hemodynamic parameter assessment for 24 hours after administration of study drug

BASIC SCIENCES

Emergence agitation (EA) in children is a major postoperative issue that increases the risk of patient self-harm, places a burden on nursing staff, and reduces parent satisfaction with treatment. Risk factors for EA include age, preoperative anxiety, patient personality, pain, anesthesia method, and surgical procedure [1,2]. Sevoflurane and desflurane are widely used anesthetics due to their low blood/gas partition coefficients, but they have recently been posited as a cause of EA in children.

Because of the high incidence and untoward effects of emergence delirium in the paediatric population, investigating pharmacologic measures for preventing this phenomenon is important to the anaesthesia provider. Various drugs have been investigated to prevent EA in paediatric patients, including dexmedetomidine (DEX), midazolam, propofol, opioids, ketamine, and ketofol [5,11,13,14].

Dexmedetomidine is an alpha agonist having sedative, anxiolytic, hypnotic, analgesic, and sympatholytic properties. It produces these effects by inhibiting central sympathetic outflow by blocking the alpha receptors in the brainstem, thereby inhibiting the release of norepinephrine. It has a selectivity of 1600 to 1 for the alpha2 receptor as compared to alpha1. This selectivity is especially significant compared to another alpha agonist, clonidine, with a selectivity of 220 to 1. The mechanism by which dexmedetomidine may increase the duration of a peripheral nerve block is not fully understood but is believed to most likely be a perineural mechanism rather than a systemic or central mechanism, which appears to prolong the duration by blocking the cation current [15,18] .

Dexmedetomidine is rapidly distributed and is mainly hepatically metabolized into inactive metabolites by glucuronidation and hydroxylation. A high inter-

individual variability in dexmedetomidine pharmacokinetics has been described, especially in the intensive care unit population. In recent years, multiple pharmacokinetic non-compartmental analyses as well as population pharmacokinetic studies have been performed [19]. Body size, hepatic impairment, and presumably plasma albumin and cardiac output have a significant impact on dexmedetomidine pharmacokinetics. Results regarding other covariates remain inconclusive and warrant further research. Although initially approved for intravenous use for up to 24 h in the adult intensive care unit population only, applications of dexmedetomidine in clinical practice have been widened over the past few years. Procedural sedation with dexmedetomidine was additionally approved by the US Food and Drug Administration in 2003 and dexmedetomidine has appeared useful in multiple off-label applications such as paediatric sedation, intranasal or buccal administration, and use as an adjuvant to local analgesia techniques [20].

Pharmacokinetic studies have shown that body size and hepatic function have a significant influence on the pharmacokinetic profile of dexmedetomidine. Plasma albumin and cardiac output are suggested to have an impact on the apparent volume of distribution and clearance. Studies of the influence of other patient characteristics have produced inconclusive results.

Unlike sedative drugs such as propofol and the benzodiazepines, dexmedetomidine does not act at the gamma-aminobutyric acid (GABA) receptors. It induces sedation through activation of α_2-receptors in the locus coeruleus and induces a state mimicking natural sleep. Whilst sedated, respiration is minimally affected, and patients remain rousable. Side effects are mainly hemodynamic and include hypertension, hypotension, and bradycardia as a result of vasoconstriction, sympatholytic, and baroreflex-mediated parasympathetic activation.

Drug Formulations and Dosing Regimens

Dexmedetomidine, or 4-[(1S)-1-(2,3-dimethylphenyl) ethyl]-1H-imidazole, with molecular formula $C_{13}H_{16}N_2$, is the dextro-enantiomer of medetomidine, which is used as a sedative and analgesic in veterinary medicine. Dexmedetomidine is commercially available as a water-soluble HCl salt. Vials of Dexdor® and Precedex® contain a concentrate of dexmedetomidine hydrochloride, equivalent to 100 µg/mL dexmedetomidine. Prior to infusion, this is diluted to 4 or 8 µg/mL. Precedex is also available in pre-diluted solutions containing the required concentrations of 4 µg/mL in sodium chloride 0.9% . The Dexdor summary of product characteristics advises an initial infusion rate of 0.7 µg/kg/h without a loading dose, followed by titration to the desired effect using a dose range of 0.2–1.4 µg/kg/h. The Precedex label specifies a dosing regimen consisting of a 1-µg/kg loading dose in 10 min followed by a maintenance infusion of 0.2–0.7 µg/kg/h for ICU sedation. For procedural sedation, a loading dose of 1 µg/kg in 10 min followed by a maintenance infusion of 0.6 µg/kg/h, titrated to the desired clinical effect with doses ranging from 0.2 to 1 µg/kg/h, is recommended. Alternative dosing regimens can be considered in frail or elderly patients.

Pharmacokinetics

Absorption

Although dexmedetomidine is only registered for IV use, multiple routes of administration have been investigated. With extravascular administration, one can avoid the high peak plasma levels normally seen after IV administration. After oral administration, an extensive first-pass effect is observed, with a bioavailability of 16%. Dexmedetomidine is well absorbed through the intranasal and buccal mucosae,

a feature that could be of benefit when using dexmedetomidine in uncooperative children or geriatric patients [18,19,20] .

Distribution

Dexmedetomidine is a highly protein-bound drug. In plasma, 94% of dexmedetomidine is bound to albumin and α_1-glycoprotein. Pre-marketing studies with radioactively labelled dexmedetomidine, showed a rapid and wide distribution throughout the body. In pre-clinical animal studies, it was found that dexmedetomidine readily crosses the blood–brain and placenta barriers. Using non-compartmental analysis, a distribution half-life of about 6 min was found in healthy volunteers. The apparent volume of distribution was found to be related to body weight, with a volume of distribution at steady state in healthy volunteers of approximately 1.31–2.46 L/kg (90–194 L). In ICU patients, values are highly variable and mean volumes of distribution from 109 to 223 L have been reported . After long-term infusion in ICU patients with hypoalbuminemia, an increased volume of distribution at steady state was observed .

Metabolism and Elimination

Dexmedetomidine is eliminated mainly through biotransformation by the liver. A hepatic extraction ratio of 0.7 was found . Less than 1% is excreted unchanged with metabolites being excreted renally (95%) and fecally (4%) . Direct N-glucuronidation by uridine 5′-diphospho-glucuronosyltransferase (UGT2B10, UGT1A4) accounts for about 34% of dexmedetomidine metabolism. In addition, hydroxylation mediated by cytochrome P450 (CYP) enzymes (mainly CYP2A6) was demonstrated in human liver microsomes . In a pre-marketing ADME study by Abbott Laboratories, a single injection of 2 µg/kg radioactively labelled dexmedetomidine was given to healthy volunteers. The majority of the total plasma

radioactivity area under the curve consisted of dexmedetomidine (14.7%), the N-glucuronide isomers G-dex-1 (35%) and G-dex2 (6%), the O-glucuronide of hydroxylated N-methyl dexmedetomidine (H-1) (21%), and the imidazole oxidation product H-3 (10%) .These metabolites were 100-fold less potent in the α_2-receptor assay and therefore considered inactive. No relevant chiral inversion to the inactive Levo-enantiomer was found [21,22,23].

An elimination half-life of 2.1–3.1 h is reported in healthy volunteers. In ICU patients, similar values were found, with half-lives ranging from 2.2 to 3.7 h. non-compartmental analysis showed that dexmedetomidine clearance in healthy adult volunteers is approximately 0.6–0.7 L/min. Values range from 0.51 to 0.89 L/min, with the highest value of 0.89 L/min being found by Wolf et al. in volunteers with a relatively high body weight (mean 93.5 kg). In ICU patients,(mostly post-surgical) clearance is similar to the clearance found in healthy volunteers and ranges from 0.53-0.80 [20,24,25] .

For dexmedetomidine, prolonged as well as shortened elimination half-lives have been reported for patients with hypoalbuminemia. Clearance, however, is only marginally affected by hypoalbuminemia . This is in line with the “well-stirred” liver model, which states that for compounds with a high extraction ratio, liver blood flow is the most important factor governing hepatic clearance and changes in plasma protein levels are expected not to result in increased drug clearance. The impact on dexmedetomidine clearance as a result of changes in liver blood flow, via changes in cardiac output, was studied by Dutta et al., .They describe an estimated reduction in cardiac output of 19% associated with a reduced clearance of 12% at plasma dexmedetomidine levels of 1.2 ng/mL .

Dose Proportionality and Inter-Individual Variability

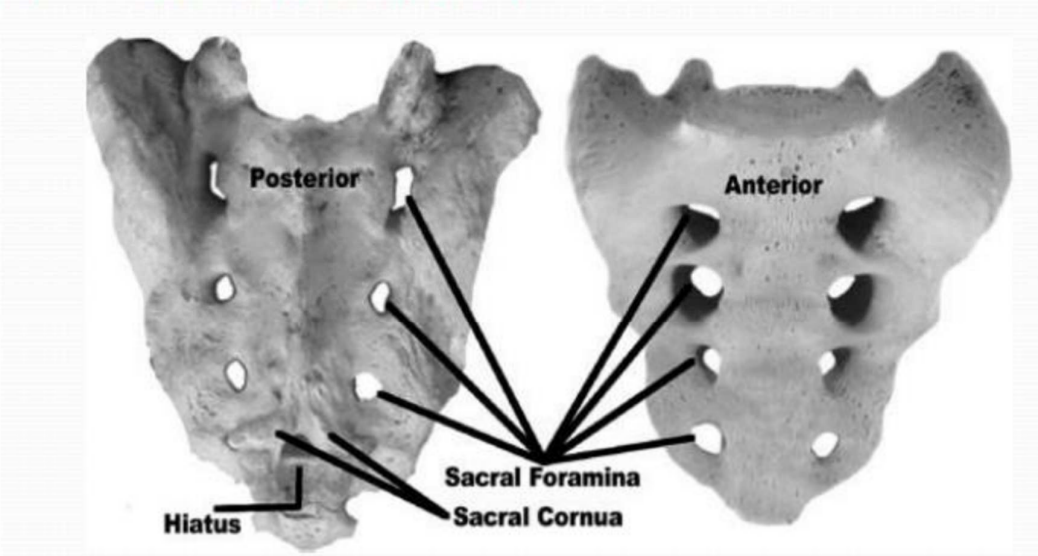
Within the therapeutic range, dose proportionality has been shown for dexmedetomidine. No relevant time dependency has been reported. Nevertheless, a high inter-individual variability is observed for clearance and distribution volumes. Hypoalbuminemia, end-organ damage, changes in hemodynamic, and decreased cardiac output may all contribute to a high inter-individual variability, especially in the ICU population.

Drug pharmacokinetics might be affected by ethnicity, especially when a drug is highly protein bound or undergoes hepatic metabolism . A few small studies evaluated the role of race in dexmedetomidine pharmacokinetics/pharmacodynamics, but no clinically relevant influence was observed . Furthermore, Kohli et al. genotyped 40 subjects for five common CYP2A6 alleles and grouped them into normal (n = 33), intermediate (n = 5), and slow (n = 2) metabolizers. Although their study population was small and effects could have been obscured by the complex clinical situation, they found no significant influence of these genotypes on dexmedetomidine disposition in ICU patients . Multiple other studies have evaluated the role of α-2A, -2B, and -2C adrenoceptor polymorphisms, but no recommendations to guide clinical dosing regimens have yet been derived .

CAUDAL ANAESTHESIA

Caudal anaesthesia was described at the turn of last century by two French physicians, Fernand Cathelin and Jean-Anthanase Sicard. The technique pre-dated the lumbar approach to epidural nerve block by several years. Caudal anaesthesia, however, did not gain in popularity immediately following its inception. One of the major reasons caudal anaesthesia was not embraced is the wide anatomical variations

of sacral bones and the consequent failure rate associated with attempts to locate the sacral hiatus. The failure rate of 5% to 10% made caudal epidural anaesthesia unpopular until a resurgence of interest in the 1940s, led by Hingson and colleagues, who used it in obstetrical anaesthesia. Caudal epidural anaesthesia has many applications, including surgical anaesthesia in children and adults, as well as the management of acute and chronic pain conditions. Success rates of 98%–100% can be achieved in infants and young children before the age of puberty, as well as in lean adults.



Anatomy of sacral hiatus

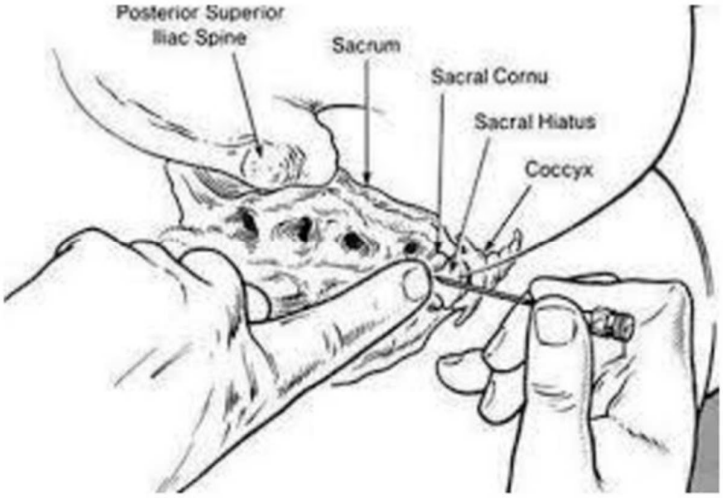
The sacrum is a large triangularly shaped bone formed by the fusion of the five sacral vertebrae. It has a blunted, caudal apex that articulates with the coccyx. Its superior, wide base articulates with the fifth lumbar vertebra at the lumbosacral angle. Its dorsal surface is convex and has a raised interrupted median crest with four (sometimes three) spinous tubercles representing fused sacral spines. Flanking the median crest, the posterior surface is formed by fused laminae. Lateral to the median

crest, four pairs of dorsal foramina lead into the sacral canal through intervertebral foramina, each of which transmits the dorsal ramus of a sacral spinal nerve . Below the fourth (or third) spinous tubercle, an arched sacral hiatus is identified in the posterior wall of the sacral canal due to the failure of the fifth pair of laminae to meet, exposing the dorsal surface of the fifth sacral vertebral body. The caudal opening of the canal is the sacral hiatus roofed by the firm elastic membrane, the sacrococcygeal ligament, which is an extension of the ligamentum flavum. The fifth inferior articular processes project caudally and flank the sacral hiatus as sacral cornua, connected to the coccygeal cornua by intercornual ligaments [26].

The sacral canal is triangular in shape. It is a continuation of the lumbar spinal canal. Each lateral wall presents four intervertebral foramina, through which the canal is in contiguous with the pelvic and dorsal sacral foramina. The posterior sacral foramina are smaller than their anterior counterparts. The sacral canal contains the cauda equina (including the filum terminale) and the spinal meninges. Near its midlevel (typically the middle one-third of S2 but varying from the midpoint of S1 to the midpoint of S3), the subarachnoid and subdural spaces cease to exist, and the lower sacral spinal roots and filum terminale pierce the arachnoid and dura mater. However, variations in the termination of the dural sac as well as pathologic conditions like sacral meningocele or sacral perineural cysts can increase the chances of inadvertent dural puncture when performing caudal nerve block in such patients with abnormal anatomy [26,27].

