DOI: 10.7860/JCDR/2022/52943.16848

Original Article

Anaesthesia Section

Effectiveness of Low Dose Dexmedetomidine for an Oligaemic Field in Middle Ear Surgery: A Randomised Control Trial

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ABSTRACT

Introduction: Microscopic middle ear surgeries are best done under hypotensive anaesthesia to provide an oligaemic field. Dexmedetomidine is an α2 agonist which produces hypotension by sympathetic response blockade.

Aim: To study the effectiveness of low dose dexmedetomidine in providing an oligaemic field. The additional requirement of inhalational agent required to maintain the oligaemic field in the control group, intubation response and postoperative analgesia were also assessed.

Materials and Methods: The randomised, placebo-controlled double-blind study was conducted from January to December 2013 by recruiting 48 patients in the study, who were divided into two groups i.e, study group (n=24) and placebo group (n=24). The study group received low dose dexmedetomidine infusion (0.5 µg/kg loading dose followed by 0.2 µg/kg/hr) and placebo group received weight-adjusted saline. The quality of the surgical field was assessed on a score of 0-5. The Minimum Alveolar Concentration (MAC) of inhalational agent needed, postoperative sedation score and pain score were noted. All the data were recorded in Statistical Package for Social Sciences (SPSS) software, version 22.0.

Results: The mean score of quality of the surgical field in the study group was 2.00±0.59, compared to 3.50±0.88 in the placebo group. The mean MAC of isoflurane was 1.32±0.31 in the placebo group and 0.85±0.23 in the study group (p-value=0.03). The study group had a Ramsay sedation score of 3.08±0.65 compared to 2.29±0.75 in the placebo group at 5 minutes after extubation (p-value <0.001). The mean pain score in the dexmedetomidine group in 10 minutes postoperative period was 1.83±2.25, while in placebo group it was 3.25±1.96 (p-value=0.025). However, over the next 20-60 minutes postoperative period the pain score between the two groups were comparable, with no significant difference in analgesia, postoperative shivering and other side-effects.

Conclusion: A low dose dexmedetomidine can effectively provide a bloodless field for middle ear microsurgical procedures.

Keywords: Bloodless surgical field, Controlled hypotension, Hypotensive anaesthesia

INTRODUCTION

The middle ear microsurgeries need a bloodless surgical field, attention to patient's head positioning, airway management, facial nerve monitoring, avoidance of nitrous oxide, smooth and calm recovery, and prevention of postoperative nausea and vomiting [1]. The bloodless surgical field is preferred for safe tympanic membrane and ossicular reconstruction as blood can obscure the surgical field. Various methods are employed including reverse Trendelenburg position, local infiltration with vasoconstrictors and pharmacological agents to provide controlled hypotension. Controlled hypotension is a method by which arterial blood pressure is decreased predictably and deliberately to decrease blood loss. The level of hypotension is determined by achieving a bloodless field within the safety limits of cerebral and coronary flow. This safety limit is not fixed and generally taken as Mean Arterial Pressure (MAP) as low as 50 mmHg or a 30% drop in MAP in American Society of Anaesthesiologists (ASA) physical status I subject [2]. The safety limit of a chronic hypertensive patient will be different and they may not tolerate a drop of more than 25% of the MAP, due to the shift of cerebral auto regulation limit towards higher blood pressure [2].

Dexmedetomidine is a centrally acting α 2A agonist. It acts by presynaptic activation of α-2 adrenoceptors, thus inhibiting the release of norepinephrine, terminating the propagation of pain signals and postsynaptic activation of α-2 adrenoceptors in the Central Nervous System (CNS), thus inhibiting sympathetic activity. Dexmedetomidine evokes a biphasic blood pressure response. At lower doses, the dominant action of dexmedetomidine is sympatholytic mediated by α-2A adrenergic receptor and thus is used to provide controlled hypotension by decreasing blood pressure and heart rate, these actions have been put to use, as an infusion, for providing ideal condition for middle ear microsurgical procedure [3].

