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Original Article

Anaesthesia Section

Effect of Dexmedetomidine versus Propofol on Sevoflurane related Emergence Agitation in Paediatric Patients: A Randomised Clinical Study

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ABSTRACT

Introduction: Postoperative Emergence Agitation (EA) is a troublesome side-effect of sevoflurane anaesthesia. Drugs like dexmedetomidine and propofol offer significant benefits in reducing the incidence and severity of sevoflurane-related postoperative EA in paediatric patients.

Aim: To compare the efficacy of intravenous dexmedetomidine and propofol in reducing the incidence and severity of EA associated with sevoflurane anaesthesia in paediatric patients.

Materials and Methods: A randomised clinical study was conducted on 140 patients, belonging to American Society of Anaesthesiologists Physical Status (ASA PS) classes I and II, aged between 2-6 years, undergoing infraumbilical surgery lasting more than one hour. The patients were divided into two equal groups receiving dexmedetomidine 0.3 µg/kg (Group SD) and propofol 1 mg/kg (Group SP) at the start of skin closure, administered over 10 minutes. The incidence of EA in the Post Anaesthesia Care Unit (PACU) was evaluated using the Watcha scale, and the severity of EA was assessed using the Paediatric Anaesthesia Emergence Delirium (PAED) scale. A Watcha score of 3 or 4 indicated the presence of EA, while a PAED score of ≥12 was deemed significant. Statistical analysis of the data was performed using International Business Machines (IBM) Statistical Package for Social Sciences (SPSS) version 22.0, with an Independent sample t-test for comparing normally distributed quantitative parameters, and the Chi-square test for comparing categorical outcomes between the study groups.

Results: The mean age of the patients in group SD was 4.19±0.78 years, and in group SP was 4.03±0.71 years. Both study groups were found to be comparable in terms of patient characteristics such as age, sex, weight, and duration of surgery (p-value >0.05). The incidence of postoperative EA, as measured by the Watcha scale, was higher in group SP compared to group SD upon arrival and up to 30 minutes in the PACU (p-value <0.001). The severity of EA, assessed using the PAED score, was greater in group SP compared to group SD at 0, 5, 10,15 and 20 minutes in the PACU (p-value <0.001).

Conclusion: Dexmedetomidine 0.3 µg/kg was more effective than propofol 1 mg/kg in reducing the incidence and severity of EA associated with sevoflurane anaesthesia, with minimal haemodynamic effects and no clinically relevant severe adverse effects in both the groups. The significantly prolonged extubation times observed in the propofol group did not result in significantly longer stays in the PACU.

Keywords: Children, Delirium, Extubation, Restlessness, Sedative hypnotic

INTRODUCTION

Postoperative EA is a common phenomenon in children undergoing sevoflurane anaesthesia [1-3]. EA is characterised by agitation, restlessness, persistent crying, confusion, delusions, hallucinations, and cognitive shifts, including memory loss. The incidence ranges from 10-67% [4]. Many possible aetiologies have been proposed, including rapid awakening in unfamiliar settings, pain, noisy environment, stress during induction, hypoxemia, the child's personality, and the duration and type of anaesthesia [4-7]. There are numerous pharmacological and non pharmacological therapies available for treating postoperative EA [8-10]. Sevoflurane is a commonly used inhalational anaesthetic agent in the paediatric population due to its non pungency, smooth and rapid induction properties. Its low blood gas partition coefficient ensures prompt induction and recovery following sevoflurane discontinuation. Sevoflurane also induces bronchodilation and causes the least airway irritation among currently available volatile anaesthetics. Postoperative EA is a troublesome side-effect of sevoflurane anaesthesia. Various drugs have been utilised to facilitate smooth emergence from sevoflurane anaesthesia, such as dexmedetomidine, propofol, midazolam, clonidine, ketorolac, and fentanyl [8-10]. While there have been numerous studies on the use of propofol or dexmedetomidine to reduce sevoflurane-related EA, there are only a few studies comparing these two drugs in the published literature [11,12].