The lowest margin of the filum terminale emerges at the sacral hiatus and traverses the dorsal surface of the fifth sacral vertebra and sacrococcygeal joint to reach the coccyx. The fifth sacral nerve roots also emerge through the hiatus medial to each of the sacral cornua. The sacral canal contains the epidural venous plexus, which

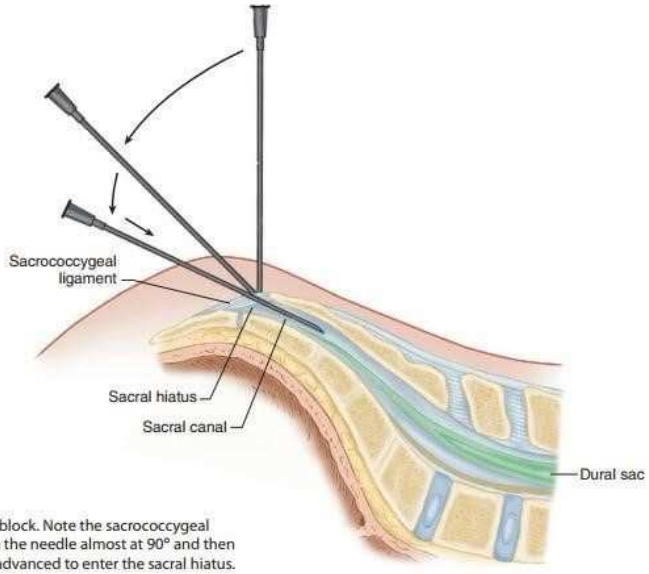
generally terminates at S4, but which may continue more caudally. Most of these vessels are concentrated in the anterolateral portion of the canal. The remainder of the sacral canal is filled with adipose tissue, which is subject to an age-related decrease in its density. This change may be responsible for the transition from the predictable spread of local anaesthetics administered for caudal anaesthesia in children to the limited and unpredictable segmental spread seen in adults.



The sacral foramina afford anatomical passages that permit the spread of injected solutions, such as local anaesthetics and adjuvants. The posterior sacral foramina are essentially sealed by the multifidus and Sacro spinalis muscles, but the anterior foramina are unobstructed by muscles and ligaments, permitting ready egress of solutions through them. The sacral curvature varies substantially. In a cadaver study, looking at the anatomy of caudal epidural space, the sacral cornua were not palpable bilaterally in 14.3% and palpable unilaterally in 24.5% of specimens. The level of maximum curvature of the sacrum was at S3 in 69.4% of cases. This variability tends to be more severe in males than in females.

In paediatric inguinal surgeries, caudal epidural blocks have been shown to provide postoperative analgesia comparable in duration to ultrasound-guided ilioinguinal and iliohypogastric blocks with dexmedetomidine as an additive without significant adverse effects [28].

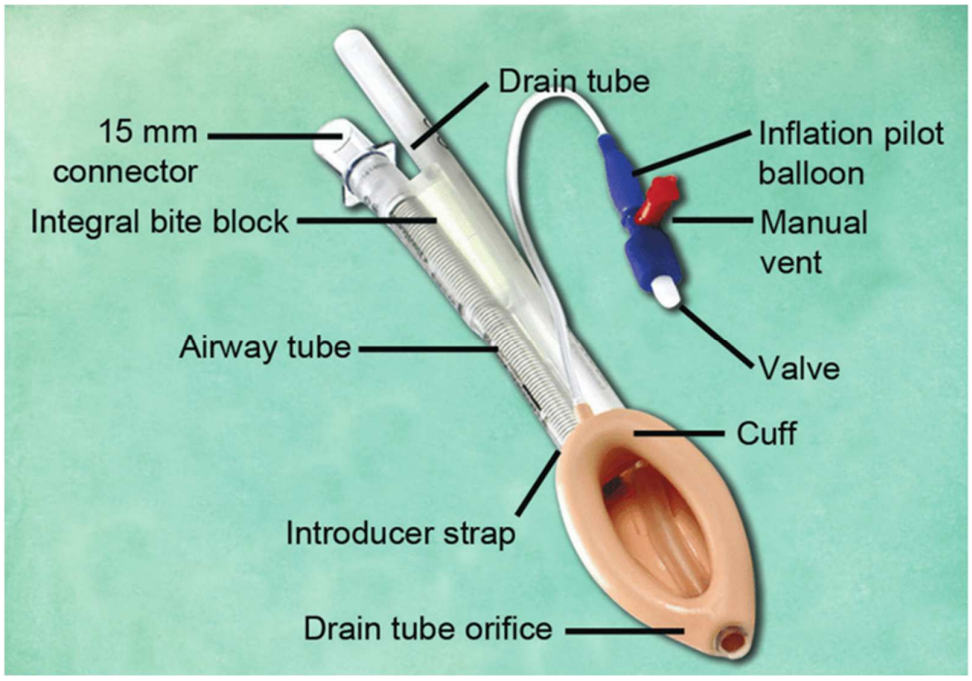
Positioning an anesthetized child for caudal block and palpation for the sacral hiatus. An assistant gently helps flex the spine.



Caudal block. Note the sacrococcygeal ligament is penetrated with the needle almost at 90° and then must be angled down and advanced to enter the sacral hiatus.

PROSEAL LMA:

The ProSeal (PLMA) was introduced by Dr. Archie Brain in 2000 [29]. PLMA is a safe and effective alternative to ET for paediatric airway management, with fewer complications. It is less irritating to the airway compared to traditional methods like endotracheal intubation with less laryngeal stimulation, leading to fewer postoperative complications, such as coughing and sore throat. The incidence of aspiration or regurgitation during induction, the intraoperative period, or after the airway device was removed has proven to be lesser. Due to reduced mucosal pressure and less stimulation to the pharyngeal mucosa, as well as a decrease in postoperative nausea and vomiting (PONV), its use is a success in managing paediatric airway be it shorter or longer duration surgeries [30].



ProSeal LMA has a gastric drainage tube, placed lateral to the main airway tube. The gastric drainage tube forms a channel for regurgitated gastric contents [31]and prevents gastric insufflation and pulmonary aspiration [32].A gastric tube can be placed through a drain tube and can detect the malposition [31] of PLMA. The paediatric PLMA lacks the dorsal cuff [31]. The paediatric ProSeal LMA available sizes are 1, 1.5, 2 and 2.5 [32]. Considering its success rates, hemodynamic stability, and fewer perioperative complications, it was the choice of airway device in this study.

Laryngeal Mask Size	Patient Weight (kg)	Max Cuff Volume (mL)
1	< 5	4
1.5	5-10	7
2	10-20	10
2.5	20-30	14
3	30-50	20
4	50-70	30
5	70-100	40

REVIEW OF LITERATURE

"Aouad MT et al.", in their study on emergence agitation summarised that it is a common and distressing occurrence in paediatric patients following general anaesthesia. The study highlights that younger child, particularly those under the age of 5, have a higher chance for developing EA. It also identifies factors such as the 'type of surgery (e.g., ear, nose, and throat procedures)', the use of certain anaesthetic agents (like sevoflurane), and rapid emergence from anaesthesia as contributing factors to EA. Pharmacological interventions, including the use of sedatives like clonidine and dexmedetomidine, are proven to reduce the occurrence of EA. . Non-pharmacological approaches, such as providing a pleasant and silent recovery environment, were also recommended as preventive measures. The article focusses on the importance of identifying high-risk patients and applying appropriate strategies to manage and prevent EA. Further research is needed to refine these strategies and improve patient care [33].

The study conducted by "Welborn et al. (1996)", compares the emergence and recovery profiles of three anaesthetic agents—'sevoflurane, desflurane, and halothane'—in paediatric ambulatory patients. The key results were that Sevoflurane and desflurane were found to provide faster emergence times compared to halothane, meaning children woke up more quickly from anaesthesia with these agents. Recovery times were also shorter for sevoflurane and desflurane. This included the time to attain an 'Aldrete score' of 9 (a score used to assess recovery after anaesthesia) and discharge readiness. Sevoflurane and desflurane showed less postoperative agitation compared to halothane, which could lead to a more comfortable recovery for children. The study concluded that both sevoflurane and desflurane provide faster emergence and recovery compared to halothane, with less

postoperative agitation, making them more favourable choices for paediatric ambulatory anaesthesia [4].

In a study that provides an in-depth review of the pharmacokinetics of sevoflurane, a commonly used inhalational anaesthetic, insights on how sevoflurane is absorbed, distributed, metabolized, and eliminated in the body is explained. It is rapidly absorbed into the bloodstream after inhalation, with high solubility in the blood. It quickly reaches the brain and other tissues, leading to fast induction of anaesthesia . It is minimally metabolized in the liver. Only a small percentage (around 2-5%) undergoes biotransformation, mainly to inorganic fluoride and other metabolites. Primarily eliminated through the lungs as unchanged gas, its relatively low hepatic metabolism and fast elimination rate contribute to a rapid recovery from anaesthesia. The drug has a short elimination half-life, which supports its quick recovery profile compared to other anaesthetics. The article concludes that sevoflurane has favourable pharmacokinetic properties, including rapid induction and emergence, minimal metabolism, and efficient elimination, which make it an ideal choice for general anaesthesia, especially in ambulatory and paediatric settings [34].

"Lerman et al. (1996)" evaluated the usage of 'sevoflurane' in paediatric subjects posted for day case surgery, comparing it with halothane. Results showed that sevoflurane was found to provide faster and smoother induction of anaesthesia in children compared to halothane. It led to a quicker recovery time post-surgery, with children awakening faster and experiencing fewer side effects such as nausea or vomiting. The safety profile of both sevoflurane and halothane was comparable , with no evident adverse effects reported during the study. Overall, the study suggests that sevoflurane can be a favourable anaesthetic agent for use in paediatric ambulatory

surgery due to its quick induction, smooth recovery, and safe profile compared to halothane [2].

"Picard et al. (2000")" compared the recovery quality in paediatric patients following anaesthesia with sevoflurane and propofol. The study evaluated how well children recovered after anaesthesia with either sevoflurane or propofol, focusing on factors like emergence time, post-operative behaviour, and complications. Children who received sevoflurane exhibited quicker emergence from anaesthesia and experienced fewer postoperative side effects, such as agitation or nausea. Propofol, while also effective, was associated with longer recovery times and a higher occurrence of side effects like nausea and vomiting compared to sevoflurane. It concluded that sevoflurane offers better recovery quality in children undergoing surgery compared to propofol, making it a preferable choice for paediatric anaesthesia [35].

"Dahmani et al. (2010)", in their study of various literature examined pharmacological interventions to decrease incidence of 'emergence agitation (EA)' in children induced by 'sevoflurane' and 'desflurane' anaesthesia. The study focuses on the common post-anaesthesia complication of emergence agitation, where children may experience confusion, restlessness, and aggressive behaviour as they wake up from anaesthesia. The authors analyse various drugs used to prevent emergence agitation in younger individuals going for surgery under either sevoflurane or desflurane. The meta-analysis examines the effectiveness of several pharmacological agents, including 'clonidine, fentanyl, midazolam & dexmedetomidine', in lowering the incidence of 'emergence agitation'. The findings indicate that certain drugs, such as 'clonidine' and 'dexmedetomidine', were potent in decreasing the occurrence of 'emergence agitation' following 'sevoflurane' and desflurane anaesthesia. To

summarize, the study suggests that pharmacological strategies, especially the use of specific agents like 'dexmedetomidine' and 'clonidine', can significantly reduce the risk of 'emergence agitation' in children undergoing surgery with sevoflurane or desflurane [36].

A meta-analysis by "Amorim et al." including RCTs that included paediatric patients undergoing elective procedures under general anaesthesia with sevoflurane found that dexmedetomidine helped to reduce the incidence of sevoflurane-induced ED compared to placebo [37].

Another comprehensive review threw light upon the 'pharmacokinetics and pharmacodynamics of dexmedetomidine', used primarily for sedation and analgesia. The article discusses how dexmedetomidine is absorbed, distributed, metabolized, and eliminated in the body. It has a quick onset of action and a short half-life, making it well-suited for short-term sedation. The drug undergoes extensive hepatic metabolism through the 'cytochrome P450 system' and is excreted mainly through urine. Dexmedetomidine's primary action is through its α2-adrenergic receptor agonism, which leads to sedation, analgesia, and sympatholysis. The article outlines its dose-dependent effects on sedation, analgesia, and the preservation of respiratory function compared to other sedatives. The pharmacodynamic properties of dexmedetomidine make it a useful sedative in 'intensive care units (ICU)', during surgery, and for sedation involving procedures. The drug provides a sedative effect that is distinct from other sedatives like benzodiazepines or propofol, with the added benefit of less respiratory depression. While generally well-tolerated, the article mentions that dexmedetomidine can cause hypotension, bradycardia, and, rarely, arrhythmias. These effects are usually dose-dependent and transient. The review highlights its use in both

adult and paediatric patients, including its role in post-operative recovery, as a supplement to general anaesthesia, and for long-term sedation in ICU settings [15].

The article by "Tobias (2007)" reviews different roles of dexmedetomidine in paediatric population, focusing on its role in both critical care and anaesthesiology. The author emphasizes the benefits of dexmedetomidine in the "paediatric intensive care unit (PICU)", where it is shown to be used for sedation in "Mechanically ventilated children", minimizing the need for conventional sedatives like benzodiazepines. 'Dexmedetomidine' helps reduce the occurrence of agitation and delirium often seen with other sedatives. In anaesthesiology, 'dexmedetomidine' is discussed as an adjunct to general anaesthesia, providing sedation and reducing the requirement for other anaesthetics. It is particularly beneficial in maintaining stable haemodynamic during surgery, as it causes minimal cardiovascular instability compared to other sedative agents. The article highlights that while dexmedetomidine is generally well-tolerated in children, it may cause side effects like bradycardia and hypotension, which are usually dose-dependent and reversible. Careful monitoring is required to manage these effects, particularly in critically ill or unstable patients. The study notes the growing body of evidence advocating for the application of dexmedetomidine across different paediatric settings, including preoperative sedation, intraoperative adjuncts, and postoperative recovery. It is also used for managing pain and anxiety in children without significant respiratory depression [38].

Multiple studies administered dexmedetomidine intranasally. It is recommended to administer dexmedetomidine intranasally as a preoperative anxiolytic because it is non-invasive, facilitates parental separation, helps reduce the anxiety of paediatric patients in the operating room and ensures a smooth induction of inhalation anaesthesia [39].