Under general anaesthesia, dexmedetomidine is commonly used to attenuate sympathoadrenal response, to provide analgesia by reducing the requirement of intraoperative and postoperative opioid requirement, emergence agitation prevention and reducing postoperative shivering [4]. The doses used varies from 1-0.5 µg/kg loading dose with the maintenance of 0.75-0.4 µg/kg/hr [3,4].

The sympatholytic effect of dexmedetomidine in adults was studied in previous research using dose of 1 µg/kg loading dose followed by 0.4-0.6 µg/kg/hr [5-8]. The adverse effects of dexmedetomidine are hypotension, hypertension, nausea, hypoxia, bradycardia, first and second degree atrioventricular block as well as atrial fibrillation at higher doses [9]. In an attempt to avoid adverse effects of higher doses, in the current study, dexmedetomidine was used at a low dose of 0.5 µg/kg intravenously over 10 minutes as a bolus dose and then infusion at the rate of 0.2 µg/kg/hr. The primary objective was to assess the effectiveness of test drug in lower doses to provide a bloodless field for middle ear microsurgical procedure. The secondary objectives were to assess haemodynamic response to laryngoscopy and intubation, the stress response of surgery by assessing heart rate and MAP and observe any other adverse effects postoperatively.

MATERIALS AND METHODS

The randomised, placebo-controlled double-blinded study was conducted at the Bangalore Baptist Hospital, Bengaluru, Karnataka, India, from January to December 2013. The approval from Institutional Ethics Committee was taken (BBH/IRB/2012/019). The

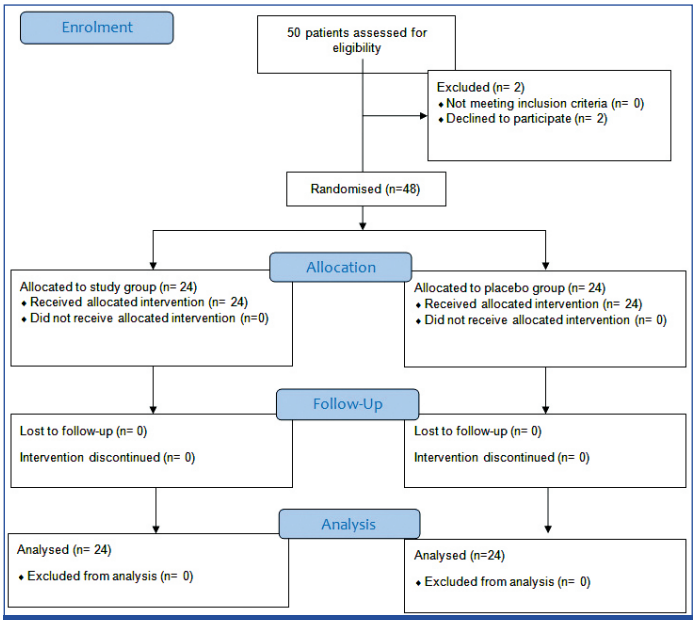
patients were explained about the procedure and informed consent was taken to include in the study.

Inclusion criteria: The patients scheduled for elective, middle ear surgeries under general anaesthesia in the age group of 16-55 years in ASA grade I and II were included in the study, if they consented to it.

Exclusion criteria: Pregnant, lactating mothers, patients with renal impairment, hepatic dysfunction, history of allergic drug reactions and on antihypertensive medication with α-2 agonists, α methyldopa were excluded from the study. Patients in whom dexmedetomidine infusion was discontinued due to haemodynamic derangements during the study were also excluded from the study.

Sample size calculation: Based on a pilot study, the surgical field scoring (primary objective) was expected to lower by 30% in patients who received dexmedetomidine compared to the placebo group. The sample size was calculated as 48 (24 patients per group) with 80% power and a significance level of 0.05. The sample size was calculated by the application online "sample size calculator" [10].