Dexmedetomidine is a selective α-2 agonist with 1600 times more affinity to α-2 than α-1. It possesses sedative, anxiolytic, and analgesic properties due to its central sympatholytic effects, making it suitable for use in intensive care and operating room settings [13,14]. It has been proven that dexmedetomidine reduces EA following sevoflurane anaesthesia in paediatric surgery and non surgical procedures [15]. Propofol is an intravenous anaesthetic agent administered as a 1% solution. It is believed to exert its sedative-hypnotic effects through γ-Aminobutyric acid type A (GABA A) receptor interaction. The quick recovery without residual sedation and low incidence of nausea and vomiting make propofol suitable for ambulatory conscious sedation techniques. Propofol also reduces postoperative sevoflurane-related EA [16,17].

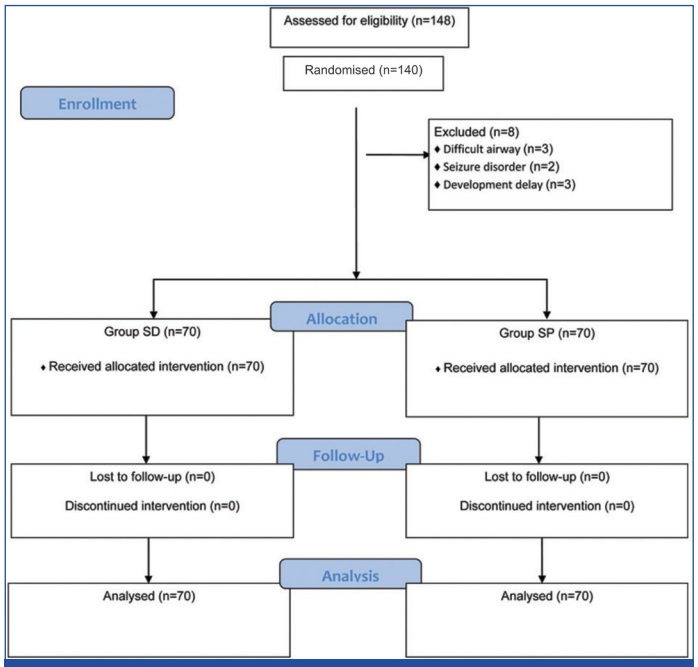
Authors hypothesised that dexmedetomidine was more effective than propofol in reducing sevoflurane-related EA in children. The aim of this study was to compare the efficacy of intravenous dexmedetomidine and propofol in reducing the incidence and severity of EA in paediatric patients undergoing sevoflurane anaesthesia.

MATERIALS AND METHODS

This randomised clinical study was conducted at Government Medical College, Kozhikode, Kerala, India from January 2018 to December 2019 after obtaining institutional review and ethical committee approval (Ref No. GMCKKD/RP2017/IEC/236), clinical trial registration (CTRI/2018/09/015838), and written informed consent from parents of children belonging to both genders undergoing urological surgeries or other infraumbilical surgeries.

Inclusion criteria: One hundred and forty-eight children aged between 2 to 6 years, scheduled for elective urological surgery and infraumbilical surgeries lasting for more than one hour, belonging to ASA PS I-II, and weighing between 10 to 25 kg, were included in the study.

Exclusion criteria: Children with developmental delay, spinal anomaly or neurological disease, psychiatric illness, cardiac disease, difficult airway, and those with a known allergy to the study drug. Based on these criteria, eight children were excluded from the study [Table/Fig-1].



[Table/Fig-1]: Consort flow diagram

Sample size: The sample size was calculated based on the incidence of EA from the randomised study by Ali MA and Abdellatif AA [11]. With an alpha error set at 0.05 and 90% power, it was estimated that 70 patients were required per group to achieve statistical significance (p-value <0.05).

After selection, the patients were randomised into two equal groups of 70 each using a random number table. Group SD included patients who received dexmedetomidine 0.3 µg/kg, and Group SP received propofol 1 mg/kg [11]. All patients underwent a detailed preanaesthetic check-up, including history, physical examination, and laboratory investigations. All children were kept nil per oral before surgery (8 hours for solid food, 6 hours for semisolid food, 2 hours for clear fluids). On the day of surgery, children were brought to the premedication room, and baseline HR, blood pressure, SpO_2, and respiratory rate were recorded. Subsequently, they received oral midazolam 0.5 mg/kg approximately half an hour before separation from parents.