Comparison of the effectiveness of “intranasal dexmedetomidine” and “oral midazolam” as premedication agents in ‘paediatric patients posted for surgery’ was done by “Kumar L et al.” The objective of the study was to evaluate and comparatively assess the ‘sedative, anxiolytic, and hemodynamic effects of intranasal dexmedetomidine with oral midazolam’ as premedication in children. The study involved randomly assigning paediatric patients to receive either “intranasal dexmedetomidine (1 µg/kg) or oral midazolam (0.5 mg/kg)” before surgery. The researchers assessed the onset of sedation, the quality of sedation, the anxiolytic effect, and the side effects of both drugs. Results showed intranasal dexmedetomidine provided faster onset and more profound sedation compared to oral midazolam. It also demonstrated better ‘anxiolytic’ effect. ‘Dexmedetomidine’ was associated with stable haemodynamics, with fewer instances of tachycardia or hypertension compared to midazolam, which sometimes caused slight changes in ‘heart rate(HR)’ and “blood pressure”. Both agents were well accepted, with minimal side effects. However, ‘dexmedetomidine’ had fewer occurrences of paradoxical reactions, such as agitation, which were seen more frequently with ‘midazolam’. The study deciphered that intranasal ‘dexmedetomidine’ is a more effective and safer option than oral midazolam for paediatric premedication. It provides better sedation and anxiolysis with fewer side effects, particularly in terms of hemodynamic stability and paradoxical reactions. In summary, the article supports the administration of “intranasal dexmedetomidine” as a superior alternative to “oral midazolam” for preoperative sedation in children, offering faster onset, better sedation quality, and minimal side effects [40].

"Prabhu MK and Mehandale SG", in their study, focussed on evaluating and comparing the effectiveness of "oral dexmedetomidine" and "oral midazolam" as premedication to prevent "emergence agitation (EA)" after "sevoflurane anaesthesia" in children. Their study aimed to compare the effectiveness of two sedative drugs, 'dexmedetomidine' and 'midazolam', in reducing the occurrence of emergence agitation following sevoflurane anaesthesia in paediatric patients. The research included paediatric patients undergoing elective surgeries who were given either oral 'dexmedetomidine' or 'oral midazolam' as pre-medication. The incidence of 'emergence agitation' was then observed and recorded. The study found that 'dexmedetomidine' was more advantageous than 'midazolam' in preventing 'emergence agitation'. Patients who received 'dexmedetomidine' exhibited a lesser incidence of EA, a smoother recovery profile, and less post-operative restlessness compared to those given 'midazolam'. The authors came to a conclusion that 'oral dexmedetomidine' is a better choice than 'oral midazolam' as a pre-medication for preventing "emergence agitation in paediatric patients undergoing sevoflurane anaesthesia". This study highlights the importance of selecting appropriate premedication in paediatric anaesthesia to enhance recovery and reduce post-operative agitation, which is common among children waking up from anaesthesia[41].

In a study where the effects of a singular dose of 'dexmedetomidine', given intravenously, on 'postop agitation' and extubation quality in children posted for adenotonsillectomy were studied & the findings revealed that 'dexmedetomidine' significantly decreased the incidence of 'emergence agitation' in the recovery room and provided smoother extubation, meaning that subjects had less difficulty waking up from anaesthesia. This improvement was associated with better post-operative recovery outcomes. The authors concluded that a single dose of dexmedetomidine is

effective in reducing agitation and promoting smooth extubation in children undergoing adenotonsillectomy, thus improving the overall anaesthetic recovery experience. They agreed that the drug particularly helped in reducing agitation and facilitating easier extubation after surgery[42].

A study by "Huang et al." compared the infusion of propofol (2 mg/kg/h) with dexmedetomidine (0.5 µg/kg/h) and found that dexmedetomidine was more effective than propofol in reducing the incidence of emergence delirium (ED). A meta-analysis examining various dosages and administration methods (both bolus and infusion) of dexmedetomidine revealed its significant effectiveness in lowering the occurrence of ED. The optimal dose of dexmedetomidine was determined to be 0.30 µg/kg (95% confidence interval: 0.21–1 µg/kg). The combined results from this meta-analysis demonstrated that dexmedetomidine was more effective than both placebo and midazolam in preventing ED [43].

Dexmedetomidine administered intravenously (whether as a bolus, continuous infusion, or both) has been shown to reduce emergence agitation (EA) following the use of volatile anaesthetics. Infusions of dexmedetomidine seem to be more effective than narcotics [44].

A prospective, randomized trial found that dexmedetomidine (2 µg/kg intravenous bolus followed by 0.7 µg/kg/h infusion) was more effective than a single dose of fentanyl (1 µg/kg intravenous) in reducing EA in children with obstructive sleep apnoea undergoing tonsillectomy or adenoidectomy with sevoflurane anaesthesia [45].

In a study by "Sharma K et al.", an intravenous infusion of dexmedetomidine (1 µg/kg) was administered over ten minutes prior to anaesthesia induction to prevent emergence delirium (ED). The PAED score demonstrated a significantly reduced

incidence of ED (P < 0.001). Intraoperative dexmedetomidine has also been used as a preventive measure in two recent studies [46].

In a similar study by Shi M et al., 0.5 µg/kg of dexmedetomidine was administered intravenously over ten minutes after anaesthesia induction, resulting in a significantly lower incidence of ED compared to the saline control (31.1% vs 53.3%; P = 0.033) [47].

In another study by "Li H et al.", a continuous intravenous infusion of 0.2 µg/kg/hour was administered after anaesthesia induction and continued until the end of surgery. This approach significantly reduced the incidence of emergence delirium (ED), as measured by the OPS (15.0% vs 82.5%; p <0.001) [48].

The results of a study by "Jeongmin Kim et al." suggest that continuous intraoperative infusion of low-dose dexmedetomidine (0.2 µg/kg/h) can effectively reduce the incidence of emergence agitation (EA) following desflurane anesthesia in pediatric patients undergoing strabismus surgery. Additionally, the use of low-dose dexmedetomidine was found to decrease postoperative pain without causing any hemodynamic instability or delaying emergence [6].

In paediatrics, nausea and vomiting are major causes of postoperative discomfort. PONV tends to be more common in general anaesthesia than in spinal anaesthesia. Moreover, PONV in paediatric anaesthesia is associated with other risk factors, including surgery lasting >30 min, age ≥3 years, previous PONV, positive family history and strabismus surgery . In addition, it can lead to electrolyte imbalance and extend the patient's stay in the recovery room. Several studies have recently focused on the effect of dexmedetomidine on PONV, finding that it has an antiemetic effect and may reduce PONV incidence [49].

In a study comparing the "ProSeal Laryngeal Mask Airway (PLMA)" with traditional Endotracheal Intubation (ET) for airway management in paediatric patients undergoing elective surgery under general anaesthesia, ninety children (aged 2-10) were randomly assigned to either the PLMA (Group P) or Endotracheal Intubation (Group E) group. The study assessed success rates, hemodynamic stability, and perioperative complications. The results indicated that PLMA is a safe and effective alternative to ET for paediatric airway management, with fewer complications. The study highlighted that supraglottic airway devices, such as the PLMA, are less irritating to the airway compared to traditional methods like endotracheal intubation. This leads to reduced laryngeal stimulation and fewer postoperative complications, including coughing and sore throat. Importantly, there were no instances of aspiration or regurgitation during induction, intraoperative procedures, or after device removal in either group. The reduced incidence of coughing and sore throat in the PLMA group was likely due to lower mucosal pressure and less stimulation of the pharyngeal mucosa, contributing to a decrease in postoperative nausea and vomiting (PONV) [31].

In another comparative study, both the" ProSeal Laryngeal Mask Airway (PLMA) and Endotracheal Intubation (ET)" demonstrated high success rates in airway management, with first-attempt success rates of 95.56% for PLMA and 97.78% for ET. However, the PLMA group experienced fewer postoperative complications, such as coughing, sore throat, and vomiting, which was likely due to reduced mucosal pressure and less irritation of the pharyngeal mucosa. There were no instances of aspiration or regurgitation during anaesthesia induction, the intraoperative period, or after the removal of the 'PLMA' in either group. PLMA also caused less laryngeal stimulation, making it a less invasive and safer option with a lower risk of postoperative complications compared to ET tube [33].

Postoperative pain is considered a significant contributing factor to ED, which is also associated with anaesthetic agents such as sevoflurane since many patients experience emergence agitation during recovery, mimicking ED [50].

In the study comparing "three equal groups of paediatric patients receiving either nalbuphine 0.1 mg/kg IV (Group N), dexmedetomidine 0.5 µg/kg IV (Group D), or saline solution (Group C) 10 minutes before the end of surgery", all children had been administered caudal block for pain management. Despite this, the "incidence of emergence agitation (EA)" in the placebo group (Group C) continued to be relatively higher at 40%. This suggests that simply providing pain relief through regional anaesthesia, such as a caudal block, is not sufficient on its own to prevent EA after sevoflurane anaesthesia in paediatric patients. Additional interventions, like "dexmedetomidine / nalbuphine ", may be necessary to address other factors contributing to EA [51].

MATERIALS AND METHODS

Source of Data:

Study subjects aged two to ten years, belonging to 'ASA grades I and II', 'undergoing lower abdominal surgeries under general anesthesia along with caudal epidural anaesthesia' at 'KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belagavi – 590010'.

"Study Design": one year randomized controlled trial

"Study Period": 'One year'

Sample Size:

Sample size at 95% CI, 80% power

"$n= \frac{(Z1-\alpha/2 - Z1-\beta)\ (p1q1+p2q2)}{(p1-p2)^2}$ "

n= 25

Though the minimum sample size obtained from the formula is 25, for the purpose of better and precise results**, a sample size of 30** will be taken in each group in this study.

where p1= 48%, p2=14% q1=52%, q2=86%

p in the above formula stands for the prevalence of independent variable of emergence agitation.

Z1-α/2 is linked with the level of significance and Z1-β is linked with the power of the test. For 5% level of the significance Z1-α/2 = 1.96 and Z1-β = 0.84 for 80% power of the test.

Sampling technique:

A one-year randomized control trial. Randomization will be achieved by computer generated randomization charts.

Inclusion Criteria:

- ASA physical status I and II.
- Age between 2 to 10 years.
- Patients undergoing lower abdominal surgeries
- Surgery time less than or equal to 2 hours.

Exclusion Criteria:

- Risk of aspiration
- Predicted difficult airway
- Patients with Upper respiratory tract infections
- Congenital malformations involving respiratory tract
- History of epilepsy
- Patients with cardiac, renal or liver diseases
- Patients with allergy to the study drug or local anaesthetic
- Cervical spine disease
- On digoxin use

MEHODOLOGY:

This prospective double blinded randomized trial was started after obtaining clearance from ethical committee of the institution. Study was also registered under Clinical Trials registry of India (CTRI/2024/04/066425).

The study included paediatric patients aged 2 to 10 years of either gender, with ASA physical status I or II, undergoing elective lower abdominal surgeries with an operation time of 2 hours or less. Exclusion criteria involved patients with risks of aspiration, known difficult airways, respiratory infections, congenital respiratory tract malformations, epilepsy, or preexisting cardiac, renal, or liver diseases. Additionally, children with allergies to the study drugs, cervical spine disease, bleeding or coagulation disorders, allergy to local anaesthetics, those receiving digoxin and most importantly those who were preoperatively agitated were excluded from the trial. Written informed consent was obtained from at least one parent or guardian for all participants.

The day before surgery, each patient underwent a standard pre-operative evaluation in accordance with the hospital protocol. Patients are kept nil-per-oral for 6 hours for solids, 4 hours for semisolids, and 2 hours for clear fluids.

In the preoperative room, a 22-gauge/ 20-guage peripheral venous access was established (as per age of the child and convenience) and the child was premedicated with Inj. Glycopyrrolate (0.005 mg/kg) IV followed by Inj. Ketamine (1mg/kg) IV.

On shifting to the operating room, children were continuously monitored using pulse oximetry, non-invasive blood pressure, electrocardiogram, capnography, temperature probes, and inspiratory and expiratory gas concentrations throughout the surgery. All patients received a standardized anaesthetic technique beginning with 100% oxygen at 6 L/min . General anaesthesia was induced with sevoflurane. Once a

Ramsay sedation score of 5 or 6 was achieved, patients were premedicated with Inj. Fentanyl (1-2 mcg/kg IV), followed by induction with Inj. Ketamine (1 mg/kg IV).

A dose of 1-2 mg/kg of intravenous succinylcholine was administered. After adequate anaesthesia and muscle relaxation were achieved, a properly sized Proseal laryngeal mask airway (PLMA), apt for the child's age and weight, was lubricated with water-soluble jelly and inserted post sufficient jaw relaxation. Proper placement of PLMA was confirmed with adequate bilateral chest movements on ventilation, bilateral air entry on auscultation and a square shaped canpnogram on the monitor. Patient was maintained on inhalational oxygen, nitrous oxide and Sevoflurane at 2% dial concentration.

As maintenance with inhalational Sevoflurane continued, patients were put in lateral position and 1.0 ml/kg of 0.25% bupivacaine was administered into the caudal epidural space using a 22-gauge needle in all children after negative aspiration for blood and CSF. After successful administration of the drug, patients were returned to supine position. Adequate caudal block was identified by a lack of increase in mean arterial pressure (MAP) and heart rate exceeding 10% above the pre-incisional baseline at the start of surgery. No propofol or narcotics were administered during the procedure.

Throughout the surgery, child was ventilated via closed circle system. Adequate lung ventilation was controlled and maintained using Pressure support ventilation. The end tidal carbon dioxide tension was maintained within 32–38 mmHg. Fluid administration was strictly as per Holliday Segar Formula, the 4-2-1 rule.

Haemodynamic parameters were monitored throughout. 10 mins before the end of surgery, **Group D 0.3**: received 0.3 mcg/ kg of Dexmedetomidine IV and **Group D 0.5**: received 0.5 mcg/kg of Dexmedetomidine IV, slowly, as an infusion over 10 minutes.

At the end of the surgery, Proseal LMA was removed in a deep plane of anaesthesia. 100% oxygen was continued after sevoflurane was discontinued. Once the patient demonstrated a regular respiratory pattern, he or she was shifted to post operative recovery in the left lateral position.

Haemodynamic parameters such as blood pressure, heart rate, respiratory rate, oxygen saturation and end tidal CO2 were monitored and assessed- at the time of drug administration, after removal of LMA, and every 10 minutes in the first hour followed by every half hourly for the next 24 hours.

After shifting to PACU, incidence and severity of emergence agitation (EA) was assessed using the Paediatric Anaesthesia Emergence Delirium (PAED) scale, Watcha scale and Cravero scale (Table A,B and C)[12], all of which included psychomotor components describing the child's behaviour in the recovery room. Scores and haemodynamic parameters such as MAP, HR, RR and blood oxygen saturation were recorded at 10, 20, 30, 40, 50 and 60 minutes post-operatively. Patients with a PAED score of ≥12 were classified as having emergence delirium (ED), with severe ED defined as a score of ≥15. Assessment of pain was also done using Visual analogue scale and Faces Pain scale.

Table A: PAED Score

Criteria	Not at All	Just a Little	Quite a Bit	Very Much	Extremely	Score
The child makes eye contact with the caregiver/parent	4	3	2	1	0	
The child's actions are purposeful	4	3	2	1	0	
The child is aware of his/her surrounding	4	3	2	1	0	
The child is restless	0	1	2	3	4	
The child is inconsolable	0	1	2	3	4	
Total score						

Table B: Watcha Scale

Behaviour	Score
Asleep	0
Calm	1
Crying, but can be consoled	2
Crying, but cannot be consoled	3
Agitated and thrashing around	4

Table C: Cravero Scale

Behaviour	Score
Obtunded with no response to stimulation	1
Asleep but responsive to movement or stimulation	2
Awake and responsive	3
Crying (for >3 min)	4
Thrashing behaviour that requires restraint	5

Rescue medication used in a situation of PAEDS score of >/= 15 , or a pain score of more than 4 was Inj. Fentanyl at 1 mcg/kg body weight.