- **Study group** (n=24): Dexmedetomidine 0.5 μg/kg intravenously over 10 minutes as a bolus dose and then infused at the rate of 0.2 μg/kg/hr (100 μg/50 mL syringe -2 μg/mL, diluted with normal saline) till the end of the surgery.
- **Placebo group** (n=24): Received weight-adjusted normal saline at the same volume (mL) and rate (mL/hr) as in study group [Table/Fig-1].



[Table/Fig-1]: CONSORT flow diagram.

Allocation process: Patients were randomised by using the block randomisation process, with each size block of 4, to get equal numbers in both study as well as the placebo group. With a block size of 4, there were six possible ways to equally assign participants to a block. Allocation concealment included sequentially structured alphabets of 4 kept in a sealed opaque envelope. The patient and the investigator involved both were blinded. The infusion was prepared by an anaesthesiologist, not involved in the study.

Study Procedure

Preoperative assessment was done for all the patients and nil per orally for six hours before surgery followed. All patients were premedicated with oral alprazolam 0.5 mg at night one day prior and in the morning of the surgery for anxiolysis. The monitors including non invasive blood pressure monitor, pulse oxymeter, capnography and integrated anaesthetic gas analyser (Aspire 7900 with Philips IntelliVue MP40) were used intraoperatively to monitor Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), Heart Rate (HR), oxygen saturation (SpO_2), end-tidal CO_2, and Minimum Alveolar Concentration (MAC) throughout the surgery. Two intravenous accesses were secured, one for fluid and the other one dedicated to study drugs. All the patients were preoxygenated and premedication with glycopyrrolate 0.2 mg intravenously (i.v.) in the operation theatre. The induction of anaesthesia was done with a graded dose of propofol up to 2 mg/kg, fentanyl 2 μg/kg and atracurium 0.5 mg/kg i.v., followed by endotracheal intubation. Intraoperative analgesia was provided by morphine 0.1 mg/kg i.v. as a bolus. Maintenance of anaesthesia was with 50% oxygen, 50% air (FiO_2 of 0.5) with low flow anaesthesia, 1% isoflurane and intermittent positive pressure ventilation. The target of induced hypotension was a 30% reduction of MAP from baseline, which was intraoperative done by titrating inhalational agents to the desired effect. Controlled hypotension was provided in all the patients and MAC of isoflurane needed was also noted. Intravenous atracurium 0.1 mg/kg was used for maintenance.

Surgical field: Quality of the surgical field was assessed at the end of the surgery by asking the operating surgeon to score on a scale of 0-5, where '0' meant no bleeding, and '5' stood for severe bleeding which needed continuous suctioning. The desired level was to achieve ≤2 [11].

Sedation score: The sedation score was assessed by Ramsay sedation score recorded preoperatively, after the bolus infusion, before induction and at 30 minutes postoperatively [12].

Any hypertension and tachycardia more than 20% from preoperative value with MAC level of 1 was considered as inadequate analgesia and i.v. fentanyl 1 μg/kg was given.

Adverse effects:

- Hypotension (MAP <60 mmHg) was treated with intravenous fluids and IV ephedrine 6 mg.
- Severe bradycardia (HR<40/min) was treated by atropine 0.6 mg IV. At the end of the surgery, ondansetron 0.1 mg/kg IV was administered to all patients, residual neuromuscular block was reversed with neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg IV.

Postoperative period: During the postoperative period nausea, vomiting, respiratory depression, shivering, Visual Pain Analogue (VAS) scores (0=no pain, 10=worst pain), sedation (Ramsay sedation score) and vitals were recorded.

Recovery: The recovery of all the patients was assessed by Aldrete's score and shifting from the postoperative ward was done after achieving satisfactory recovery [13].