In the operating room, after attaching all standard ASA non invasive monitors, which included an electrocardiogram, pulse oximeter, and non invasive blood pressure monitor, anaesthesia was induced with 8% sevoflurane and 66% nitrous oxide in oxygen via a face mask. Following the establishment of intravenous access under adequate anaesthetic depth, atracurium 0.5 mg/kg was administered intravenously, and oral endotracheal intubation was performed. Heart rate, blood pressure, end-tidal carbon dioxide, and oxygen saturation using a pulse oximeter were monitored throughout the procedure. Anaesthesia was maintained with 1.5% to 2% sevoflurane in 66% nitrous oxide in oxygen throughout the operation. Atropine 0.02 mg/kg was given during the procedure if the heart rate decreased to more than 30% of baseline values. For intraoperative and postoperative pain relief, all children received intravenous paracetamol 15 mg/kg, and a caudal epidural block with 0.25% bupivacaine was performed immediately after intubation. At the start of skin closure, group SD received dexmedetomidine 0.3 µg/kg diluted in 10 mL of normal saline, while group SP received 1 mg/kg of propofol intravenously over 10 minutes using a syringe pump. Sevoflurane, dexmedetomidine, and propofol were discontinued upon completion of skin closure. With the onset of spontaneous ventilation, residual muscle relaxation was reversed with Neostigmine 0.05 mg/kg and atropine 0.02 mg/kg intravenously. Extubation was performed after restoration of the child's gag reflex, regular respiration, and adequate muscle tone, and the children were transferred to PACU. The incidence and severity of EA were assessed using the Watcha scale, PAED score at various follow-up intervals, and the time of extubation were considered as primary outcome variables, while haemodynamic parameters such as Heart Rate (HR), Mean Blood Pressure (MAP), and the occurrence of various adverse effects were considered as secondary outcome variables [18-20].

HR, peripheral capillary oxygen saturation (SpO_2), and blood pressure were recorded after induction, at the start of the operation, just before loading of study drugs, after the end of the operation, and in the PACU. Since EA is more common within 30 minutes of PACU arrival, the incidence and severity of EA were measured upon arrival (T0) and then every five minutes (T5, T10, T15, T20, T25, T30) for up to 30 minutes in the PACU. This assessment was conducted by an anaesthesiologist who was blinded to the study intervention used. Watcha scores and PAED scores were employed as primary outcome measures for comparing the incidence and severity of EA, respectively. A score of 3 or 4 on the Watcha scale was considered indicative of EA [19]. The severity of EA was determined using the PAED scale [20], where a score of ≥12 was considered significant. Children were classified as severely agitated if their PAED score was 15/20 or higher, and these severely agitated children were administered 0.5 mg/kg fentanyl intravenously [11]. Children were discharged to the ward when the modified Aldrete score was above 9.

STATISTICAL ANALYSIS

All the raw data were entered into a Microsoft Excel spreadsheet, and the statistical analysis of the data was performed using IBM SPSS version 22.0 The study group (Group SP vs. Group SD) was regarded as the primary explanatory variable, with age and gender serving as other explanatory variables. For normally distributed quantitative parameters, mean values were compared between study groups using an unpaired t-test. Categorical outcomes were compared between study groups using the Chi-square test. Data are presented as mean±standard deviation, median (Interquartile Range), or as the number of patients and percentages. A p-value of <0.05 was considered statistically significant.

RESULTS

Both study groups were found to be comparable in terms of patient characteristics, including age, sex, weight, and duration of surgery [Table/Fig-2]. The incidence of EA was higher in group SP compared to group SD (p-value <0.001) [Table/Fig-3]. The severity of EA, as indicated by the PAED score, was significantly higher in group SP compared to group SD, except at 25 minutes and 30 minutes in the PACU (p-value >0.05) [Table/Fig-4]. The time of extubation between the two groups was statistically significant, with group SD exhibiting shorter extubation times (p-value <0.001) [Table/Fig-5].