STATITICAL ANALYSIS:

METHODS:

Data is analysed using statistical software R version 4.4.1. and Microsoft Excel. Categorical variables given in the form of frequency tables. Continuous variables given in Mean ± SD / Median (Min, Max) form. Chi square test is used to check the association of categorical variables. Normality of variable is checked by Shapiro Wilk test and QQ plot. If data follows normal distribution, parametric tests will be used. Otherwise, non-parametric tests will be used. Two sample t test is used to compare mean of variables over groups. Mann Whitney U test is used to compare the distribution of variables over groups. Friedman test is used to compare the distribution of variables over time. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS

The data consists of measurements from sixty subjects, evenly divided into "two groups: Group D 0.3 and Group D 0.5", with thirty subjects in each group. The following table gives the comparison of demographic variables over groups.

"Table 1: Comparison of demographic variables over groups"

Variables	Subcategory	Group D 0.3	Group D 0.5	p-value
Age (years)	Mean ± SD	15.97 ± 8.8	15.97 ± 8.8	1^t
	Median (Min, Max)	16.15 (1.3, 30.4)	16.15 (1.3, 30.4)	
Gender	Female	8 (26.67%)	8 (26.67%)	1^C
	Male	22 (73.33%)	22 (73.33%)	

"Abbreviation: t – Two sample t test, C – Chi square test"

The mean age of participants in both groups was identical at 15.97 ± 8.8 years, with a median age of 16.15 years (range: 1.3 to 30.4 years). From two-sample t-test, it is observed that there is no statistically significant difference in age between the groups. Regarding gender distribution, both groups had the same proportion of females (26.67%) and males (73.33%). "From Chi-square test, it is observed that there is no significant difference in gender distribution between the two groups".

"The following table gives the comparison of PAEDS Score, WATCHA Scale and CRAVERO Scale over time and group.

Table 2: Comparison of PAEDS Score, WATCHA Scale and CRAVERO Scale over time and group.

Variables	**Time points**	**Sub Category**	**Group D 0.3**	**Group D 0.5**	**p-value**
PAEDS Score	10 mins	Mean ± SD Median (Min, Max)	11.23 ± 2.03 11 (6, 14)	10.6 ± 1.61 10 (6, 14)	0.2125[MW]
	20 mins	Mean ± SD Median (Min, Max)	10.73 ± 1.74 11 (6, 13)	10.27 ± 1.44 10 (6, 13)	0.2760[MW]
	30 mins	Mean ± SD Median (Min, Max)	10.53 ± 1.72 10 (6, 13)	10 ± 1.34 10 (6, 13)	0.2331[MW]
	40 mins	Mean ± SD Median (Min, Max)	9.93 ± 1.55 10 (6, 12)	9.5 ± 1.25 9 (6, 12)	0.2447[MW]
	50 mins	Mean ± SD Median (Min, Max)	9.6 ± 1.4 9 (7, 12)	9.17 ± 1.23 9 (6, 12)	0.4818[MW]
	60 mins	Mean ± SD Median (Min, Max)	9.47 ± 1.7 9 (6, 12)	8.93 ± 1.28 9 (6, 12)	0.2510[MW]
p-value			**< 0.001[F]***	**< 0.001[F]***	-
WATCHA Scale	10 mins	Mean ± SD Median (Min, Max)	1.5 ± 1.07 1 (0, 3)	1.03 ± 0.93 1 (0, 3)	0.0919[MW]
	20 mins	Mean ± SD Median (Min, Max)	1.43 ± 1.07 1 (0, 3)	0.97 ± 0.89 1 (0, 3)	0.0910[MW]
	30 mins	Mean ± SD Median (Min, Max)	1.2 ± 0.76 1 (0, 2)	0.93 ± 0.74 1 (0, 2)	0.1683[MW]
	40 mins	Mean ± SD Median (Min, Max)	1.17 ± 0.75 1 (0, 2)	0.97 ± 0.67 1 (0, 2)	0.2629[MW]
	50 mins	Mean ± SD Median (Min, Max)	0.97 ± 0.76 1 (0, 2)	0.8 ± 0.61 1 (0, 2)	0.3940[MW]
	60 mins	Mean ± SD Median (Min, Max)	0.83 ± 0.46 1 (0, 2)	0.8 ± 0.48 1 (0, 2)	0.7761[MW]

p-value			**< 0.001^{F*}**	0.2440^{F}	-
CRAVERO Scale	10 mins	Mean ± SD Median (Min, Max)	2.93 ± 0.94 3 (1, 4)	2.63 ± 0.85 2 (1, 4)	0.2019^{MW}
	20 mins	Mean ± SD Median (Min, Max)	2.5 ± 0.86 2 (2, 4)	2.53 ± 0.73 2 (1, 4)	0.4743^{MW}
	30 mins	Mean ± SD Median (Min, Max)	2.77 ± 0.82 3 (2, 4)	2.43 ± 0.63 2 (2, 4)	0.1078^{MW}
	40 mins	Mean ± SD Median (Min, Max)	2.7 ± 0.84 2 (2, 4)	2.4 ± 0.62 2 (2, 4)	0.1710^{MW}
	50 mins	Mean ± SD Median (Min, Max)	2.8 ± 0.81 3 (2, 4)	2.5 ± 0.63 2 (2, 4)	0.1507^{MW}
	60 mins	Mean ± SD Median (Min, Max)	2.6 ± 0.5 3 (2, 3)	2.5 ± 0.51 2.5 (2, 3)	0.4401^{MW}
p-value			**0.0042^{F*}**	0.4438^{F}	-

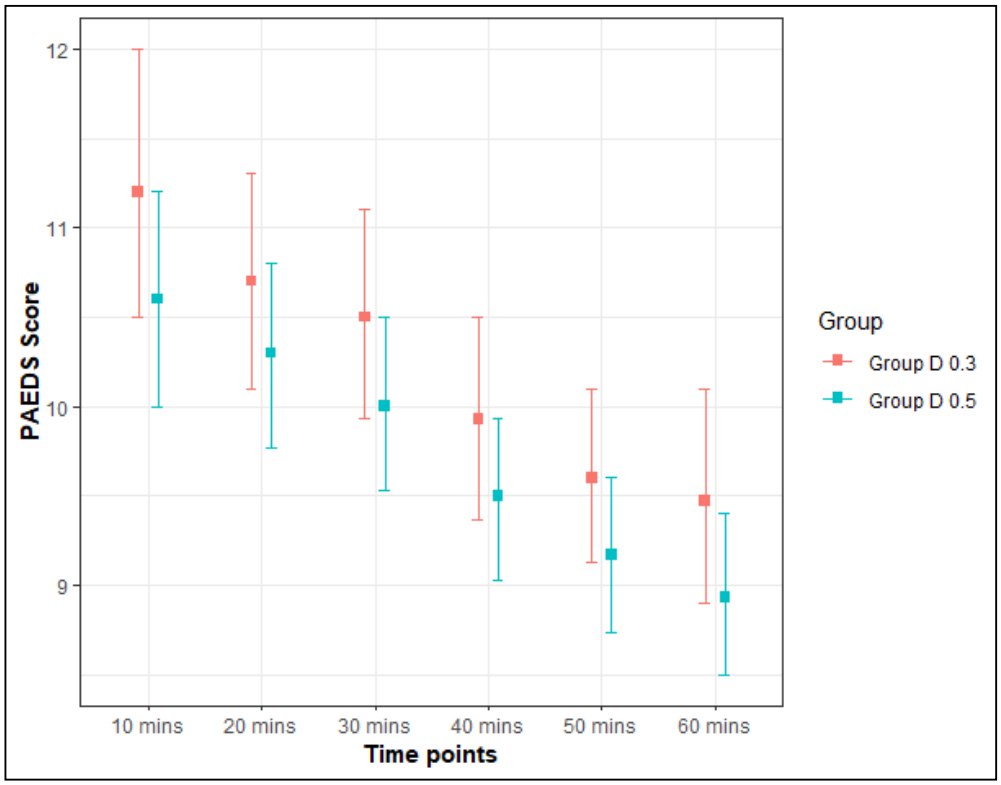
*Abbreviation: MW – Mann Whitney U test, F – Friedman test, * indicates statistical significance.*

For the PAEDS Score, both groups showed a gradual decrease over time, with Group D 0.3 having slightly higher mean scores than Group D 0.5 at all-time points. However, the Mann-Whitney U test showed no statistically significant differences between the groups at any time point (p-value > 0.05). The Friedman test revealed a statistically significant reduction in scores over time for both Group D 0.3 and Group D 0.5 (p-value < 0.001 for both groups).

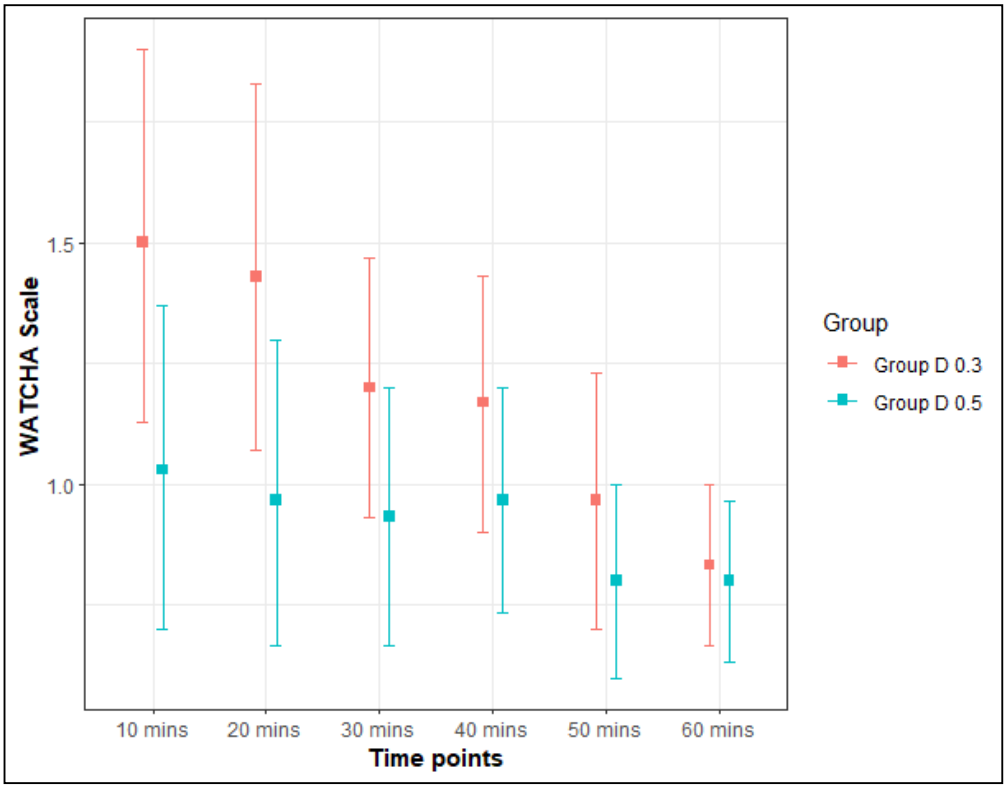
For the WATCHA Scale, the mean scores were slightly higher in Group D 0.3 compared to Group D 0.5 across all time points. While no significant differences were observed between the groups at any time point (p-value > 0.05), a significant reduction in scores over time was noted for Group D 0.3 (p-value < 0.001), but not for Group D 0.5".

For the CRAVERO Scale, Group D 0.3 consistently showed marginally higher mean scores compared to Group D 0.5 across almost all time points, with no significant differences between the groups (p-value > 0.05). The Friedman test indicated a significant change in scores over time for Group D 0.3 (p-value = 0.0042), but not for Group D 0.5.

Overall, while "Group D 0.3 demonstrated significant reductions over time in all three scales, no significant differences were observed between the two groups at individual time points for any variable".



'Figure 1: Mean plot of PAEDS score over time and group'



"Figure 2: Mean plot of WATCHA Scale over time and group".

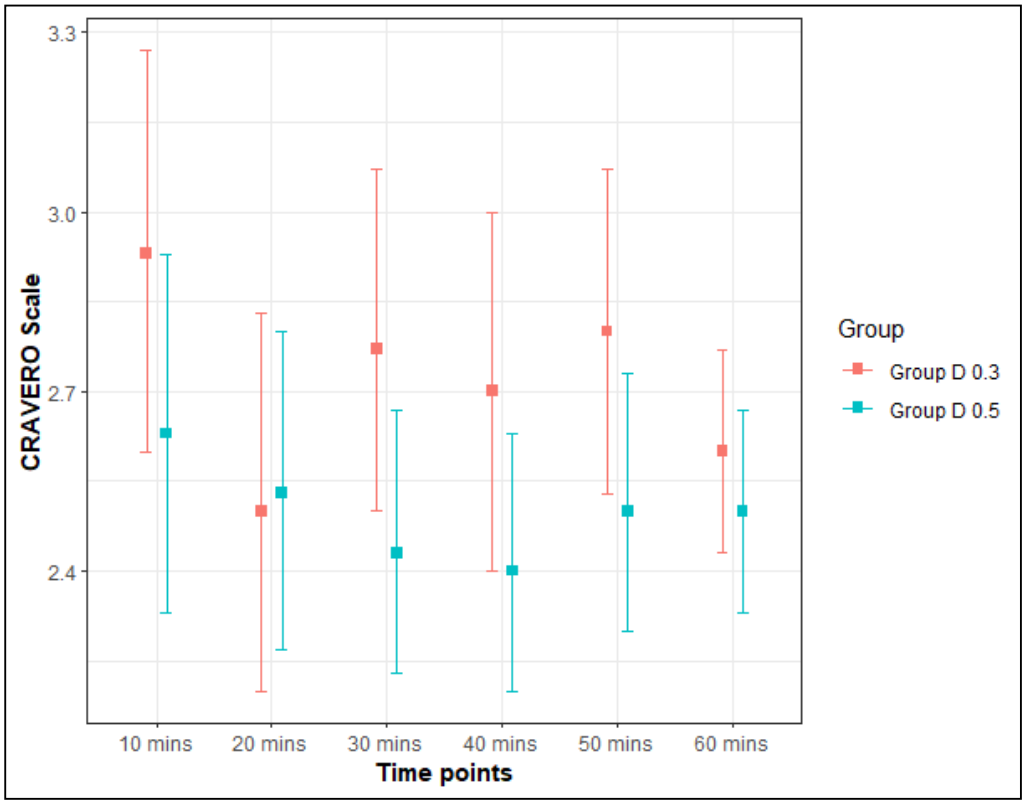


Figure 3: Mean plot of CRAVERO Scale over time and group.

"The following table gives the comparison of MAP, HR and RR over time and group.

Table 3: Comparison of MAP, HR and RR over time and group.