STATISTICAL ANALYSIS

All the data were recorded in Statistical Package for Social Sciences (SPSS) software version 22.0. The primary objective, quality of the surgical field was compared by the Mann-Whitney U test. Results on continuous measurements like vitals were presented as Mean±SD (Minimum-Maximum), and an independent student t-test was used to compare between the groups. Significance was assessed at a 5% level of significance.

RESULTS

The basic characteristics including, age, gender, weight and ASA grade were comparable between the two groups [Table/Fig-2]. The quality of the surgical field was better in the study group [Table/Fig-3]. The mean MAC of isoflurane was 1.32±0.31 in the placebo group and 0.85±0.23 in the study group, with p-value of 0.03.

The effect on stress response of intubation assessed by change in HR and MAP at 30 seconds after intubation and the increase in HR was more in placebo group compared to study group. This was statistically significant (p-value <0.001). The MAP in the study group decreased compared to baseline after 30 seconds of intubation while increase in MAP was noted in placebo group.

From 30 seconds postintubation to postoperative period, there was a significant difference between the two groups in HR and MAP [Table/Fig-4,5].

Age in years	Study group n (%)	Placebo group n (%)	p-value (Student t-test)
16-25	11 (45.8)	8 (33.3)	0.592
26-35	6 (25)	7 (29.2)	
36-45	4 (16.7)	6 (25)	
46-55	3 (12.5)	3 (12.5)	
Gender			
Male	17 (70.8)	15 (62.5)	0.54
Female	7 (29.2)	9 (37.5)	
ASA grade			
I	18 (75)	15 (62.5)	0.35
II	6 (25)	9 (37.5)	
Weight (kg)			
Mean±SD	61.54±7.80	64.25±8.96	0.27

[Table/Fig-2]: Basic characteristics of the two groups of patients studied.

Quality of surgical field	Study group n (%)	Placebo group n (%)	p-value (Student t-test)
1	4 (16.7)	-	
2	16 (66.7)	3 (12.5)	
3	4 (16.7)	9 (37.5)	
4	-	9 (37.5)	
5	-	3 (12.5)	
Mean±SD	2.00±0.59	3.50±0.88	<0.001

[Table/Fig-3]: Quality of surgical field.

Heart rate	Study group (Mean±SD)	Placebo group (Mean±SD)	p-value (Student t-test)
Baseline	80.46±12.26	83.83±9.82	0.298
5 minutes (min) after bolus	73.50±10.97	78.46±6.82	0.066
10 min	67.63±8.73	72.63±6.22	0.027
After intubation			
30 sec	83.96±9.06	105.29±7.98	<0.001
5 min	80.71±13.91	96.33±7.54	<0.001
10 min	75.21±12.94	83.71±7.35	0.007
15 min	76.67±13.52	83.71±7.35	0.030
45 min	67.21±9.66	87.04±6.12	<0.001
75 min	67.96±13.95	87.04±6.12	<0.001
105 min	75.42±13.37	89.42±5.69	<0.001
135 min	69.42±14.37	90.17±6.05	<0.001
165 min	68.58±15.69	91.33±5.85	<0.001
195 min	65.75±12.67	91.00±5.66	<0.001
225 min	65.33±11.45	90.13±4.66	<0.001
255 min	65.38±12.80	88.17±4.97	<0.001
Immediate postextubation	80.83±16.83	107.96±5.05	<0.001
5 min	77.33±14.70	86.83±4.93	0.004
10 min	76.29±14.04	85.96±5.17	0.003
20 min	75.04±14.09	85.96±5.48	0.001
30 min	70.88±20.63	85.67±4.74	0.001

[Table/Fig-4]: Comparison of heart rate among the two groups.