Characteristics	Group SD (n=70)	Group SP (n=70)	p-value
Age (years)*	4.19±0.78	4.03±0.71	0.193
Gender			
Male	61 (87.14%)	61 (87.14%)	1.00
Female	9 (12.86%)	9 (12.86%)	1.00
Weight (kg)*	16.29±2.39	16.41±2.35	0.749
Duration of surgery (minutes)*	65.36±8.05	63.93±7.27	0.272

[Table/Fig-2]: Comparison of demographic and baseline characteristics.
Data represented as *mean±SD; †number of patients n (%), Unpaired t test, Chi-square test

Time point*	Group SD (n=70)		Group SP (n=70)		p-value
	Incidence n (%)	Watcha score (Mean±SD)	Incidence n (%)	Watcha score (Mean±SD)	
T0	0	0.59±0.6	41 (58.57)	2.51±0.93	<0.001
T5	0	0.8±0.67	44 (62.85)	2.57±0.86	
T10	2 (2.85)	1.07±0.82	41 (58.57)	2.66±0.7	
T15	2 (2.85)	1.29±0.74	35 (50)	2.73±0.83	
T20	9 (12.85)	1.20±0.99	31 (44.28)	2.47±0.86	
T25	10 (14.28)	1.14±0.87	40 (57.14)	2.41±1.01	
T30	10 (14.28)	1.10±0.83	22 (31.42)	1.79±1.14	

[Table/Fig-3]: Comparison of incidence of Emergence Agitation (EA) using Watcha scale at PACU.
*Time in minutes after arrival at PACU (post anaesthesia care unit.) Data expressed as percentage of patients, p value <0.05 considered significant (Chi-square test)

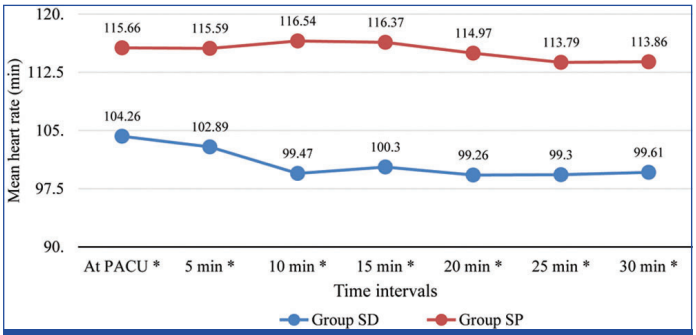
Time point	Group SD (n=70) (Mean±SD)	Group SP (n=70) (Mean±SD)	p-value
T0	6.56±2.83	11±3.55	<0.001
T5	7.2±2.98	11.64±2.65	<0.001
T10	8.97±3.2	11.76±3.52	<0.001
T15	10.20±3.53	12.10±3.31	0.001
T20	9.47±3.35	12.0±3.1	<0.001
T25	9.26±3.54	9.70±3.12	0.433
T30	9.17±3.12	9.01±3.39	0.776

[Table/Fig-4]: Comparison of PAED score at different follow-up intervals between study groups in PACU.
*Time in minutes after arrival at PACU (post anaesthesia care unit.) Data expressed as percentage of patients, p value <0.05 considered significant (T- test)

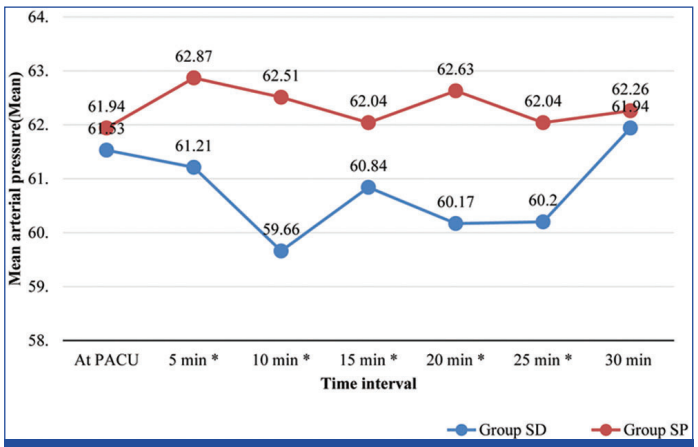
Parameter	Group SD (n=70) (Mean±SD)	Group SP (n=70) (Mean±SD)	p-value
Duration of PACU stay (minutes)	39.93±6.89	41±7.55	0.382
Time of extubation (minutes)	12.56±1.65	15.57±1.92	< 0.001*