Variables	**Time points**	**Sub Category**	**Group D 0.3**	**Group D 0.5**	**p-value**
Mean arterial pressure	At drug	Mean ± SD Median (Min, Max)	72.13 ± 6.42 72 (60, 84)	70.93 ± 6.38 71 (60, 84)	0.4706[t]
	LMA out	Mean ± SD Median (Min, Max)	72.2 ± 6.82 73 (58, 86)	70.6 ± 6.55 71 (58, 86)	0.3578[t]
	10 mins	Mean ± SD Median (Min, Max)	70.6 ± 5.65 70 (58, 80)	69.6 ± 5.88 69 (58, 80)	0.5045[t]
	20 mins	Mean ± SD Median (Min, Max)	69.33 ± 5.68 68 (56, 80)	68.77 ± 6.02 68 (56, 80)	0.5690[MW]
	30 mins	Mean ± SD Median (Min, Max)	68.27 ± 5.88 68 (56, 80)	67.6 ± 5.81 68 (56, 80)	0.6603[t]
	40 mins	Mean ± SD Median (Min, Max)	67.07 ± 4.95 66 (58, 78)	66.13 ± 5.09 66 (58, 78)	0.4743[t]
	50 mins	Mean ± SD Median (Min, Max)	67.57 ± 4.71 68 (60, 79)	66.63 ± 4.79 67 (60, 79)	0.4497[t]
	60 mins	Mean ± SD Median (Min, Max)	66.83 ± 4.02 68 (60, 76)	65.7 ± 4.28 65 (60, 76)	0.2945[t]
p-value			**< 0.001[F]***	**< 0.001[F]***	-
Heart rate	At drug	Mean ± SD Median (Min, Max)	105.33 ± 22.54 96.5 (66, 153)	106.03 ± 24.19 101.5 (66, 153)	0.8996[MW]
	LMA out	Mean ± SD Median (Min, Max)	102.23 ± 16.54 98 (65, 139)	102.8 ± 17.86 98 (65, 139)	0.8990[t]
	10 mins	Mean ± SD Median (Min, Max)	104.7 ± 15.7 104 (68, 145)	103.8 ± 17.19 102 (68, 145)	0.8330[t]
	20 mins	Mean ± SD Median (Min, Max)	102.53 ± 14.77 102 (65, 138)	102.4 ± 15.71 100 (65, 138)	0.9731[t]

	30 mins	Mean ± SD Median (Min, Max)	102.73 ± 14.27 108 (60, 126)	101.13 ± 15.16 100 (60, 126)	0.5934^{MW}
	40 mins	Mean ± SD Median (Min, Max)	101 ± 13.55 105.5 (64, 128)	99.6 ± 13.78 99 (64, 128)	0.6506^{MW}
	50 mins	Mean ± SD Median (Min, Max)	97.2 ± 11.46 100 (66, 124)	97 ± 12.41 97 (66, 124)	0.6867^{MW}
	60 mins	Mean ± SD Median (Min, Max)	90.10 ± 7.88 89.50 (64, 107)	89.93 ± 8.36 88.50 (64, 108)	0.5154^{MW}
p-value			**< 0.001^{F}***	**< 0.001^{F}***	-
Respiratory rate	At drug	Mean ± SD Median (Min, Max)	22.83 ± 4.46 22 (14, 34)	22.77 ± 4.3 22 (14, 34)	0.8148^{MW}
	LMA out	Mean ± SD Median (Min, Max)	22.13 ± 2.89 22 (16, 29)	21.57 ± 3.37 22 (16, 29)	0.5542^{MW}
	10 mins	Mean ± SD Median (Min, Max)	24.27 ± 4.13 24 (18, 32)	23.07 ± 3.92 22 (18, 32)	0.2197^{MW}
	20 mins	Mean ± SD Median (Min, Max)	24.23 ± 4.3 23 (16, 34)	22.87 ± 3.95 22 (16, 34)	0.2050^{t}
	30 mins	Mean ± SD Median (Min, Max)	24.47 ± 4.26 24 (15, 30)	23.03 ± 3.65 24 (15, 30)	0.1930^{MW}
	40 mins	Mean ± SD Median (Min, Max)	24.2 ± 5.37 24 (14, 32)	22.37 ± 4.25 22 (14, 32)	0.2150^{MW}
	50 mins	Mean ± SD Median (Min, Max)	23.2 ± 3.83 23 (16, 28)	21.9 ± 3.59 22 (16, 28)	0.2099^{MW}
	60 mins	Mean ± SD Median (Min, Max)	22.73 ± 2.98 22 (16, 27)	21.73 ± 3.13 22 (16, 27)	0.2576^{MW}
p-value			**< 0.001^{F}***	**< 0.001^{F}***	-

*Abbreviation: t – Two sample t test, MW – Mann Whitney U test, F – Friedman's test, * indicates statistical significance.*

For MAP, both groups showed a gradual decline over time, with no statistically significant differences between the groups at any time point (p-value > 0.05). However, the Friedman test showed a significant reduction in MAP over time for both groups (p-value < 0.001).

For HR, both groups exhibited a decreasing trend over time, with no significant differences between the groups at any time points (p-value > 0.05). The Friedman test indicated a significant reduction in HR over time for both groups (p-value < 0.001).

For RR, both groups showed a similar pattern of gradual decline over time, with no significant differences between the groups at any time point (p-value > 0.05). The Friedman test demonstrated a significant reduction in RR over time for both groups (p-value < 0.001).

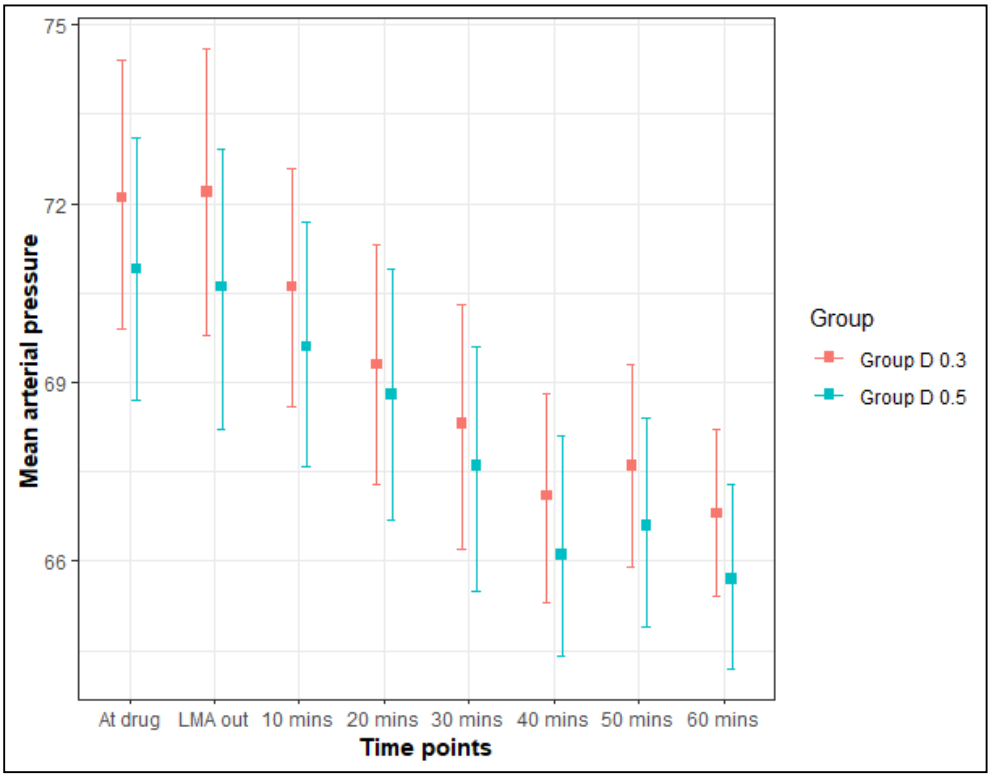


Figure 4: Mean plot of mean arterial pressure over time and groups.

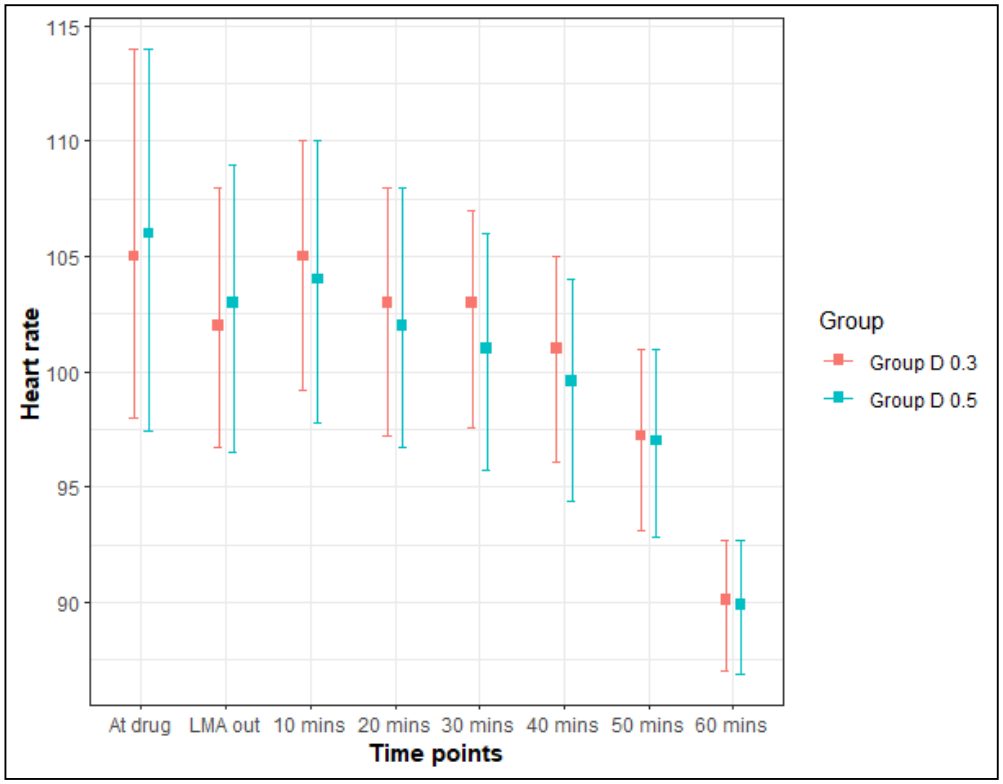


Figure 5: Mean plot of heart rate over time and groups.

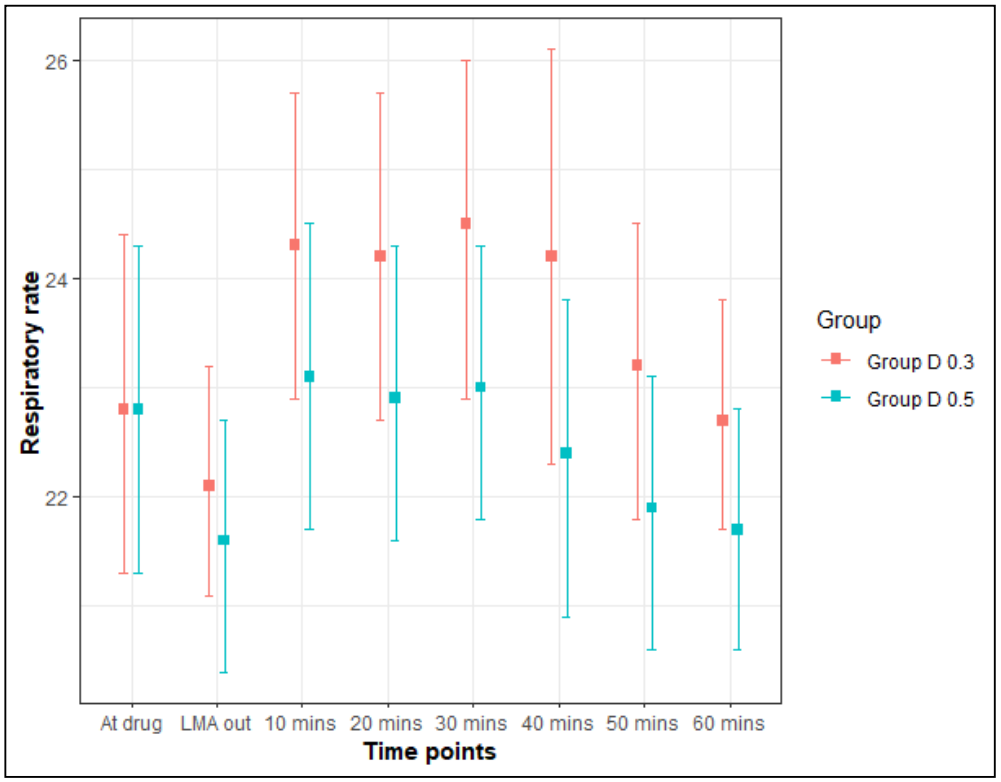


Figure 6: Mean plot of respiratory rate over time and groups.

Note: The FACES Pain Scale scores were consistently 0, and SpO2 levels remained at 100% across all time points and groups. Therefore, their analysis over time and between groups is not reported.

The following table gives the comparison of incidence of EA at different time points over groups.

Table 4: Comparison of incidence of EA at different time points over groups.

Time points	Group D 0.3	Group D 0.5	p-value
10 mins	7 (23.33%)	2 (6.67%)	0.1494^{MC}
20 mins	7 (23.33%)	2 (6.67%)	0.1494^{MC}
30 mins	7 (23.33%)	2 (6.67%)	0.1494^{MC}
40 mins	0 (0%)	0 (0%)	1^{MC}
50 mins	0 (0%)	0 (0%)	1^{MC}
60 mins	0 (0%)	0 (0%)	1^{MC}

Abbreviation: MC – Chi square test with Monte Carlo simulation.

Note: The incidence of emergence agitation (EA) is defined as a PAEDS score > 12. At 10, 20, and 30 minutes, EA was observed in 7 (23.33%) subjects in Group D 0.3 and 2 (6.67%) subjects in Group D 0.5. While EA incidence was higher in Group D 0.3 at these time points, the difference between the groups was not statistically significant (p-value = 0.1494). EA was completely resolved in both groups by 40 minutes and remained absent thereafter.

The following table gives the comparison of incidence of EA at different time points over groups.

Table 5: Comparison of incidence of EA at different time points over groups.

Bradycardia	**Group D 0.3**	**Group D 0.5**	**p-value**
No	25 (83.33%)	24 (80%)	0.7386[C]
Yes	5 (16.67%)	6 (20%)	

Abbreviation: C – Chi square test"

"Bradycardia was observed in 5 (16.67%) subjects in Group D 0.3 and 6 (20%) subjects in Group D 0.5. However, this difference was not statistically significant (p-value = 0.7386). Majority of the subjects in both groups did not experience bradycardia, with 25 (83.33%) subjects in Group D 0.3 and 24 (80%) subjects in Group D 0.5.

Note: The incidence of bradycardia is defined as a heart rate (HR) decrease of ≥30% from baseline value".

DISCUSSION

Emergence agitation is a real challenge facing anaesthesiologists during emergence and immediately after the operative period & in PACU. ED usually is short lived but, in the meantime, can lead to self-injury, create an unpleasant environment, require additional nursing staff, cause a delay in discharge time making it a concern to both the medical team and parents. EA, with an incidence of up to 80%, warrants management to avoid this unwelcome scene in the recovery room. A variety of factors could play a role such as age, preoperative behaviour or anxiety, inhalational and intravenous anaesthetic agents, hostile environment on awakening, type of surgery, parental absence on emergence from anaesthesia and pain[33].