MAP (mmHg)	Study group (Mean±SD)	Placebo group (Mean±SD)	p-value (Student t-test)
Baseline	99.67±9.61	99.99±8.82	0.926
5 min (after bolus)	90.29±11.45	97.33±4.48	0.007
10 min	82.42±10.09	87.58±7.46	0.050
After intubation			
30 sec	90.71±10.36	114.13±9.41	<0.001
5 min	77.38±13.97	100.38±8.85	<0.001
10 min	69.79±9.99	92.96±8.79	<0.001
15 min	74.46±11.69	99.00±8.54	<0.001
45 min	74.25±13.43	101.58±7.93	<0.001
75 min	75.71±13.12	103.04±8.15	<0.001
105 min	76.29±11.94	105.00±9.56	<0.001
135 min	74.71±9.61	105.75±6.63	<0.001
165 min	74.58±10.59	105.29±6.13	<0.001
195 min	77.75±10.34	103.04±4.22	<0.001
225 min	77.79±9.86	95.92±3.90	<0.001
255 min	77.83±9.07	94.58±3.78	<0.001
Immediate postextubation	95.67±12.43	113.17±8.03	<0.001
5 min	91.46±11.43	104.08±4.45	<0.001
10 min	89.63±8.90	100.13±4.72	<0.001
20 min	86.38±7.82	98.17±5.13	<0.001
30 min	85.38±11.64	97.50±3.72	<0.001

[Table/Fig-5]: Comparison of MAP among the two groups.

The SpO_2 and $EtCO_2$ did not show significant changes except at 45 minutes and 15 minutes, respectively. The baseline SpO_2 was lesser in the study group but not clinically significant [Table/Fig-6].

Parameters	Study group (Mean±SD)	Placebo group (Mean±SD)	p-value (Student t-test)
SpO_2 (%)			
Baseline	98.92±1.10	99.67±0.48	0.004
10 min (after bolus dose)	99.83±0.48	99.79±0.41	0.750
After intubation			
30 sec	99.42±1.10	99.67±0.64	0.340
15 min	99.50±1.06	99.79±0.41	0.217
45 min	99.08±1.28	99.75±0.53	0.023
75 min	98.83±0.96	99.50±0.78	0.011
135 min	99.38±1.01	99.54±0.78	0.526
195 min	99.79±0.59	99.58±0.78	0.300
255 min	98.96±1.16	99.29±1.23	0.340
Immediately after estuation	98.58±1.25	98.79±1.02	0.530
30 min	99.79±0.59	99.67±0.48	0.425
End tidal carbon dioxide			
30 sec after intubation	27.75±1.94	27.71±2.93	0.954
15 min	31.46±2.45	28.83±3.19	0.002
45 min	31.13±2.63	30.08±3.24	0.228
75 min	30.92±2.21	29.83±3.61	0.216
135 min	31.46±2.23	31.17±3.69	0.742
195 min	31.04±2.49	29.79±3.53	0.163
255 min	31.75±2.36	30.96±3.65	0.377

[Table/Fig-6]: Oxygen saturation (SpO_2) and end tidal CO_2 ($EtCO_2$) in both the groups.

The Ramsay sedation score was higher in the study group after 10 minutes of bolus dose and immediately postextubation postoperatively (p-value <0.001). After 30 minutes postextubation sedation scores were comparable in both the groups. All the patients in the study group were arousable, comfortable and maintained normal oxygen saturation on room air in Post Anaesthesia Care Unit (PACU) [Table/Fig-7].

The mean VAS pain score was lower in the study group in the immediate postoperative period. However, over the next 20-60 minutes postoperative period the pain score between the two groups were comparable.

Parameters	Study group (Mean±SD)	Placebo group (Mean±SD)	p-value
Sedation score			
Baseline	1.71±0.46	1.71±0.46	1.000
10 min	2.75±0.68	2.04±0.62	<0.001
Postextubation	3.08±0.65	2.29±0.75	<0.001
30 min	2.79±0.58	2.46±0.59	0.056
VAS score			
5 min postoperative	1.71±2.01	3.21±1.96	0.012
10 min postoperative	1.83±2.25	3.25±1.96	0.025
20 min postoperative	1.96±2.35	3.00±1.91	0.099
30 min postoperative	1.54±1.69	3.08±1.17	0.001

[Table/Fig-7]: Sedation score and VAS score among the two groups.