[Table/Fig-5]: Comparison of mean duration of PACU stay and mean time of extubation between study groups (n=140).
*p value <0.05 considered significant (T- test)

Group SD demonstrated lower heart rates at various follow-up intervals compared to group SP, a difference that was statistically significant (p-value <0.001) [Table/Fig-6]. The MAP was comparable upon arrival in the PACU and after 30 minutes between group SD and group SP (p-value >0.05) [Table/Fig-7]. The few episodes of bradycardia and hypotension recorded in both groups were not clinically relevant and resolved without treatment [Table/Fig-8]. Respiratory rates and SpO_2 recorded at various time intervals were found to be comparable, except at 20 minutes when group SP exhibited lower respiratory rates (p-value=0.03); however, this was not deemed clinically relevant [Table/Fig-9].



[Table/Fig-6]: Comparison of Heart Rate (HR) (min) at different follow-up intervals between study groups (n=140).
*p-value <0.05 significant



[Table/Fig-7]: Comparison of mean arterial pressure at different follow-up intervals between study groups (n=140).
*p-value <0.05 considered significant

Side-effects	Group SD (n=70)	Group SP (n=70)	Chi square	p-value
Bradycardia	5 (7.14%)	2 (2.86%)	1.891	0.595
Hypotension	2 (2.86%)	1 (1.43%)		
Vomiting	2 (2.86%)	3 (4.29%)		

[Table/Fig-8]: Comparison of side-effects between study groups (n=140).
Data expressed as number of patients (%)

Parameter	Group SD (n=70)	Group SP (n=70)	p-value Unpaired t-test
At PACU			
SpO_2	98.73±0.72	98.69±0.71	0.724
Respiratory rate	24.34±0.76	24.36±0.66	0.906
5 mins			
SpO_2	98.74±0.74	98.84±0.67	0.403
Respiratory rate	24.44±0.67	24.51±0.56	0.495
10 mins			
SpO_2	98.61±0.57	98.84±0.77	0.051
Respiratory rate	24.43±0.65	24.5±0.5	0.469
15 mins			
SpO_2	98.61±0.64	98.44±1.29	0.322
Respiratory rate	24.31±1.14	24.4±0.97	0.632
20 mins			
SpO_2	98.79±0.76	98.81±0.73	0.821
Respiratory rate	24.64±1.42	24.17±1.12	0.031*
25 mins			
SpO_2	98.71±0.64	98.76±0.77	0.721
Respiratory rate	24.39±1.12	24.36±1.23	0.886
30 mins			
SpO_2	98.73±0.8	98.84±0.73	0.379
Respiratory rate	24.31±1.27	24.41±1.5	0.671

[Table/Fig-9]: Comparison of SpO_2 (%) and respiratory rate (per min) at different follow-up intervals between study groups (n=140).
*p value <0.05 considered significant, Data expressed as mean±SD

DISCUSSION

The EA is a common adverse effect of sevoflurane anaesthesia in children. Although it often resolves within 30 minutes, agitated

children may inadvertently harm themselves by bumping into objects, pulling off tubes, drains, and wound dressings. This situation can be distressing for parents and caregivers. With multifactorial origins, numerous pharmacological and non pharmacological measures have been proposed to mitigate sevoflurane EA [3].

In the current study, the efficacy of dexmedetomidine and propofol was compaared in preventing EA associated with sevoflurane anaesthesia in the paediatric population. Present study analysed the incidence and severity of EA, extubation time, haemodynamic changes, and side-effects. Zhu M et al., evaluated the effect of dexmedetomidine in various doses and routes for sevoflurane-related EA, encompassing a total of 1,364 patients (696 in the dexmedetomidine group and 668 in the placebo, fentanyl, and midazolam group) from 20 prospective Randomised Controlled Trial (RCT) in their meta-analysis. They reported a reduced incidence of EA with prolonged extubation times (WMD 0.61, 95% CI, 0.27-0.95) compared to placebo, and a similar incidence of EA, comparable extubation times, and less nausea and vomiting compared to opioids. The results of the current study align with these findings [8]. Another meta-analysis by Yang X et al., yielded similar results [21].