One of the inevitable risk factors is the volatile anaesthetic agents used. Due to their low blood gas partition coefficient, inhalational agents like sevoflurane and desflurane are preferred over others for use in paediatric age group. Beneficial characteristics such as rapid onset and recovery are also the main reason for EA in these patients. "Welborn et al. (1996)[4]" investigated and compared the emergence and recovery times of three volatile anaesthetic agents—desflurane, sevoflurane & halothane—in paediatric subjects posted for ambulatory surgery. The study's results were that sevoflurane and desflurane resulted in faster emergence times compared to halothane. Desflurane showed the quickest emergence times, followed by sevoflurane, and halothane had the slowest. Recovery characteristics were similar between sevoflurane and desflurane, with both agents enabling faster return to baseline function compared to halothane. Faster recovery has been attributed as one of the mechanisms of EA, implying that the agents with a lower blood gas partition coefficient are the ones resulting in higher incidence of EA.

Sevoflurane's pharmacokinetics are similar in both children and adults. Though both, Desflurane and sevoflurane result in faster recovery, sevoflurane is preferred for mask induction due to its pleasant odour, lack of airway irritation, and ability to maintain stable haemodynamic. Studies report a higher incidence of excitement or agitation upon emergence . This is believed to be linked to the rapid recovery from anaesthesia and earlier awareness of postoperative pain. Though research confirms that proper pain management significantly reduces episodes of emergence agitation, however pain reduction measures are not proving to be enough to limit this complication[34,2,35,4,52,53].

Various methods, both non-pharmacological and pharmacological methods have been researched and employed for the management of EA. Non-pharmacological methods such as presence during induction (PPIA), using a smart phone to play various animated videos , taking prior tours of operation theatres have been suggested to prevent EA [54]. These approaches were used to reduce preoperative anxiety. Children often fear the unknown and the medical environment, which can result in distress during the induction of anaesthesia. By using these techniques, healthcare providers aimed to minimize psychological stress and create a more positive experience for the child, which may also improve the overall outcomes of the anaesthesia process. The use of these non-pharmacological strategies aligns with the growing focus on holistic care that addresses both physical and emotional needs, especially in children undergoing medical procedures.

Under pharmacological methods, various drugs have been tried in its attenuation such as opioid agents, benzodiazepines, ketamine, clonidine, magnesium and propofol have been studied and administered to decrease the incidence of EA [55,56,36].

Though all the above-mentioned drugs have been good options is mitigating EA, there hasn't yet been a consensus on a particular intervention. Broadly, strategies for mitigation of EA include Behavioural management, selection of volatile anaesthetic, preferred anaesthetic technique, choice of drugs such as "Benzodiazepine, Opioid, α_2-Agonist, Gabapentin, Melatonin, Propofol, Ketamine, Magnesium, Acupuncture, Regional anaesthesia and adequate pain control"[3].

A drug that is being popularly considered due to its promising results is Dexmedetomidine.

Dexmedetomidine, a selective alpha-2 blocker which causes a hypnotic state by activating α2-receptors which are located in the locus coeruleus, promoting unconsciousness that resembles natural sleep but with an advantageous feature that patient can still be easily aroused and continues to remain co-operative [13]. It has an analgesic effect that is due to its action on 'spinal cord(dorsal horn) along with inhibition of substance P' [38].

"Dexmedetomidine" is used in various routes & dosages in order to decrease the 'incidence of EA' and enhance the sedation scores in paediatric subjects [40,41]. Its administration in different routes has proved to give positive results in comparison with other drugs/ strategies or placebo. "Kumar L et al."[40], proved in his study that, when compared to oral midazolam, "intranasal dexmedetomidine at a dose of 1 μg/kg" resulted in better sedation scores during separation & during induction but "normal behavioural scores" in paediatric subjects.

"Prabhu MK et al."[41], had studied and compared the "effects of oral dexmedetomidine with oral midazolam in children undergoing elective surgeries under sevoflurane anaesthesia". They concluded that pre-medication with 'oral dexmedetomidine' gives smoother induction and better recovery, causing reduction in

the occurrence in the 'EA' and promotes enhanced and improved analgesia and better sedation when compared to 'oral midazolam'. In a placebo-controlled study, the effect of a single dose of IV dexmedetomidine in paediatric patients undergoing adenotonsillectomy was studied. The study showed that '0.5 $\mu g \cdot kg^{-1}$ dexmedetomidine' resulted in reduced 'emergence agitation' and pain scores significantly compared to the placebo group after 'sevoflurane anaesthesia'[42].

Following this, literature has also revealed research on the efficacy of different doses of dexmedetomidine, possibly to be able to find the most effective dose with reduced occurrence of untoward events like bradycardia & hypotension. A study was performed where 'one group received dexmedetomidine 1 μg/kg bolus, followed by 0.1 μg/kg/h until the surgery was completed was compared with a normal saline group which was volume matched', it was observed that intraoperative infusion of 'dexmedetomidine' in paediatric patients undergoing ambulatory surgery was shown to reduce the need for sevoflurane and decrease emergence agitation, allowing for smoother recovery without delaying discharge. However, bradycardia & hypotension are potential adverse effects which needed caution [57].

There is more research on various doses of dexmedetomidine given IV for reduction in sevoflurane associated agitation. Doses ranging from 0.3 mcg/kg upto 2mcg/kg, either as boluses, infusions or both, all consistently known to decrease the agitation scores but varying haemodynamic stability as the dose increased, have been used and effects described.

"Ramachandran et al."[58] conducted a 'randomised study' on eighty paediatric subjects between five and fourteen years of age posted for 'adenotonsillectomy /tonsillectomy under sevoflurane anaesthesia' and 'effects of two different doses of dexmedetomidine 0.3 μg/kg/h and 0.5 μg/kg/h infusion after a bolus dose of 0.5

μg/kg' were evaluated and compared for EA. Results of both groups were relatively comparable & the objective pain scores also was found to be not statistically significant. In the above research, measures to completely eliminate pain were not included as part of the anaesthetic management.

Pain is a significant risk factor for EA which needs to be eliminated well, in order to conclude that the resulting agitation is solely due to sevoflurane anaesthesia. Hence eliminating pain through caudal anaesthesia was crucial for assessing the efficacy of dexmedetomidine in preventing EA, as pain is a significant contributor to post-anaesthesia agitation. Caudal anaesthesia was incorporated as part of the standard anaesthetic plan to establish a more reliable conclusion of reduction in EA due to dexmedetomidine. With regards to caudal anaesthesia, in a study which compared 'three equal groups receiving either nalbuphine 0.1 mg/kg IV (Group N), dexmedetomidine 0.5 mic/kg IV in (Group D) or a saline solution (Group C) at 10 min before the end of surgery , in spite of all children having received caudal block, the occurence of EA in placebo group was relatively high (40%) suggesting the fact that painless treatment is not solely enough for the prevention of EA in paediatric patients after sevoflurane anaesthesia[51.]

The objective of the study was therefore to focus on comparing and studying the effects of 'two different doses of dexmedetomidine - 0.3 mcg/kg and 0.5 mcg/kg' , given as an infusion in paediatric patients posted for elective lower abdominal surgeries with sevoflurane as inhalational anaesthetic agent along with caudal anaesthesia to avoid pain as a confounder for EA. There has been paucity of studies comparing the effects of these two doses.

Sixty patients were enrolled in the study, with thirty subjects in 'each group (Group D0.3 and Group D 0.5)'. 'PAEDS score, Watcha scale and Cravero scale'

were implemented to assess the incidence and severity of EA. Scores were recorded at 10, 20, 30, 40, 50 and 60 minutes after completion of surgery and shifting to PACU, for the first hour. PAEDS score of more than 12 was taken as EA and a score of 15 or more was considered severe EA which would be managed with Inj. Fentanyl 1 mcg/kg as rescue medication.

In general, all three agitation scores were less in both groups, clearly indicating a reduction in EA by both doses. Both 'Group D 0.3 and Group D 0.5' exhibited scores which was statistically not significant (p- value >0.05). "Ramachandran et al.",[58] found in their study that "intraoperative dexmedetomidine infusion of 0.3 μg/kg/h and 0.5 μg/ kg/h following an initial bolus dose of 0.5μ g/kg is equally effective". The primary aim of attenuating EA with dexmedetomidine was fulfilled and was in consensus with the conclusion of the above-mentioned study.

However, 'Group D 0.3' demonstrated higher mean agitation scores compared to 'Group D 0.5' at the 10^{th} minute after shifting to PACU. This was followed by a reduction, overtime in all three scales, whereas Group D 0.5 maintained lesser scores from the time of shifting to PACU with no evident reduction over time. Statistically significant differences were not found to be the case between the two groups at any given point. Inference of this trend was concluded as a more stable reduction in EA with Group D 0.5 which was maintained from the time of shifting to PACU till the end of the first hour. Whereas Group D 0.3 showed higher mean scores (PAEDS <12) than the D 0.5 Group, in the beginning which eventually decreased and maintained by the end of the first hour. In a study, 'the mean PAED score, though significantly low ($p<0.05$) in group D receiving 0.3 mcg/kg Dexmedetomidine in comparison to group C receiving normal saline at 5, 15 & 30 minutes, there was a slightly higher mean score just after extubation & gradually decreased over the time, showing the incidence

of EA had peaking just after extubation to first 15 min. Total 7 (20%) patients in group D exhibited EA in comparison to 15 (42.9%) in group C' [59]. The higher mean scores in Group D 0.3, in the immediate 10 minutes of admission to PACU, could possibly be attributed to the removal of LMA as mentioned in the above study.

The primary goal was to assess and evaluate the 'effectiveness of two different doses of dexmedetomidine' in reducing the incidence and severity of EA. Considering the PAEDS scores, primarily, the aim has been achieved, with the mean score being <12 in both groups. It is important to include that Group D 0.5 was more consistent in its scores as time passed in recovery room, though both groups were equally effective.

None of the patients were given rescue medication in the PACU. Even if the initial mean scores were higher in Group D 0.3 with the child exhibiting slight disorientation, it eventually settled in the next 10 to 20 minutes, eliminating the need for any active intervention. Remaining 24 hours were uneventful with respect to patients in both groups.

"Garg et al." [60] conducted a study to assess the 'efficacy of dexmedetomidine in decreasing the incidence of emergence agitation (EA)' in 72 patients posted for nasal surgeries under desflurane anaesthesia. The patients received a '1 μg/kg bolus of dexmedetomidine followed by a 0.4 μg/kg/h infusion, compared to a placebo group'. The research findings revealed that 'dexmedetomidine' significantly reduced the 'incidence of EA(5.6% in the dexmedetomidine group vs. 52.8% in the placebo group)'. However, the use of 'dexmedetomidine;1 mcg/kg' was also associated with delayed extubation, residual sedation, and a longer stay in the 'post-anaesthesia care unit (PACU)'.

There were no such cases, which resulted in delay in extubation, prolonged PACU stay or residual sedation seen with either of the groups in this study. This

infers that both doses could be administer in children without the fear or doubt of higher dosing per kg body weight for the said age group.

The trends of hemodynamic parameters clearly demonstrated a decrease in MAP, HR and RR from 10 minutes after the removal of LMA. During removal of LMA an increase in the above-mentioned parameters was possibly due to the stressor response which eventually settled within 10 minutes on shifting to PACU. In our study, bradycardia was observed in 5 (16.67%) subjects in 'Group D 0.3' and 6 (20%) subjects in 'Group D 0.5', though this difference was not statistically significant (p-value = 0.7386). Most of the participants in both groups did not experience bradycardia, with 25 (83.33%) subjects in 'Group D 0.3' and 24 (80%) subjects in 'Group D 0.5' remaining unaffected. We defined bradycardia as reduction in 'heart rate (HR)' of 30% or more from the baseline value. RR decreased after and during the administration of the dexmedetomidine and eventually normalised 20 minutes after completion of infusion. The decrease in the RR was very minimal and stayed within 10% of the baseline rate. The study by "Patel et al."[45] compared "the use of dexmedetomidine (2 μg/kg over 10 minutes followed by 0.7 μg/kg/h) with fentanyl (1 μg/kg bolus) in 122 children aged 2-10 years undergoing adenotonsillectomy" . The results showed that 'emergence agitation (EA) was less common in the dexmedetomidine group as compared to the fentanyl group'. However, mean 'heart rate (HR) and systolic blood pressure' were significantly lower in the 'dexmedetomidine' group. This could be attributed to the higher dose of 'dexmedetomidine (0.7 μg/kg/h)' used in the study. In another research study conducted by Kim et al.,[57] '1 μg/kg bolus of dexmedetomidine followed by 0.1 μg/kg/h infusion was compared with normal saline group in children undergoing ambulatory surgeries. The results showed that the 'incidence of emergence agitation

was significantly lower in the dexmedetomidine group (5% vs 55%). However, mean arterial pressure (MAP) and heart rate (HR) were decreased by 22%–28% in the dexmedetomidine group. Atropine was administered to 6 children who developed bradycardia, with or without hypotension'.

In cumulation, we deciphered that '0.5 mcg/kg of dexmedetomidine', (a higher dose than 0.3 mcg/kg, though D 0.3 group could equally result in a considerable decrease in EA, statistically)', could bring about and maintain a more stable reduction in EA yet causing minimal adverse effects such as hypotension and bradycardia.

The secondary objective of assessing the haemodynamic parameters showed, no statistically significant variability between the groups and within each group, with the incidence of bradycardia being minimal or statistically not significant.

To avoid untoward hemodynamic instability, decision to administer the drug as an infusion over 10 minutes was taken. This attempt was more or else successful, with a smaller number of cases having experienced bradycardia, which was statistically not significant.

There was no incidence of '(PONV)Post operative nausea & vomiting' as long as the patients were in the PACU. No episodes of desaturation were noted during administration of dexmedetomidine intraoperatively and post operatively.

This research study doesn't come without it's few limitations that might affect the 'generalizability and interpretation' of its findings. Firstly, the use of subjective scoring systems like the PAED, Watcha and Cravero scales may lead to variability in results based on the anaesthesiologist's interpretation. Also, the study was performed at a single centre which limits its applicability to subsequent alternate settings. The absence of 'parental presence' during anaesthesia induction and lack of parental

anxiety assessment may have influenced the child's emotional response and recovery. Furthermore, postoperative nausea and vomiting (PONV) after 24 hours was not assessed, and the study did not take of the exact duration of PACU stay or sedation scores, both of which could provide useful insights into recovery. These factors suggest areas for improvement in future research.