The hypotension and bradycardia were not found in any patient throughout the study, while the incidence of shivering and postoperative nausea and vomiting (PONV) were similar in both groups [Table/Fig-8].

Adverse events	Study group n (%)	Placebo group n (%)	p-value
Shivering	2 (8.3%)	2 (8.3%)	1
PONV	2 (8.3%)	2 (8.3%)	1

[Table/Fig-8]: Adverse events.

DISCUSSION

The present study was designed to study the effect of low dose dexmedetomidine in middle ear surgeries, which was found to provide an oligaemic surgical field without clinically significant adverse effects of higher doses. The blunting of intubation response and prolongation of postoperative analgesia was not found with low doses of dexmedetomidine.

Controlled hypotension and oligaemic surgical field: In current study, oligaemic surgical field could be achieved with 0.5 µg/kg loading dose followed by 0.2 µg/kg/hr of dexmedetomidine in middle ear surgery. In children for cochlear implant surgery and nasal surgery, to optimise the surgical field, dexmedetomidine was used in low doses of 0.5 µg/kg followed by 0.2-0.5 µg/kg/hr. Out of 35 patients, 33 patients had bleeding score ≤2 in cochlear surgery [14]. For nasal surgery in pediatric patients, 97% of patients attained an intraoperative surgical field score of ≤2 [15]. In another study, a loading dose of 1 µg/kg of dexmedetomidine followed by 0.4-0.8 µg/kg/hr helped in achieving intraoperative surgical field score of ≤2 in Functional Endoscopic Sinus Surgery (FESS) surgery [16]. A randomised control trial, conducted with dexmedetomidine with loading dose of 1 µg/kg followed by 0.4-0.8 µg/kg/hr for endoscopic surgery, concluded similar findings of lower Besant's scale score of intraoperative blood loss [17]. Another study by Durums M et al., using dexmedetomidine 1 µg/kg for 10 minutes followed by 0.5 µg/kg/hr, in tympanoplasty surgeries found significantly low blood loss in patients who received dexmedetomidine compared to placebo [5]. Other than Ear, Nose and Throat (ENT) surgeries, research was done on scoliosis patients where dexmedetomidine was used in a dose of 1 µg/kg followed by 0.2-0.5 µg/kg/hr for spine deformity correction surgery. They concluded that the blood loss was significantly low [18]. In the present study, low dose of dexmedetomidine as bolus and maintenance was found to be effective in achieving oligaemic surgical field.

Nasreen F et al., conducted a study in middle ear surgery, intending to maintain a 30% reduction in MAP, comparing dexmedetomidine 1 µg/kg for 10 minutes followed by 0.4 µg/kg/hr with placebo, concluded lower percentage of halothane use in dexmedetomidine group [6]. Gupta K et al., also conducted a study in patients requiring middle ear surgery with the goal of a 30% reduction in MAP for the oligaemic surgical field. They used dexmedetomidine in a dose of 0.5 µg/kg/hr, which was started after induction of anaesthesia and used isoflurane as an inhalational agent [7]. The mean dose of isoflurane in the placebo group was 1.6%, while, in the dexmedetomidine group mean was 0.8%. The use of an inhalational agent to produce controlled hypotension is standard practice, but the anticipated effect to decrease blood loss is controversial as the vasodilation produced by higher doses of inhalational agents can increase the blood loss [19]. At a higher concentration of inhalational agents, vasodilatation can increase cerebral circulation and in effect lead to increased intracranial pressure. The cerebral auto regulation gets impaired at higher concentrations of isoflurane [20]. In the present study, to achieve oligaemic field in the placebo group, higher MAC of the inhalational agent (isoflurane) was needed, which was comparable to the previous study [7].

The low dose dexmedetomidine decreases the requirement of isoflurane and is an effective adjuvant to decrease blood loss in safer doses of isoflurane. More than 1.3 MAC of isoflurane was found to be MAC of burst suppression which will not be required with dexmedetomidine as an adjuvant [21].