In the present study, the incidence and severity of EA were lower in the group that received dexmedetomidine compared to patients who received propofol. The difference in the incidence of EA in the initial 30 minutes in the PACU between the two groups was statistically significant (p-value <0.05), with the dexmedetomidine group exhibiting a lower incidence. group SD showed an increasing trend in the incidence of EA during the first 30 minutes in the PACU. This result in group SD contrasted with the study conducted by Ali MA and Abdellatif AA [11]. This difference may be attributed to the fact that their study population included children undergoing adenotonsillectomy, a surgery known for a high propensity for EA. group SD showed the highest incidence of EA at 25 and 30 minutes (14.2%). group SP exhibited the highest incidence within the first 15 minutes in the PACU, with the peak incidence (62.8%) occurring at five minutes in the PACU. This result reported in group SP in present study was consistent with the findings of the study by Ali MA and Abdellatif AA [11]. They observed that the incidence and severity of EA were high within the first 15 minutes in the control group, propofol group, and dexmedetomidine group in the PACU. The control group displayed a higher incidence of EA compared to the other two groups. The dexmedetomidine group exhibited the lowest incidence of EA compared to propofol, which aligns with our results.

The time of extubation between the two groups was statistically significant, with group SD having lower extubation times (p-value <0.001). The side-effects and duration of PACU stay between the two groups were comparable (p-values 0.595 and 0.382, respectively). These results were consistent with the study report by Ali MA and Abdellatif AA (p-value ≥0.05) [11]. Wu et al., found lower extubation times (11.35±3.17) with propofol 2 mg/kg i.v. compared to saline placebo (21.41±4.62) (p-value<0.001) given towards the end of surgery. They correlated this positively with lower PAED scores in the propofol group [17].

Ibacache ME et al., compared dexmedetomidine in two doses (0.15 g/kg, 0.3 µ/kg) with saline placebo given i.v. soon after sevoflurane induction in infraumbilical surgeries. They reported a lower incidence of EA, along with stable haemodynamics with dexmedetomidine [15]. Abu Shahwan I had similar results with propofol (1 mg/kg) given i.v. at the completion of an Magnetic Resonance Imaging (MRI) procedure in children under sevoflurane anaesthesia. There were no significant changes in heart rates or mean arterial pressure when compared to saline placebo [16].

The present study also found comparable results with the above-mentioned studies in terms of haemodynamic parameters. The HR and MAP showed lower values in the dexmedetomidine group, exhibiting a significant difference at various time intervals compared to the propofol group, but neither of these differences were clinically relevant to warrant treatment. In a study by Kim NY et al., on the effect of dexmedetomidine in sevoflurane-related EA, it was found that intraoperative infusion of dexmedetomidine (1 µg/kg followed by 0.1 µg/kg/hour) reduced sevoflurane requirements and EA without delaying discharge in children. In their study, the mean arterial blood pressure and heart rate were significantly lower in the dexmedetomidine group compared to the saline group [22]. This difference may be attributed to the higher dose of dexmedetomidine administered. Costi D et al., in their Cochrane review, suggested combining effective interventions as a multimodal approach to further reduce the risk of EA [3].

Limitation(s)

Postoperative pain, which may be an independent correlate of EA, was not assessed in the present study.

CONCLUSION(S)

Dexmedetomidine 0.3 µg/kg reduces the incidence and severity of EA more effectively than propofol 1 mg/kg when administered 10 minutes before the end of surgery in paediatric infraumbilical surgery under sevoflurane anaesthesia. Although the extubation time was significantly longer with propofol, it was not found to associate with significantly longer PACU stays. Lower MAP and HR were documented with dexmedetomidine, showing a significant difference between the groups. Both propofol and dexmedetomidine did not produce any clinically relevant changes in haemodynamic parameters or any adverse effects that warranted treatment. Thus, it can be concluded that the administration of dexmedetomidine is safe and more effective in reducing sevoflurane-related EA. Further research may explore alternate routes and doses or multimodal approaches to prevent EA.

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