CONCLUSION

This study concluded that both ‘0.3 mcg/kg and 0.5 mcg/kg doses of intravenous dexmedetomidine’ are comparable and effective in ‘reducing emergence agitation in paediatric patients undergoing lower abdominal surgeries’. Both doses showed significant reductions in the severity of EA, with no substantial differences in efficacy. While the ‘0.5 mcg/kg’ dose provided a more consistent reduction in EA, the ‘0.3 mcg/kg’ dose was also highly effective and demonstrated a similar safety profile. Therefore, based on the findings, we recommend both doses—'0.3 mcg/kg and 0.5 mcg/kg’ as safe and effective options for preventing emergence agitation in paediatric anaesthesia. These doses provide adequate relief from EA without the need for higher doses that may prolong recovery time or cause more significant side effects. With both doses having the advantage of haemodynamic stability and minimal chance of fluctuations they can be secure and better choices for the purpose of EA reduction. This study supports the use of dexmedetomidine in paediatric anaesthesia for improving patient comfort and reducing EA while maintaining overall safety.

SUMMARY

Emergence agitation (EA) is a significant concern for paediatric patients following general anaesthesia, especially in surgeries involving younger children. EA can lead to discomfort, distress, and potentially harmful complications if not managed properly. Dexmedetomidine, a selective α2-adrenergic agonist, has shown promise in reducing EA due to its sedative, anxiolytic, and analgesic properties. This study aimed to compare the effects of two intravenous doses of dexmedetomidine, 0.3 mcg/kg and 0.5 mcg/kg, on the reduction of EA in paediatric patients undergoing lower abdominal surgeries.

The study, titled *"Comparison of Two Different Doses of Dexmedetomidine in Attenuation of Sevoflurane associated Emergence Agitation in Paediatric Patients, posted for Lower Abdominal Surgeries – A Hospital-Based, Randomized Controlled Trial,"* investigated the effects of two different doses of dexmedetomidine on emergence agitation (EA) in pediatric patients. A total of 60 children were randomly assigned to two groups: Group D 0.3, which received 0.3 mcg/kg of dexmedetomidine, and Group D 0.5, which received 0.5 mcg/kg. EA severity was assessed using three validated scoring systems: the Paediatric Anaesthesia Emergence Delirium (PAED) score, the Watcha scale, and the Cravero scale at multiple intervals, from immediately after Proseal LMA removal to up to 24 hours postoperatively.

The findings indicated that both doses effectively reduced EA, as demonstrated by a steady decline in agitation scores over time. Although Group D 0.3 had slightly higher PAED scores than Group D 0.5, the difference was not statistically significant ($p > 0.05$), suggesting that both doses were similarly effective. However, Group D 0.5 exhibited a more stable reduction in agitation, with consistently lower scores throughout the PACU stay, particularly during the first hour. In contrast, Group

D 0.3 initially showed higher agitation scores, which gradually declined over time. This suggests that while both doses are effective, the 0.5 mcg/kg dose may provide a more sustained calming effect during recovery.

The study also monitored hemodynamic parameters, including mean arterial pressure (MAP), heart rate (HR), and respiratory rate (RR). An initial increase in these parameters was observed following LMA removal, likely due to stress, but they returned to baseline within 10 minutes of PACU admission. Bradycardia was recorded in a small proportion of patients in both groups (16.67% in Group D 0.3 and 20% in Group D 0.5), though the difference was not statistically significant ($p = 0.7386$). Most patients (around 80%) did not experience significant bradycardia. Additionally, respiratory rate slightly decreased during dexmedetomidine infusion but normalized within 20 minutes post-infusion.

Since pain can contribute to EA, the study also assessed pain using the Visual Analog Scale (VAS) and Faces Pain Scale. No significant differences in pain scores were found between the groups, with all patients reporting a score of 0, ensuring that the reductions in EA were independent of pain relief owing to the effect of caudal anaesthesia.

Dexmedetomidine's dual action, both as a sedative and analgesic, likely contributed to its effectiveness in reducing EA. This dual effect makes dexmedetomidine a unique and useful agent in paediatric anaesthesia, especially for managing EA during the recovery phase.

Furthermore, the study found that there were no significant episodes of postoperative nausea and vomiting (PONV) or desaturation events in the operating room or PACU, which further supports the safety profile of dexmedetomidine in this

patient population. This is an important finding, as PONV is a common concern following general anaesthesia, especially in paediatric patients.

It was hence, concluded that both doses - 0.3mcg/kg and 0.5 mcg/kg of dexmedetomidine, provided similar efficacy in reducing EA without the drawback of haemodynamic instability.

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ANNXURES - 1

INFORMED CONSENT FORM

"COMPARISON OF TWO DIFFERENT DOSES OF DEXMEDETOMIDINE IN ATTENUATION OF SEVOFLURANE ASSOCIATED EMERGENCE AGITATION IN PAEDIATRIC PATIENTS, POSTED FOR LOWER ABDOMINAL SURGERIES- A HOSPITAL BASED, RANDOMIZED CONTROLLED TRIAL"

Name of Student/Principal Investigator: BA0122019

Name of Guide/Co Investigators:

Introduction:

Post anaesthetic agitation occurs frequently in paediatric population, with incidence range of 10% to 80%. It is a state of acute confusion during recovery from anaesthesia which may present as purposeless hyperactive/ agitated physical behavior, restlessness, hallucinations, disorientation and crying. This is a distressing and disappointing condition for the parents. In this state the child cannot be consoled by caregivers too.

Because of its high incidence and above-mentioned reasons, interventions in preventing it are important for the anesthesia provider.

Dexmedetomidine, United States FDA approved drug is useful in this condition. Multiple studies are being conducted on the best practice of timing, method and dosing of this drug. I am too, conducting a similar study and comparing the effects of three different doses of this drug in attenuation of post anesthetic agitation**.**

Explanation of procedure: On the day of surgery, after obtaining consent from parents and verbal assent from the patient and confirming the NBM status, in the operating room, the patient is premedicated with Inj. Glycopyrrolate 0.005mg/kg IV and Inj. Fentanyl 1-2mcg/kg IV. Patient is induced with Inj. Ketamine 1-2 mg/kg IV following which a Laryngeal Mask Airway is inserted according to the patient's body weight.

Patient is maintained on inhalational oxygen, nitrous oxide and Sevoflurane. Following this, in lateral position, 1 ml/ kg of 0.25% Bupivacaine, Caudal anaesthesia is given.

Different vital parameters will be monitored throughout and 10 mins before the end of surgery the patient will receive the study drug as per allocated group. Post operative assessment of agitation will be done based on standard scoring systems.

If you agree to enroll your child in my study, I will be enquiring regarding your child's present, past and family history. After which he/she will be examined in detail. He/she will be allotted into one of the two groups randomly using computer generated software.

- Group D 0.3: who will receive 0.3 mcg/kg of Inj. Dexmedetomidine IV
- Group D 0.5: who will receive 0.5 mcg/kg of Inj. Dexmedetomidine IV

Slowly over 10 minutes prior to the end of surgery respectively.

Withdrawal from participation in the study:

Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will not get any benefits by participating in this study. The data gathered will help children suffering from this transient condition at large post operatively and decrease stress for parents.

Possible benefits from participating in the study: You will not get any benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study**.**

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify your child. Your child's identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the principal investigator.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact the principal Investigator

If you have any question or complaints with regardto your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**COMPARISON OF TWO DIFFERENT DOSES OF DEXMEDETOMIDINE IN ATTENUATION OF SEVOFLURANE ASSOCIATED EMERGENCE AGITATION IN PAEDIATRIC PATIENTS, POSTED FOR LOWER ABDOMINAL SURGERIES- A HOSPITAL BASED, RANDOMIZED CONTROLLED TRIAL**". My signature below indicates that I have decided to participate, and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions, and they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant: Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator: Signature of the investigator:

ANNXURES - II

PROFORMA

"COMPARISON OF TWO DIFFERENT DOSES OF DEXMEDETOMIDINE IN ATTENUATION OF SEVOFLURANE ASSOCIATED EMERGENCE AGITATION IN PAEDIATRIC PATIENTS, POSTED FOR LOWER ABDOMINAL SURGERIES- A HOSPITAL BASED, RANDOMIZED CONTROLLED TRIALAT DR. PRABHAKAR KORE CHARITABLE HOSPITAL, BELAGAVI"

Group allotted:

Name:

Age:

Gender:

Weight:

Height:

Date of surgery:

Address:

Occupation

Pre examination evaluation Past History

Congenital disorders	**Yes**	**No**
ICU admission	**Yes**	**No**
URTI	**Yes**	**No**
H/o previous surgery	**Yes**	**No**
If yes, Previous anaesthetic complications	**Yes**	**No**

General physical examination

Weight (Kg): Temperature:

Pallor :

Pedal edema:

Cyanosis:

Clubbing:

PR:

BP:

RR:

Musculoskeletal disorders:

Teeth:

Jaw movements:

Airway assessment :

Spine:

Investigations

Hb%:

Platelet Count :

TLC:

INR:

FBS:

Systemic examination:

CNS: RS:

CVS: GIT:

Premedication:

Preoperative physical status:

American society of anesthesiologist:

I ☐ II ☐

Diagnosis:

Proposed surgery:

Monitors attached:

Pulse oximetry : NIBP:

ECG:

ASSESSMENT:

1.PAED SCORE (Paediatric anaesthetic emergence delirium score)

Behaviour	Not at all	Just a little	Quite a bit	Very much	extremely
The child makes eye contact with the care giver	4	3	2	1	0
The child's action are purposeful	4	3	2	1	0
The Child is aware of his/her surroundin gs	4	3	2	1	0
The child is restless	0	1	2	3	4
The child is inconsolab le	0	1	2	3	4

2. WATCHA SCALE

BEHAVIOUR	SCORE
Asleep	0
Calm	1
Crying,but can be consoled	2
Crying,but cannot be consoled	3
Agitated and thrashing around	4

HEMODYNAMIC PARAMETERS

PARAMETERS	AT THE TIME OF DRUG ADMINISTRATION	ON REMOVAL OF LMA
BLOOD PRESSURE		
HEART RATE		
RESPIRATORY RATE		
SpO2		
EtCO2		

3. CRAVERO SCALE

BEHAVIOUR	SCORE
Obtunded with no response to stimulation	1
Asleep but responsive to movement or stimulation	2
Awake and responsive	3
Crying (for > 3mins)	4
Thrashing behaviour that requires restraint	5

PARAMETERS	10 MINS AFTER SURGERY	20 MINS AFTER SURGERY	30 MINS AFTER SURGERY	40 MINS AFTER SURGERY	50 MINS AFTER SURGERY	1 HOUR AFTER SUGERY
BLOOD PRESSURE						
HEART RATE						
RESPIRATORY RATE						
SpO2						

Hemodynamic parameter assessment for 24 hours:

Eventful

Uneventful

PAIN ASSESSMENT- TO DIFFERENTIATE FROM EMERGENCE AGITATION

1.Visual Analogue Scale/Numerical Rating Scale

On a Scale of 0-10, with 0 being no pain and 10 being the worst pain you can

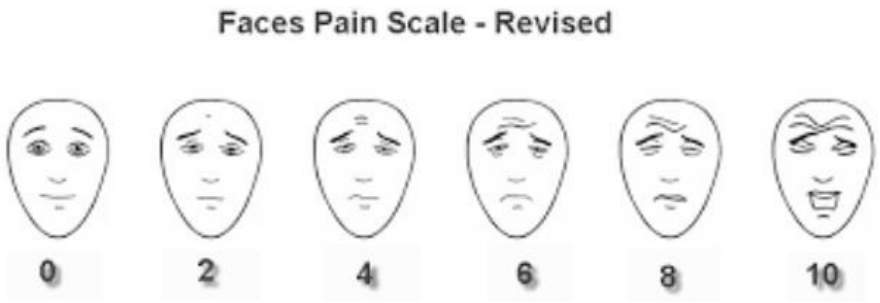
imagine, what are you feeling right now on movement and at rest?

Self-reporting pain tool for children aged 5 years and above.

0 No pain	1	2	3	4	5	6	7	8	9	10 Worst pain

2. The Faces Pain Scale Revised (Bieri faces)

A self-report tool for children ages 3 years and above.