Intubation response: Dexmedetomidine is known to prevent intubation response in a dose of 1 µg/kg and 0.75 µg/kg effectively [22,23]. The lower dose of 0.5 µg/kg used in the present study was effective in preventing any change in MAP, while in HR there was an almost 4% increase. The sympathetic response was prevented but complete abolition was not there.

Sedation, postoperative analgesia and side effects: In the present study, hypotension and bradycardia were not found in any patient. The oxygen saturation and $EtCO_2$ remained well preserved in both the groups, the difference in sedation score was highly significant between the two groups after the bolus infusion and in the immediate postoperative period. The patients in dexmedetomidine were asleep but arousal in the immediate postop period. These findings were in line with a previous study [24].

Intraoperative dexmedetomidine helps in providing postoperative analgesia and decreasing postoperative shivering [25]. These effects were found to be more prominent after loading dose of 0.75 µg/kg and 1 µg/kg than 0.5 µg/kg [26]. In present study, the mean pain score in dexmedetomidine group was low only in the immediate postoperative period. Any prolonged effect was not noted. In the recovery area, the incidence of PONV and shivering were similar in both groups and comparable. The postoperative effects of dexmedetomidine in form of prolonged analgesia and decrease in shivering (two patients in both groups) were not found with low dose in the present study.

Limitation(s)

The sample population consisted only adult patients who were relatively healthy (ASA physical status grade I and II). It needs to be determined whether the present findings can be generalised to other age group and ASA status as well.

CONCLUSION(S)

Dexmedetomidine 0.5 µg/kg bolus infusion over 10 minutes before induction followed by 0.2 µg/kg/hr maintenance infusion can be recommended for all patients of ASA status I and II undergoing middle ear microsurgeries for providing controlled hypotension and better surgical field for the ENT surgeon. This technique could also be used for patients undergoing any other surgical procedure safely where the operating field has to be clear and to reduce blood loss.

REFERENCES

[1] Deacock AR. Aspects of anesthesia for middle ear surgery and blood loss during stapedectomy. Proc R Soc Med. 1971;64(12):1226-28.
[2] Degoute CS. Controlled hypotension: A guide to drug choices. Drugs. 2007;67(7):1053-76.
[3] Naaz S, Ozair E. Dexmedetomidine in current anaesthesia practice- A review. J Clin Diagn Res. 2014;8(10):01-04.

[4] Murthy TVPS, Singh R. Alpha 2 adrenoceptor agonist-dexmedetomidine role in anaesthesia and intensive care: A clinical review. J Anaesth Clin Pharmacol. 2009;25(3):267-72.
[5] Durmus M, But AK, Dogan Z, Yucel A, Miman MC, Ersoy MO. Effect of dexmedetomidine on bleeding during tympanoplasty or septorhinoplasty. Eur J Anaesthesiol. 2007;24(5):447-53.
[6] Nasreen F, Bano S, Khan RM. Dexmedetomidine used to provide hypotensive anaesthesia during middle ear surgery. Indian J Otlaryngol Head Neck Surg. 2009;61:205-07.
[7] Gupta K, Bansal M, Gupta PK, Pandey MN, Agrawal S. Dexmedetomidine infusion during middle ear surgery under general anaesthesia to provide oligaemic surgical field: A prospective study. Indian J Anaesth. 2015;59(1):26-30.
[8] Ayoglu H, Yapakci O, Ugur MB, Uzun L, Altunkaya H, Ozer Y, et al. Effectiveness of dexmedetomidine in reducing bleeding during septoplasty and tympanoplasty operations. J Clin Anesth. 2008;20(6):437-41.
[9] Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colinco MD. The effects of increasing plasma concentrations of dexmedetomidine in humans. Anesthesiology. 2000;93:382-94.
[10] Available from: https://www.surveysystem.com/sscalc.htm.
[11] Boezaart AP, van der Merwe J, Coetzee A. Comparison of sodium nitroprusside- and esmolol-induced controlled hypotension for functional endoscopic sinus surgery. Can J Anaesth. 1995;42(5 Pt 1):373-76.
[12] Das M, Ray M, Mukherjee G. Hemodynamic changes during laparoscopic cholecystectomy: Effect of clonidine premedication. Indian Journal of Anaesthesia. 2007;51(3):205-10.
[13] Crispian Scully, 3-Perioperative care, Editor(s): Crispian Scully, Scully's Medical Problems in Dentistry (Seventh Edition). Churchill Livingstone. 2014:51-96. ISBN 9780702054013. Available from: https://doi.org/10.1016/B978-0-7020-5401-3.00003-05.
[14] Amin SM, Elmawy MGE. Optimising surgical field during cochlear implant surgery in children: Dexmedetomidine versus Small. Egyptian Journal of Anesthesia. 2016;32(3):255-61.
[15] Amin SM, Elmawy MG, Mohamed RM. Controlled hypotensive anesthesia in children undergoing nasal surgery. J Anesth Clin Res. 2016;7(8):658-65.
[16] Shams T, Bahnasawe NSE, Abu-Samra M, El-Masry R. Induced hypotension for functional endoscopic sinus surgery: A comparative study of dexmedetomidine versus esmolol. Saudi J Anaes. 2013;7(2):175-80.
[17] Parvizi A, Haddadi S, Habibi AF, Nemati S, Akhtar N, Ramezani H. Dexmedetomidine efficacy in quality of surgical field during endoscopic sinus surgery. Iran J Otorhinolaryngology. 2019;31(106):281-88.
[18] EL Gohary MM, Arafa AS. Dexmedetomidine as hypotensive agent: Efficacy and hemodynamic response during spinal surgery for idiopathic scoliosis in adolescents. Egyptian Journal of Anaesthesia. 2010;26(4):305-11.
[19] Simpson P. Perioperative blood loss and its reduction: The role of the anaesthetist. Br J Anaesth. 1992;69:498-07.
[20] Slupe AM, Kirsch JR. Effects of anaesthesia on cerebral blood flow, metabolism, and neuroprotection. J Cereb Blood Flow Metab. 2018;38(12):2192-08.
[21] Pilge S, Jordan D, Kreuzer M, Kochs EF, Schneider G. Burst suppression-MAC and burstt suppression-CP50 as measures of cerebral effects of anaesthetics. Br J Anaesth. 2014;112(6):1067-74.
[22] Aho M, Lehtinen AM, Erkola O, Kallio A, Korttila K. The effect of intravenously administered dexmedetomidine on perioperative hemodynamics and isoflurane requirements in patients undergoing abdominal hysterectomy. Anesthesiology. 1991;74(6):997-02.
[23] Nava D, Núñez JC, Hernandez B, Barito AP, Diab AS. Evaluation of the effect of dexmedetomidine on the suppression of the adrenergic response to laryngoscopy and intubation. J Anesth Clin Res. 2018;9(6):828.
[24] Hall JE, Uhrich TD, Barney JA, Arian SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. Anaesth Analg. 2000; 90:699-05.
[25] Bicer C, Esmaoglu A, Akin A, Boyaci A. Dexmedeomidine and meperidine prevent postanaesthetic shivering. Eur J Anaesthesiol. 2006;23:149-53.
[26] Kim YS, Kim YI, Seo KH, Kang HR. Optimal dose of prophylactic dexmedetomidine for preventing postoperative shivering. Int J Med Sci. 2013;10(10):1327-32.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Oct 23, 2021
- Manual Googling: Mar 30, 2022
- iThenticate Software: Jul 07, 2022 (16%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Oct 20, 2021**
Date of Peer Review: **Nov 20, 2021**
Date of Acceptance: **Apr 26, 2022**
Date of Publishing: **Sep 01, 2022**