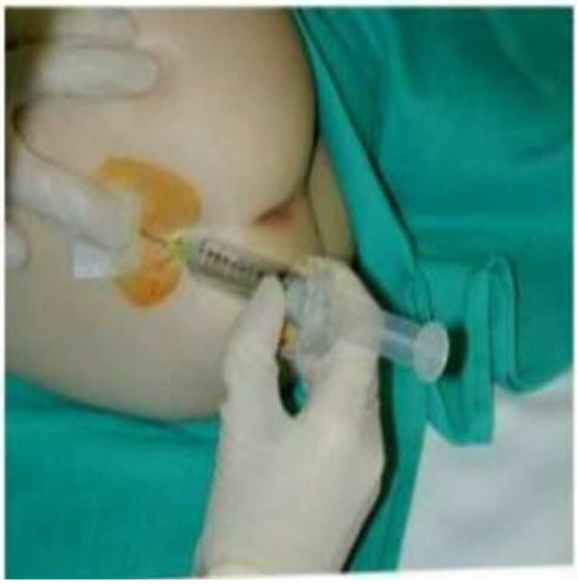
ANNXURES - III

PHOTOGRAPHS

a) Sevoflurane vapouriser

b) Dexmedetomidine ampoule

c) Haemodynamic monitors



d) Performing Caudal anaesthesia

e) Dexmedetomidine infusion

ANNXURES - IV

MASTER CHART

	DEX 0.3 GROUP																																																											
	PARAMETERS																																																											
SL.NO.	PAEDS SCORE							WATCHA SCALE						CRAVERO SCALE						MEAN ARTERIAL PRESSURE								HEART RATE								RESPIRATORY RATE								SpO2								EtCO2		FACES PAIN SCALE						
			10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	10 MINS	20 MINS	30 MOINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20MINS	30 MINS	40 MINS	50 MINS	60 MINS
1	1.3y/M	11	10	10	8	8	7	1	1	1	1	0	0	2	2	3	3	3	3	83	78	74	72	70	70	72	72	84	65	78	65	60	64	66	64	22	24	24	20	18	18	18	18	100	100	100	100	100	100	100	100	28	28	0	0	0	0	0	0	
2	2.6y/F	12	11	11	10	9	9	0	0	0	0	0	0	2	2	2	2	2	2	78	74	72	68	66	68	64	68	74	69	68	70	70	72	70	70	20	18	18	19	20	22	22	24	100	100	100	100	100	100	100	100	34	30	0	0	0		0	0	
3	3.7y/M	10	10	9	7	7	7	2	2	2	1	1	1	4	3	3	3	3	3	78	72	70	70	72	68	74	70	131	139	145	138	116	112	110	107	18	20	18	16	15	14	16	18	100	100	100	100	100	100	100	100	30	34	0	0	0	0	0	0	
4	4.5y/M	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	94	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
5	5.4y/M	12	11	10	10	9	9	0	0	0	1	1	1	2	2	2	2	3	3	82	84	80	80	80	78	79	76	110	115	115	112	120	96	100	90	26	27	26	26	26	25	25	24	100	100	100	100	100	100	100	100	27	25	0	0	0	0	0	0	
6	6. 7y/F	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	96	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
7	7. 3y/M	9	9	9	9	9	8	2	2	2	2	1	1	3	2	3	3	3	3	68	69	70	70	66	64	62	60	120	118	110	112	114	110	100	88	26	24	26	24	24	24	24	22	100	100	100	100	100	100	100	100	27	25	0	0	0	0	0	0	
8	8. 2y/F	6	6	6	6	9	6	2	2	2	2	2	2	4	2	3	3	3	3	68	67	66	64	60	62	64	64	130	132	130	128	126	128	124	87	24	22	24	23	24	24	24	24	100	100	100	100	100	100	100	100	26	28	0	0	0	0	0	0	
9	9. 8y/M	10	9	9	9	9	8	1	1	1	1	1	1	2	2	2	2	2	3	70	72	68	66	68	66	68	66	98	85	85	86	85	84	84	88	18	16	18	18	20	16	18	20	100	100	100	100	100	100	100	100	30	30	0	0	0	0	0	0	
10	10. 10y/M	10	10	10	9	9	9	1	1	1	1	1	1	3	2	3	3	3	3	70	72	68	66	66	68	70	70	99	98	95	94	99	96	94	87	18	16	18	20	18	16	16	16	100	100	100	100	100	100	100	100	31	30	0	0	0	0	0	0	
11	11. 4y/M	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	99	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
12	12. 7y/M	11	11	10	9	9	9	1	1	1	1	1	1	2	2	2	2	3	3	72	70	68	70	72	68	64	60	84	88	112	100	97	100	92	88	24	29	32	34	30	26	27	27	100	100	100	100	100	100	100	100	34	34	0	0	0	0	0	0	
13	13. 2y/M	12	12	11	10	9	10	0	0	0	0	0	0	2	2	2	2	2	2	78	74	76	70	72	68	70	70	95	85	100	97	95	92	93	88	34	28	32	30	29	30	27	27	100	100	100	100	100	100	100	100	29	28	0	0	0	0	0	0	
14	14. 2y/M	12	12	11	11	9	10	0	0	0	0	0	1	1	2	2	2	2	2	62	60	58	56	60	62	60	63	109	112	129	116	114	112	100	89	33	24	28	26	22	24	22	22	100	100	100	100	100	100	100	100	34	33	0	0	0	0	0	0	
15	15. 6y/M	11	10	10	10	9	9	1	1	1	0	0	0	2	2	2	2	3	3	84	86	80	78	77	76	74	72	93	98	90	88	87	82	84	88	14	22	22	20	22	24	22	22	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
16	16. 7y/M	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	95	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
17	17. 3y/F	12	12	11	10	9	10	0	0	0	0	0	1	2	2	2	2	2	2	74	78	77	76	76	74	72	70	153	113	115	114	111	110	106	88	28	22	24	22	23	22	22	22	100	100	100	100	100	100	100	100	38	30	0	0	0	0	0	0	
18	18. 3y/F	11	11	11	10	9	9	0	0	0	0	0	1	2	2	2	2	2	2	64	66	64	68	63	62	62	62	123	110	110	108	106	102	102	94	24	22	22	23	24	22	22	22	100	100	100	100	100	100	100	100	28	30	0	0	0	0	0	0	
19	19. 9y/M	9	9	9	9	9	9	1	1	1	1	1	1	3	2	2	2	2	2	72	70	74	72	70	68	68	66	118	110	98	96	97	96	94	88	24	22	20	22	24	20	20	20	100	100	100	100	100	100	100	100	32	28	0	0	0	0	0	0	
20	20. 6y/M	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	98	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
21	21. 2y/M	12	11	11	11	9	10	2	1	1	1	1	1	4	2	3	2	2	2	60	58	62	59	56	58	60	62	135	123	118	116	116	112	110	90	24	24	26	25	24	22	24	22	100	100	100	100	100	100	100	100	34	30	0	0	0	0	0	0	
22	22. 10y/M	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	96	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
23	23.9y/M	10	9	9	9	9	9	1	1	1	1	0	0	3	2	2	2	2	2	84	86	80	78	77	76	74	72	93	98	90	88	87	82	84	88	14	22	22	20	22	24	22	22	100	100	100	100	100	100	100	100	29	28	0	0	0	0	0	0	
24	24.7y/M	9	9	9	9	9	9	1	1	1	1	1	1	3	2	2	2	2	2	69	68	66	67	64	62	64	62	86	96	98	100	100	99	97	90	18	20	20	22	24	24	24	24	100	100	100	100	100	100	100	100	34	33	0	0	0	0	0	0	
25	25.4y/M	10	10	10	10	9	8	1	1	1	1	1	1	2	2	2	2	2	2	74	78	77	76	76	74	72	70	153	113	115	114	111	110	106	88	28	22	24	22	23	22	22	22	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
26	26.6y/F	12	11	11	11	9	10	2	1	1	1	1	1	4	2	3	2	2	2	64	66	64	68	63	62	62	62	123	110	110	108	106	102	102	96	24	22	22	23	24	22	22	22	100	100	100	100	100	100	100	100	28	30	0	0	0	0	0	0	
27	27.5y/F	10	10	10	9	9	9	1	1	1	1	0	0	2	2	2	2	2	2	72	70	74	72	70	68	68	66	118	110	98	96	97	96	94	92	24	22	20	22	24	20	20	20	100	100	100	100	100	100	100	100	38	30	0	0	0	0	0	0	
28	28.4y/M	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	98	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	
29	29.3y/M	9	9	9	9	9	8	2	2	2	2	1	1	3	2	3	3	3	3	60	58	62	59	56	58	60	62	135	123	118	116	116	112	110	89	24	24	26	25	24	22	24	22	100	100	100	100	100	100	100	100	32	28	0	0	0	0	0	0	
30	30.4y/F	9	9	9	9	9	8	2	2	2	2	1	1	3	2	3	3	3	3	74	72	78	79	72	70	68	64	66	92	86	100	96	98	94	90	22	18	20	22	20	19	17	18	100	100	100	100	100	100	100	100	30	27	0	0	0	0	0	0	

	DEX 0.5 GROUP	PAEDS SCORE						WATCHA SCALE						CRAVERO SCALE						MEAN ARTERIAL PRESSURE						HEART RATE							RESPIRATORY RATE							SpO2						EtCO2		FACES PAIN SCALE						
	PARAMETERS	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20 MINS	30 MINS	40 MINS	50 MINS	60 MINS	AT DRUG	LMA OUT	10 MINS	20MINS	30 MINS	40 MINS	50 MINS	60 MINS	
1	1.3y/M	11	10	10	8	8	7	1	1	1	1	0	0	2	2	3	3	3	3	83	78	74	72	70	70	72	72	84	65	78	65	60	64	66	64	22	24	24	20	18	18	18	18	100	100	100	100	100	100	100	100	28	28	0	0	0	0	0	0	0	
2	2.6y/F	12	11	11	10	9	9	0	0	0	0	0	0	2	2	2	2	2	2	78	74	72	68	66	68	64	68	74	69	68	70	70	72	70	70	20	18	18	19	20	22	22	24	100	100	100	100	100	100	100	100	34	30	0	0	0	0	0	0	0	
3	3.7y/M	10	10	9	7	7	7	2	2	2	1	1	1	4	3	3	3	3	3	78	72	70	70	72	68	74	70	131	139	145	138	116	112	110	108	18	20	18	16	15	14	16	18	100	100	100	100	100	100	100	100	30	34	0	0	0	0	0	0	0	
4	4.5y/M	10	10	9	9	9	8	0	0	1	1	1	0	2	2	2	2	3	3	68	64	66	68	67	64	66	64	90	85	80	82	80	85	85	98	22	23	22	22	23	24	24	22	100	100	100	100	100	100	100	100	26	27	0	0	0	0	0	0	0	
5	5.4y/M	12	11	10	10	9	9	0	0	0	1	1	1	2	2	2	2	3	3	82	84	80	80	80	78	79	76	110	115	115	112	120	96	100	99	26	27	26	26	26	25	25	24	100	100	100	100	100	100	100	100	27	25	0	0	0	0	0	0	0	
6	6. 7y/F	10	10	10	9	9	9	0	0	0	0	1	1	2	2	2	2	2	2	72	74	70	68	68	66	68	68	90	95	94	92	90	89	90	98	22	22	22	20	24	24	24	24	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	0	
7	7. 3y/M	9	9	9	9	8	8	2	2	2	2	1	1	3	3	3	3	3	3	68	69	70	70	66	64	62	60	120	118	110	112	114	110	100	89	26	24	26	24	24	24	24	22	100	100	100	100	100	100	100	100	27	25	0	0	0	0	0	0	0	
8	8. 2y/F	6	6	6	6	6	6	2	2	2	2	2	2	4	4	3	3	3	3	68	67	66	64	60	62	64	64	130	132	130	128	126	128	124	88	24	22	24	23	24	24	24	24	100	100	100	100	100	100	100	100	26	28	0	0	0	0	0	0	0	
9	9. 8y/M	10	9	9	9	9	8	1	1	1	1	1	1	2	2	2	2	2	3	70	72	68	66	68	66	68	66	98	85	85	86	85	84	84	87	18	16	18	18	20	16	18	20	100	100	100	100	100	100	100	100	30	30	0	0	0	0	0	0	0	
10	10. 10y/M	10	10	10	9	9	9	1	1	1	1	1	1	3	3	3	3	3	3	70	72	68	66	66	68	70	70	99	98	95	94	99	96	94	88	18	16	18	20	18	16	16	16	100	100	100	100	100	100	100	100	31	30	0	0	0	0	0	0	0	
11	11. 4y/M	11	11	10	10	10	9	0	0	0	1	1	1	2	2	2	3	3	3	69	68	66	67	68	64	64	66	142	136	127	124	122	122	122	98	22	22	22	24	24	22	20	22	100	100	100	100	100	100	100	100	31	29	0	0	0	0	0	0	0	
12	12. 7y/M	11	11	10	9	9	9	1	1	1	1	1	1	2	2	2	2	3	3	72	70	68	70	72	68	64	60	84	88	112	100	97	100	92	89	24	29	32	34	30	26	27	27	100	100	100	100	100	100	100	100	34	34	0	0	0	0	0	0	0	
13	13. 2y/M	12	12	11	10	10	10	0	0	0	0	0	0	2	2	2	2	2	2	78	74	76	70	72	68	70	70	95	85	100	97	95	92	93	88	34	28	32	30	29	30	27	27	100	100	100	100	100	100	100	100	29	28	0	0	0	0	0	0	0	
14	14. 2y/M	12	12	11	11	11	10	0	0	0	0	0	1	1	1	2	2	2	2	62	60	58	56	60	62	60	63	109	112	129	116	114	112	100	87	33	24	28	26	22	24	22	22	100	100	100	100	100	100	100	100	34	33	0	0	0	0	0	0	0	
15	15. 6y/M	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	87	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	0	
16	16. 7y/M	11	11	11	10	10	10	0	0	0	0	1	1	2	2	2	2	2	3	69	68	66	67	64	62	64	62	86	96	98	100	100	99	97	99	18	20	20	22	24	24	24	24	100	100	100	100	100	100	100	100	28	30	0	0	0	0	0	0	0	
17	17. 3y/F	12	12	11	10	10	10	0	0	0	0	0	1	2	2	2	2	2	2	74	78	77	76	76	74	72	70	153	113	115	114	111	110	106	89	28	22	24	22	23	22	22	22	100	100	100	100	100	100	100	100	38	30	0	0	0	0	0	0	0	
18	18. 3y/F	11	11	11	10	9	9	0	0	0	0	0	1	2	2	2	2	2	2	64	66	64	68	63	62	62	62	123	110	110	108	106	102	102	89	24	22	22	23	24	22	22	22	100	100	100	100	100	100	100	100	28	30	0	0	0	0	0	0	0	
19	19. 9y/M	9	9	9	9	9	9	1	1	1	1	1	1	3	3	2	2	2	2	72	70	74	72	70	68	68	66	118	110	98	96	97	96	94	87	24	22	20	22	24	20	20	20	100	100	100	100	100	100	100	100	32	28	0	0	0	0	0	0	0	
20	20. 6y/M	10	10	10	10	9	8	1	1	1	1	1	1	2	2	2	2	2	2	64	66	64	62	64	60	62	60	104	98	98	96	94	94	92	97	20	16	20	18	19	18	17	16	100	100	100	100	100	100	100	100	30	27	0	0	0	0	0	0	0	
21	21. 2y/M	12	11	11	11	10	10	2	1	1	1	1	1	4	3	3	2	2	2	60	58	62	59	56	58	60	62	135	123	118	116	116	112	110	87	24	24	26	25	24	22	24	22	100	100	100	100	100	100	100	100	34	30	0	0	0	0	0	0	0	
22	22. 10y/M	10	10	10	9	9	9	1	1	1	1	0	0	2	2	2	2	2	2	74	72	78	79	72	70	68	64	66	92	86	100	96	98	94	97	22	18	20	22	20	19	17	18	100	100	100	100	100	100	100	100	26	24	0	0	0	0	0	0	0	
23	23.9y/M	10	9	9	9	9	9	1	1	1	1	0	0	3	3	2	2	2	2	84	86	80	78	77	76	74	72	93	98	90	88	87	82	84	88	14	22	22	20	22	24	22	22	100	100	100	100	100	100	100	100	29	28	0	0	0	0	0	0	0	
24	24.7y/M	9	9	9	9	9	9	1	1	1	1	1	1	3	3	2	2	2	2	69	68	66	67	64	62	64	62	86	96	98	100	100	99	97	88	18	20	20	22	24	24	24	24	100	100	100	100	100	100	100	100	34	33	0	0	0	0	0	0	0	
25	25.4y/M	10	10	10	10	9	8	1	1	1	1	1	1	2	2	2	2	2	2	74	78	77	76	76	74	72	70	153	113	115	114	111	110	106	89	28	22	24	22	23	22	22	22	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	0	
26	26.6y/F	12	11	11	11	10	10	2	1	1	1	1	1	4	3	3	2	2	2	64	66	64	68	63	62	62	62	123	110	110	108	106	102	102	88	24	22	22	23	24	22	22	22	100	100	100	100	100	100	100	100	28	30	0	0	0	0	0	0	0	
27	27.5y/F	14	13	13	12	12	12	3	3	2	2	2	1	4	4	4	4	4	3	72	74	70	68	68	66	68	68	90	95	104	102	108	109	100	87	22	22	28	29	30	32	28	26	100	100	100	100	100	100	100	100	29	27	0	0	0	0	0	0	0	
28	28.4y/M	10	9	9	9	9	9	1	1	1	1	0	0	3	3	2	2	2	2	64	66	64	62	64	60	62	60	104	98	98	96	94	94	92	99	20	16	20	18	19	18	17	16	100	100	100	100	100	100	100	100	28	30	0	0	0	0	0	0	0	
29	29.3y/M	9	9	9	9	8	8	2	2	2	2	1	1	3	3	3	3	3	3	60	58	62	59	56	58	60	62	135	123	118	116	116	112	110	87	24	24	26	25	24	22	24	22	100	100	100	100	100	100	100	100	32	28	0	0	0	0	0	0	0	
30	30.4y/F	9	9	9	9	8	8	2	2	2	2	1	1	3	3	3	3	3	3	74	72	78	79	72	70	68	64	66	92	86	100	96	98	94	89	22	18	20	22	20	19	17	18	100	100	100	100	100	100	100	100	30	27	0	0	0	0	0	0	0	